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Editorial

Family Medicine at the Crossroads: Chronic Disease, Neurodegeneration, Tobacco, Artificial Intelligence, and the Expanding Complexity of Modern Care

Family medicine is entering one of the most transformative periods in its history. The modern primary care physician is no longer managing isolated diseases but rather confronting a convergence of chronic illness, behavioral risk factors, neurodegeneration, mental health challenges, technological disruption, and rapidly evolving societal realities. The five papers presented in this issue collectively illustrate the widening scope and complexity of contemporary family medicine across the Middle East and beyond.

Several unifying themes emerge from these contributions. Most importantly, they emphasize that healthcare is increasingly shaped by interconnected biological, behavioral, technological, and psychosocial forces that cannot be adequately addressed through fragmented or disease-centered models of care.

The study examining smoking prevalence among primary healthcare patients highlights a continuing and evolving public health challenge in the region. Tobacco use remains deeply embedded within many societies, yet its forms are rapidly changing. The transition from traditional cigarettes toward vaping devices and medwakh among younger populations reflects not merely a behavioral shift but also the influence of marketing, social perception, and technological modernization. Family physicians now face the challenge of addressing nicotine addiction in forms that many patients mistakenly perceive as safer alternatives. The findings reinforce the essential role of primary care in prevention, counseling, and long-term behavioral intervention.

At the same time, chronic disease management itself is becoming increasingly complex. The "ABCDEF Ultra Violet S" framework represents an important attempt to restore comprehensiveness and structure to primary care practice. Modern family physicians must simultaneously manage diabetes, hypertension, cardiovascular disease, chronic kidney disease, obesity, depression, vaccination gaps, cancer screening, and lifestyle counseling within limited clinical encounters. In many healthcare systems, fragmentation of care has become one of the greatest barriers to effective chronic disease management. Structured frameworks may therefore help reduce omission errors, standardize preventive care, and improve continuity across increasingly complicated patient populations.

The pediatric chronic spontaneous urticaria case report further demonstrates another defining characteristic of family medicine: individualized patient-centered care. Not every clinical challenge can be solved through rigid algorithms alone. While evidence-based medicine remains fundamental, frontline clinicians frequently encounter patients who do not respond predictably to standard pathways. In such circumstances, careful therapeutic trials, close follow-up, and individualized decision-making remain essential clinical skills. The report also reminds us that even seemingly benign therapies may carry important neuropsychiatric implications, underscoring the importance of cautious prescribing and shared decision-making.

Perhaps the most provocative contribution in this issue concerns artificial intelligence and the phenomenon described as "artificial hallucination." Medicine is increasingly entering an era in which patients interact with AI systems not simply as information tools, but as emotional companions, sources of validation, and psychological influences. This development may profoundly reshape patient behavior, mental health dynamics, and healthcare communication. Family physicians are likely to become the first clinicians to observe the psychosocial consequences of excessive digital immersion, AI-mediated misinformation, emotional dependency, and distorted perceptions of reality. The challenge moving forward will not be whether artificial intelligence should be used, but how it can be integrated responsibly while preserving human judgment, empathy, and psychological resilience.

The review on Parkinson's disease dementia highlights another critical demographic reality confronting healthcare systems: population aging and the growing burden of neurodegenerative disease. Family physicians increasingly manage patients with overlapping motor, cognitive, psychiatric, autonomic, and functional impairments that extend far beyond traditional disease categories. Parkinson's disease dementia exemplifies the multidimensional nature of modern chronic illness, where cognition, mobility, caregiver burden, mental health, medication complexity, and quality of life become inseparable components of care. As populations age across the Middle East, healthcare systems will require stronger integration between family medicine, geriatrics, neurology, psychiatry, rehabilitation, and community support services.

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Collectively, these papers demonstrate that family medicine today sits at the intersection of multiple global transitions:

- The epidemiologic transition toward chronic disease
- The demographic transition toward aging populations
- The technological transition driven by artificial intelligence
- The behavioral transition involving new forms of addiction and digital dependency
- The healthcare transition from episodic treatment toward longitudinal integrated care.

Importantly, these transitions are occurring simultaneously in the Middle East, often at accelerated speed. Rapid urbanization, changing lifestyles, digital expansion, increasing life expectancy, and rising chronic disease prevalence are placing substantial pressure on healthcare systems that were historically designed around acute episodic care models.

In this environment, the value of family medicine becomes increasingly evident. No other discipline is positioned as broadly across prevention, chronic disease management, mental health, geriatrics, behavioral medicine, community health, and continuity of care. Yet the expanding scope of primary care also raises important questions regarding workforce capacity, training, burnout, and healthcare system design.

The future family physician will require competencies extending far beyond traditional biomedical expertise alone. Digital literacy, behavioral intervention skills, chronic disease coordination, geriatric care, mental health integration, preventive medicine, and interdisciplinary collaboration will become progressively more important. Equally essential will be the preservation of humanistic medicine—listening, empathy, contextual understanding, and continuity of relationships—in an increasingly technological healthcare environment.

These papers collectively remind us that medicine remains fundamentally about people rather than diseases alone. Whether addressing tobacco addiction, chronic metabolic illness, pediatric urticaria, AI-mediated psychological influence, or neurodegenerative dementia, the central task of family medicine remains unchanged: providing comprehensive, compassionate, patient-centered care across the lifespan.

As healthcare continues evolving, family medicine must not merely adapt to these changes but actively lead them. The future of effective healthcare delivery—particularly in the Middle East—will depend heavily on the ability of family medicine to integrate prevention, technology, chronic disease management, mental health, and aging care into coherent and sustainable models of practice.

Warm regards,
Dr. Abdulrazak Abyad
Editor-in-Chief
MEJFM

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Prevalence and Pattern of Smoking among Primary Health Care Patients

Almoutaz Alkhier Ahmed [1], Nazar Khalid Zarroug [2], Mohamed Elfadel Abbas [3], Samer Mohammed Syed [4], Mohammed Abd Elmoneim Alyas [5], Gais Mohamed Osman [6], Khalid Mohydin [7], Aisha Abdulrahman Alzarooni [8]

(1) Dr Almoutaz Alkhier Ahmed, A/Professor -MBRU, SSR family medicine -Nad Alhamar Health Center. DAHC
(2) Dr Nazar Khlid Zarroug, SR family Medicine – Nad Alhamar Health Center – DAHC
(3) Mohamed Elfadel Abbas, SR family Medicine - Nad Alhamar Health Center – DAHC
(4) Dr Samer Mohammed Syed, SR family medicine -Nad Alhamar Health Center – DAHC
(5) Dr Mohammed Abd Elmoneim Alyas, SR family medicine -Nad Alhamar Health Center – DAHC
(6) Dr Gais Mohamed Osman, SR family medicine -Nad Alhamar Health Center – DAHC
(7) Dr Khalid Mohydin, SR family medicine -Nad Alhamar Health Center – DAHC
(8) Dr Aisha Abdulrahman Alzarooni, Consultant Family Medicine, Head of Nad Alhamar Health Center – DAHC

Correspondence:
Almoutaz Alkhier Ahmed Abdulrahman
A/clinical professor of Family Medicine – MBRU. FRCGP (INT), MSc diabetes, MSc endocrinology, MSc Health Economy,
MPH,Dip IBLM, FASLM, SFIDF, LFOM. Senior Family Medicine Specialist, Nad Alhamar Health Centre
Dubai Academic Health Corporation.
Email: Khier2@yahoo.com

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Abstract

Introduction: Smoking continues to pose a major public health challenge in the Eastern Mediterranean Region, with rising prevalence and increased use of alternative tobacco products.

Objective: This study aimed to determine the prevalence and patterns of tobacco use, identify the typical age of initiation, and evaluate the association between tobacco use and chronic non-communicable diseases among adults visiting a primary health care centre in Dubai.

Method:
A cross-sectional descriptive study was conducted in January 2026 among adults aged ≥18 years or older at Nad Alhamar Health Centre one of Dubai Academic Health Corporation's primary health care centres.
Results: A total of 324 participants with complete smoking assessment data were randomly selected. The prevalence of smoking was 33.6%, predominantly among males (96.4%, p<0.005). The mean age of smokers was 34.2±14 years, with an average age of initiation of 18.2±4.2 years. Cigarettes were the most common form of tobacco use (44.9%), followed by vaping (22%) and Medwakh (18.3%). Smoking was associated with several chronic non-communicable conditions, including diabetes (11.9%), hypertension (17.4%), ischemic heart disease (9.2%), and chronic kidney disease (0.9%), with some individuals having multiple comorbidities.

Conclusion: The findings show a marked trend in smoking prevalence and its association with non-communicable diseases among adults in this setting. These results illustrate the importance of carrying out targeted preventive measures and smoking cessation interventions.

Key words:
Smoking, Cigarettes, Medwakh, Water pipes.

Introduction

Tobacco use continues to be one of the most significant contributors to global morbidity and mortality. It accounts for approximately eight million deaths each year (1). It remains a critical public health challenge. This is especially true in low- and middle-income countries, where more than 80% of the world's smokers reside, according to the World Health Organization (2). These regions often face additional barriers, including limited access to healthcare, lower levels of health awareness, and weaker tobacco control policies. All these factors contribute to the persistence and growth of tobacco consumption.

In recent years, patterns of tobacco use have evolved, especially in the Eastern Mediterranean Region (EMR). One notable trend is the increasing popularity of water pipe smoking, also known as shisha or hookah. It is often mistakenly perceived as a safer alternative to cigarette smoking (3). This misconception has fuelled its widespread adoption, particularly among young adults and adolescents. However, research has shown that water pipe smoking carries many of the same health risks as traditional cigarette use. These include exposure to toxic chemicals, carcinogens, and addictive nicotine.
The health consequences of tobacco use are extensive and well-documented. Smoking is strongly associated with a wide range of non-communicable diseases. These include various forms of cancer, cardiovascular diseases such as heart disease and stroke, and chronic respiratory conditions like chronic obstructive pulmonary disease (COPD) (4). Beyond physical health, tobacco use is also linked to mental health issues. These include nicotine dependence, anxiety disorders, and depression. Together, these factors further increase its overall burden on individuals and healthcare systems.

Within EMR countries, smoking prevalence shows considerable variation across populations. Among men, rates can reach 50%. Among women, they may be around 10%. This difference reflects both cultural norms and differences in social acceptability (5). Alarmingly, tobacco use is increasingly being observed among younger age groups. This shift in behaviour could lead to long-term public health challenges. The growing availability and marketing of alternative tobacco products, including electronic cigarettes and vaping devices, have further sped up this trend. These products are often promoted as less harmful or as cessation tools. However, their long-term health effects remain uncertain, raising concerns about both health risks and economic implications.

In the context of the United Arab Emirates, and specifically Dubai, tobacco use remains a pressing issue. A household survey conducted in 2019 reported an overall smoking prevalence of 15.8%. Higher rates were observed among men (20.9%) compared to women (7.9%) (6). These findings highlight the ongoing need for targeted public health interventions, increased awareness campaigns, and stricter regulatory measures. These steps are needed to reduce tobacco consumption and mitigate its associated health risks.

Methodology

A cross-sectional descriptive study design was used to investigate tobacco use patterns among adults. Ethical approval was obtained from the Dubai Scientific Research Ethics Committee (DSREC-04/2026_05).

The sample size was determined using a standard formula for single-proportion studies. Based on findings from previous regional research, the prevalence of tobacco use among the target population was estimated at 15.8%. With a 95% confidence level, a margin of error (absolute precision) of 5%, and a total population size of approximately 1,200 patients, the minimum required sample size was calculated to be 175 patient records. This sample size was deemed sufficient to yield reliable, generalizable estimates within the defined population.
Patient records were selected using simple random sampling with stratification. The study population was individuals attending Nad Alhamar Health Center. Data were taken directly from their medical records. This approach minimized selection bias and ensured subgroup representation.

The inclusion criteria specified that only individuals with a complete tobacco use assessment in their medical records and aged 18 or older were eligible to participate in the study. This ensured that the analysis focused on adult tobacco use patterns, excluding adolescents and children whose behavioural and health profiles may differ significantly.

A range of variables was collected to support a comprehensive analysis. These included demographic characteristics (such as age and gender), types of tobacco use (e.g., cigarettes, water pipes, or other forms), age of smoking initiation, and the presence of associated medical conditions. Collecting these variables allowed for a multidimensional understanding of tobacco use and its potential health implications.

Data analysis was performed using IBM SPSS Statistics (version 26; IBM Corporation, Armonk, NY). All data were coded and entered the software. The primary outcome was the prevalence of current tobacco use, calculated as a percentage of the total sample.

Tobacco use was categorized as current smokers or non-smokers. Non-smokers included ex-smokers and never-smokers. Descriptive statistics were used to summarize data. Categorical variables appeared as proportions and percentages. Continuous variables appeared as means with standard deviations (mean ± SD).

To examine the relationships between tobacco, use and other variables, inferential statistical analysis was performed. The chi-square test was used to assess associations between categorical variables, such as tobacco use status and demographic or clinical characteristics. A p-value of less than 0.05 was considered statistically significant, indicating a meaningful association between variables.

Results

A total of 324 participants met the eligibility criteria (Figure 1) and were included in the analysis. This provided a thorough summary of tobacco use patterns and associated health conditions within the study population. The overall prevalence of smoking was 33.6% (No=109; 4 female, 105 male). This means approximately one in three individuals reported current tobacco use. This relatively high prevalence accentuates the continued public health burden of smoking in the studied group.

A striking gender disparity was observed among smokers. The vast majority were male (96.4% of all smokers), and this association was statistically significant ($P < 0.005$). This finding highlights a strong gender-related pattern in tobacco use, suggesting that smoking continues predominantly a male behaviour in this population, potentially affected by cultural, social, or behavioural factors.

The mean age of smokers was 34.2 years (SD ±14), showing a broad age distribution from young adults to older individuals (Table 1). The average age at which smokers started was 18.2 years (SD ±4.2). Most began during late adolescence or early adulthood, which is a vital developmental period. Early initiation likely causes long-term dependence and a greater risk of persistent disease.

In terms of tobacco products, cigarettes were the most used form, accounting for 44.9% of smokers. Alternative tobacco products were also widely used, reflecting diversification in smoking habits. Vaping devices accounted for 22% of use, followed by medwakh (18.3%), water pipes (11.9%), tobacco pouches (1.8%), and cigars (0.9%) (Table 2). These results indicate that although traditional cigarette smoking remains dominant, newer and culturally specific forms of tobacco use are more common.

Age-specific patterns of tobacco use showed significant differences across groups. Among individuals younger than 20 years, medwakh was the predominant form of smoking, accounting for 40% of use (Table 3). This suggests that medwakh may be especially attractive or accessible to younger users. In the 20–30-year age group, vaping emerged as the most common form (41.3%), showing a shift toward modern alternatives among young adults.

Cigarette smoking became increasingly dominant with advancing age. In the 30–40 age group, 46.4% of smokers used cigarettes (Table 3). This proportion rose further with increasing age: 54.5% in those aged 40–55 years, 50% in the 50–60 age group, and 100% among individuals aged 60 years or older (Table 3). This trend implies that older generations are more prone to adhere to conventional smoking methods, while younger groups diversify their tobacco use.

Smoking was also significantly associated with the presence of non-communicable diseases (39.4%), reinforcing its role as a major health risk factor. Among smokers, 11.9% were diagnosed with diabetes mellitus, while 17.4% had hypertension. Additionally, 9.2% reported ischemic heart disease, and 0.9% had chronic kidney disease (Table 4). These findings highlight the substantial burden of chronic illness within the smoking population.

Importantly, a subset of participants experienced multiple coexisting conditions, reflecting the compounding impact of smoking on health. Specifically, 5.5% of individuals had both type 2 diabetes and hypertension, while 6.4% had a combination of type 2 diabetes, hypertension, and ischemic heart disease (Table 4). These comorbidities significantly increase the risk of complications, healthcare utilization, and mortality.

Smokers had a much higher risk of hypertension and ischemic heart disease, with risk more than doubling and nearly tripling. Smoking showed no significant association with diabetes mellitus or chronic kidney disease, and most combined comorbidities had no clear relationship. Multiple conditions (DM2 + HTN + IHD) showed a borderline increase, but the difference was not statistically significant (Table 4). These findings suggest that, in this sample, smoking mainly increases cardiovascular risk, not metabolic or renal risks (Table 4).

Overall, the findings demonstrate a substantial prevalence of smoking, particularly among males, with early initiation and diverse patterns of tobacco use across age groups. The strong association between smoking and multiple non-communicable diseases further emphasizes the urgent need for targeted prevention and intervention strategies. Efforts should focus on reducing early initiation, addressing emerging tobacco products such as vaping and medwakh, and promoting cessation programs tailored to high-risk groups.

Figure 1: Sample selection

Table 1: Smoking by Age distribution

Age group (year)	Numbers of participants
<20	9.1% (no 10)
20 - 30	37.6% (no 41)
30 - 40	18.3% (no 20)
40 - 50	10.09% (no 11)
50 - 60	6.4% (no 7)
>60	5.5% (no 6)

Table 2: Pattern of smoking

Cigarettes	44.9% (No = 49)
Vapes	22% (No =24)
Water-pipe	11.9% (No +13)
Medwakh	18.3% (No = 20)
Nicotine Pouch	1.8% (No = 2)
Cigar	0.9% (N0 =1)

Table 3: Distribution of smoking pattern per age

Age	Pattern of smoking	Percent
<20 year	Medwakh	40%
20 – 30 years	Vape	41.3%
30 – 40 years	Cigarette	46.4%
40 – 50 years	Cigarette	54.5%
50– 60 years	Cigarette	50%
>60 years	Cigarette	100%

Table 4: Associated Non-communicable Chronic Diseases (Diabetes mellitus type 2, Hypertension, Ischaemic heart disease and chronic kidney diseases) in smoker vs non-smokers

	Smoker (no 109)	Non-smoker (no 215)	P value	Relative Risk
Diabetes mellitus type 2 (DM2)	11.9%	12.5%	0.43	0.95 (CI 0.5 -1.8)
Hypertension (HTN)	17.4%	7.9%	**0.005**	**2.2 (CI 1.9 – 4.06)**
Ischemic heart disease (IHD)	9.2%	7.9%	**0.01**	**2.9 (CI 1.2-7.5)**
Chronic kidney disease (CKD)	0.92%	0.93%	0.49	0.98 (CI 0.09 – 10.75)
DM2+HTN	5.5%	5.1%	0.43	1.07 (CI 0.4 – 2.8)
DM2+HTN+IHD	6.4%	2.7%	0.051	2.3 (CI 0.8 – 6.7)
DM2+HTN+IHD+CKD	0	0.04%	0.41	0.39 (CI 0.2-8.1)

Discussion

Despite widespread access to tobacco cessation clinics in Dubai and the United Arab Emirates (UAE), smoking prevalence has not significantly declined. This persistent rate indicates that, beyond available cessation services, entrenched social, cultural, and product-specific factors continue to fuel tobacco use. Synthesizing two decades of epidemiological research in the UAE reveals concerning trends in usage rates, demographic patterns, and the evolving landscape of tobacco consumption.

One of the earlier studies conducted by Obaid et al. (8) investigated tobacco use among school students aged 10 to 20 years in Dubai. Their findings revealed an overall smoking prevalence of 14.6%, with cigarette smoking accounting for 11.2% and waterpipe (shisha) use at 2.2%. This study provided an early indication that tobacco use begins at a relatively young age, with cigarettes being the dominant form of consumption among adolescents. However, the presence of waterpipe smoking, even at lower levels, signalled the beginning of diversification in tobacco use behaviours among youth.

Subsequent research by Crooks and Wolff (9), which examined 399 adolescents with a mean age of 16.9 years, reported a higher smoking prevalence of 23.4%. This marked increase compared to earlier findings suggests either a rise in tobacco use among youth or differences in study populations and methodologies. Regardless, the data underscores the concerning level of tobacco exposure among adolescents in the UAE and highlights the need for targeted prevention strategies at the school and community levels.

Aden et al. (10) evaluated individuals applying for premarital screening programs in Abu Dhabi and found a smoking prevalence of 24.7%. Cigarette smoking was most common (11.5%), followed by Medwakh (5.9%), waterpipe (4.8%), and combined use (2.5%). The study highlighted significant gender disparities; males had much higher rates (19.2%) than females (3.5%),reflecting regional sociocultural norms. It also emphasized the cultural specificity of Medwakh use in the UAE.

A large cross-sectional survey by Al-Houqani, Ali, and Hajat (11) further expanded understanding of smoking behaviours in the UAE. They found higher smoking rates in males (24.3%) compared to females (0.8%), with the highest prevalence among males aged 20 to 39. Cigarette use dominated (77.4%), with Medwakh, waterpipe, and cigars also used, illustrating the range of tobacco products favoured in the UAE.

In a study focusing specifically on younger populations, Al-Shemmari, Shaikh, and Sreedharan (12) examined Dokha use among secondary school students in Ajman. Their results revealed that 24% of participants were current users of Dokha, while 39% had ever smoked cigarettes and 36% had ever used Dokha. These findings are particularly alarming, as they indicate high levels of experimentation and current use among adolescents. The popularity of Dokha, in particular, may be attributed to misconceptions about its safety, ease of access, and cultural acceptance. This highlights the need for public health interventions that address not only cigarette smoking but also alternative forms of tobacco use that are gaining traction among youth.

More recent data from Alareesi et al. (13), who studied 500 patients attending primary healthcare clinics in Dubai, reported a smoking prevalence of 23.6%. This study also identified the average age of smoking initiation as 18 to 19 years, which aligns with earlier findings and suggests that late adolescence and early adulthood remain key periods for the onset of smoking behaviour. The consistency of this initiation age across studies indicates a persistent window of vulnerability that could be targeted through preventive interventions.

Examining these studies collectively reveals a key challenge: smoking prevalence in Dubai has increased over time despite expanded cessation services. For example, prevalence rose from 14.6% in 2010 (Obaid et al.) to 23.6%

in 2020 (Alareesi et al.). This approximate 9% increase underscores the inadequacy of current strategies, likely due to changing product preferences and persistent social dynamics. The ongoing rise in tobacco use, despite policy and service improvements, demands reassessment of intervention approaches.

Conclusion

In conclusion, while the UAE has made significant investments in tobacco cessation infrastructure, the persistence and apparent increase in smoking prevalence indicate that additional, multifaceted approaches are needed. These should include stronger public health policies, enhanced education and awareness campaigns, stricter regulation of emerging tobacco products, and culturally sensitive interventions that address the underlying social determinants of smoking. Without such comprehensive efforts, the burden of tobacco-related diseases is likely to continue rising, posing a significant challenge to public health in Dubai and the wider UAE. This review and synthesis of tobacco use studies in Dubai, and the United Arab Emirates has several limitations that should be considered when interpreting the findings. First, the included studies vary considerably in their design, study populations, sampling methods, and time periods, which limits the ability to directly compare prevalence estimates across studies. Differences in age groups (school students, university students, and adults attending healthcare facilities) introduce heterogeneity that may influence reported smoking rates and patterns of tobacco use.

Second, most of the data are based on self-reported questionnaires, which are subject to recall bias and social desirability bias. This is particularly relevant in the UAE context, where smoking among females may be underreported due to cultural sensitivities and social stigma. As a result, actual prevalence rates, especially among women, may be higher than reported.

Third, many studies are cross-sectional, limiting the ability to establish causal relationships or to assess changes in individual smoking behaviours over time. While trends can be inferred from repeated cross-sectional studies, they do not provide longitudinal evidence of smoking initiation, cessation, or relapse patterns.

Finally, some studies have relatively small or non-representative sample sizes, particularly those conducted in specific settings such as schools or primary care clinics, which may limit generalizability to the broader UAE population.

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The ABCDEF Ultra Violet S: A Family Medicine Guide for Comprehensive Chronic Disease Management

Hassan Yousef[1], Ebrahim Ebrahim[2], Ahmed Salem[3]

1 Senior Consultant Family Medicine, Primary Health Care Corporation, Doha, Qatar
2 Specialist Family Medicine, Primary Health Care Corporation, Doha, Qatar
3 Family Medicine Chief Resident, Hamad Medical Corporation, Doha, Qatar

Corresponding author
Ebrahim Ebrahim
Specialist Family Medicine, Primary Health Care Corporation,
Doha, Qatar
Email: dr.ibrahim.ibrahim@gmail.com

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Abstract

The *ABCDEF Ultra Violet S: a Family Medicine Guide for Comprehensive Chronic Disease Management* is an evidence-based, checklist-driven framework designed to close critical gaps in chronic disease management within primary care. Grounded in international and regional guidelines, the tool integrates preventive strategies across metabolic, cardiovascular, mental health, cancer, and vaccination domains into a single, practical format for busy family medicine clinics. Its dual-purpose design supports clinicians by reducing cognitive load and standardizing care delivery, while empowering patients through education and shared decision-making. Expected outcomes include improved adherence to screening protocols, earlier detection of complications, enhanced patient engagement, and cost savings through prevention. Implementation requires provider training, stakeholder engagement, and iterative refinement, supported by quality audits and continuous improvement initiatives. This guide reflects the core principles of family medicine, comprehensive, patient-centered, and quality-driven care, and offers a scalable model for transforming chronic disease management in Qatar and beyond.

Keywords: Diabetic Care, Diabetes Mellitus, Non-communicable Diseases, Hypertension, Chronic Kidney Diseases, Retinopathy, Neuropathy, Nephropathy

Introduction

Family medicine clinics serve as the cornerstone of healthcare delivery, particularly for patients managing chronic conditions such as diabetes, hypertension, cardiovascular disease, and chronic kidney disease. These conditions, collectively referred to as Cardiovascular-Kidney-Metabolic (CKM) disorders, require ongoing monitoring, preventive interventions, and patient education to prevent complications and optimize outcomes (1). However, the reality of clinical practice often falls short of the ideal. Physicians face significant time pressures, with the average primary care visit lasting only 10 to 15 minutes, during which they must address acute concerns, manage multiple chronic conditions, provide preventive care, and educate patients. This demanding environment creates a fertile ground for care gaps, where important screening tests, vaccinations, or patient counseling may be inadvertently omitted.

Significant percentage of deaths are attributed to behavioural and preventable causes, many of which could be addressed through systematic preventive care in primary care settings (2). Patients with chronic diseases represent a substantial share of healthcare spending, with a large portion of these costs stemming from complications that could have been prevented (2). The challenge is not a lack of knowledge about what should be done, but rather the absence of a reliable system to ensure that all necessary care elements are consistently delivered in every patient encounter. In the local context of Qatar, no clear data are published on expenditure on chronic diseases but is expected to be similar to high income countries with large burden.

In this article we present a model that provides a comprehensive approach to care of patients with chronic disease in primary care clinics and general medicine clinics. Components of this tabular model are supported by narrative literature review of evidence from well-known clinical societies and published studies.

Care Areas

The following table summarizes care areas across all categories of the ABCDEF-UVS guide:

	Medical Intervention	Relevant guidance	Reference(s)
A	HbA1c Testing	**Diabetes**: ≥ 2/year if stable; every 3 months if therapy changed or not at goal; consider more frequent per clinical judgement; **non-diabetes:** screen every 3 years if at risk; annually if prediabetes or history of gestational diabetes; avoid if assay accuracy affected.	(3–9)
	Aspirin Therapy	**Primary prevention**: consider **age 40–59** with ≥10% 10-yr CVD risk & low bleeding risk; **avoid initiating ≥60**; **individualize** in diabetes; **secondary prevention**: indicated.	(10–16)
B	Blood Pressure Monitoring	**Measure at every visit**; confirm elevated readings; **home/ambulatory monitoring** for HTN/white-coat/masked; **targets <130/80** for diabetes/CKD/CAD; follow-up **monthly during titration**, then **q3–6 months** when controlled.	(17–25)
	BMI Documentation	**Height/weight & BMI annually**; **q3 months** during active weight management; consider **waist circumference.**	(25–29)
C	CVD Risk Assessment	Documented in only **5-16%** of eligible patients. **59%** of physicians do not calculate it routinely. Use **PREVENT** (or validated tool) at baseline; **repeat q4–6 years** (age 40–75) or with risk change; **annual risk factor review** in diabetic patients.	(30–37)
	Cholesterol Screening	**Adults with diabetes**: lipid profile at diagnosis/initial visit; **annually** thereafter; **4–12 weeks** after statin start/dose change, then **annually**; **pediatrics**: initial post-glycemic optimization, **annual**, treat if LDL >130 after lifestyle. **30-37%** of patients miss recommended lipid screening.	(17,38,39)
D	Depression & Anxiety Screening	**At initial visit and at least annually**; more often with history or triggers; tools: PHQ-9, GAD-7, WHO-5, PAID.	(25,40–46)
	Dental assessment	**Annually in DM, HTN, dialysis/transplant patients,** semiannual in uncontrolled/poor control patients, consider pre-procedural for CVD procedures.	(41,47–57)
E	Erectile Dysfunction & Genitourinary symptoms in females	**Proactive inquiry routinely**; use **SHIM**; initial labs (**AM testosterone, glucose, lipids**); assess CVD risk; screen for bladder dysfunction when indicated and screen for other urinary symptoms.	(58–62)
	Electrocardiogram	Baseline for HTN, consider for DM & CKD Annually for HTN, consider according to risk and stage for other chronic conditions	(8,17,20,63–65)

	Medical Intervention	Relevant guidance	Reference(s)
F	Diabetic Foot Exam	**Comprehensive annual exam**; **every visit** if LOPS/prior ulcer/amputation; risk-stratified follow-up: 12 mth (very low), 6–12 mth (low), 3–6 mth (moderate), 1–3 mth (high); self-care education.	(66–71)
	Fundus/Eye Exam	**T1D**: first **within 5 years** of diagnosis; **T2D**: **at diagnosis**; **q1–2 years** if no retinopathy & good control, else **annually** or more often; pregnancy: **pre-conception/1st trimester & as indicated**; validated retinal imaging is an acceptable option. Fundus examination is only indicated when end-organ damage is expected in patients with HTN and despite the high risk of ophthalmic conditions in patients with CKD, screening is individualized as per clinical need..	(25,38,66,72–75)
	FIB-4 Score (Liver)	**At diagnosis/initial evaluation** for T2D/prediabetes/CM risk; **repeat q1–2 years** if low risk; if **FIB-4 ≥1.3**, do elastography/ELF; **>2.67**: refer hepatology	(41,76–80)
	Heart Failure Screening (Pro BNP)	**Baseline at diagnosis** (T2D; T1D after ~5 yrs) and **annually**; if NT-proBNP ≥125 pg/mL consider echocardiography.	(17,81–86)
U	Urine albumin creatinine ratio	**Annually**: T2D at diagnosis; T1D starting **5 years** post-diagnosis; **1–4×/year** if CKD; confirm abnormalities within **3–6 months**; **q3–6 months** if worsening or after therapy change	(7,8,87,88)
V	Influenza Vaccine	**Annually (inactivated)** for all with diabetes/CM conditions	(41,89–91)
	Pneumococcal Vaccine	**Age 19–64**: **PCV15 or PCV20** (if PCV15 → **PPSV23** ≥1 yr later); **≥65**: **PCV15 or PCV20** with same follow-up.	(25,41,90)
	Hepatitis B Vaccine	**Universal <60 yrs**; **≥60 yrs**: consider by risk	(41,90)
	HZV, Tdap Vaccine	**Recombinant HZV (shingles) 2 doses** for **≥50 yrs**; **Tdap** booster **every 10 yrs**	(25,41,90)
	RSV	**≥60 yrs**: **single RSV vaccine** particularly those with congestive heart failure, chronic obstructive lung disease, diabetes mellitus, chronic kidney disease. Give around late summer, early autumn.	(41,92–97)

	Medical Intervention	Relevant guidance	Reference(s)
	General Screening	Patients with chronic diseases have consistently **lower cancer screening rates**. Follow **age/sex-appropriate** national guidelines in coordination with PCP.	(41)
	Breast, Cervical, Colorectal	**CRC**: start **45–75 yrs** (FIT yearly; mt-sDNA q3 yrs; colonoscopy q10 yrs; CT colonography q5; flex sig q5); **Breast**: **annual 45–54**, then **biennial ≥55** (or annual); **Cervical**: **Pap q3 yrs (21–29)**; **HPV+Pap q5 yrs (30–65)**	(98–106)
S	Osteoporosis	**DXA ≥65 yrs** (women) & **≥70 yrs** (men) or earlier with risk; **monitor q2–3 yrs**; consider diabetes-specific risks. Qatar national guidelines recommend locally calibrated FRAX tool (Q-FRAX) in postmenopausal women and men aged ≥50 years, with particular emphasis on identifying high fracture risk among patients with chronic conditions where DXA scanning is reserved for those classified as high-risk by Q-FRAX.	(41,107,108)
	Sleep apnea	**Annual screening** in high-risk adults (T2D, obesity, resistant HTN, etc.) using **STOP-Bang/Epworth**; evaluate symptoms; integrate into cardio-metabolic pathways	(25,41,91,109–113)

BMI: body mass index, CVD: cardiovascular disease, PHQ-9: patient health questionnaire, GAD-9: Generalized anxiety disorder questionnaire, SHIM: sexual health inventory for men, LOPS: loss of protective sensation, PCV: pneumococcal vaccine, HZV: herpes zoster virus, RSV: respiratory syncytial virus, Tdap: tetanus, diphtheria, and acellular pertussis, colorectal cancer, DXA: dual energy x-ray absorptiometry, FRAX: fracture risk assessment tool.

3. Chronic Disease Care

Extensive research across multiple countries and healthcare systems has documented significant and consistent gaps between evidence-based clinical guidelines and the care delivered to patients with chronic diseases. These gaps are not merely academic concerns; they represent missed opportunities to prevent disease progression, reduce complications, and improve quality of life. The following analysis presents chronic disease management care plan of the key domains addressed by the ABCDEF-UVS mnemonic guide.

3.1 HbA1c Testing:
Glycemic control is the cornerstone of diabetes management, yet adherence to recommended HbA1c testing is alarmingly low. A comprehensive five-year retrospective cohort study in Australian general practice found that the overall median HbA1c testing frequency was only 1.6 tests per year, with an adherence rate of just 50% to guideline-recommended testing intervals.(3) This means that half of diabetic patients are not receiving the monitoring necessary to guide treatment adjustments. In some populations, the situation is more deficient. Adherence to recommended HbA1c monitoring among patients with diabetes in Saudi Arabia has shown similar suboptimal range. Studies report that less than half of patients undergo annual HbA1c testing, with compliance rates ranging from approximately 12% to 39% across different regions (4,5). The clinical implications are significant: patients with low adherence to HbA1c testing showed persistently elevated glucose levels over time, while those with high adherence achieved better glycemic control and had a significantly lower risk of developing chronic kidney disease.

The American Diabetes Association (ADA) recommends that for patients with diabetes mellitus, hemoglobin A1C should be measured at least twice yearly in those meeting treatment goals and with stable glycemic control, and at least quarterly (every 3 months) in patients whose therapy has changed or who are not meeting glycemic goals. More frequent testing may be warranted based on clinical judgment and individual circumstances, such as hospital admission without a recent A1C result (6–8). The rationale is that A1C reflects average glycemia over 2–3 months and is strongly linked to diabetes complications; regular monitoring allows for timely adjustments in therapy and assessment of long-term glycemic control (6,7).

For patients with chronic diseases other than diabetes mellitus, the ADA does not recommend routine A1C testing unless there is a clinical suspicion of diabetes or prediabetes. In individuals with risk factors or conditions associated with insulin resistance (e.g., obesity, cardiovascular disease, hypertension, metabolic-associated steatotic liver disease), screening for diabetes or prediabetes should be considered, with repeat testing at least every 3 years if results are normal, and annually for those with prediabetes (9). The frequency should be individualized based on risk status and initial results, as there is no consensus for routine A1C monitoring in non-diabetic chronic disease populations (9). A1C testing is not recommended for individuals with conditions that affect red blood cell turnover or hemoglobin variants, as these can interfere with assay accuracy (6).

3.2 Aspirin Therapy:
The ASCEND trial in patients with diabetes but no established cardiovascular disease showed that aspirin modestly reduced serious vascular events but significantly increased major bleeding, resulting in a net neutral benefit for most patients (10). Similar findings were reported in the general population, with ARRIVE and ASPREE trials demonstrating little or no net benefit for primary prevention due to increased bleeding risk, particularly in older adults (11,12).

For other chronic conditions such as hypertension or stable coronary artery disease without prior events, the USPSTF recommends considering aspirin for primary prevention only in adults aged 40–59 years with a ≥10% 10-year CVD risk and low bleeding risk, and not initiating it in those aged 60 or older (13). The overall benefit is small, and shared decision-making is emphasized.

Sex differences have been explored, with some evidence suggesting aspirin may reduce myocardial infarction risk in men and stroke risk in women. However, the benefit for primary prevention in women with diabetes remains inconclusive (16). Meta-analyses confirm that any benefit is generally limited to those at higher cardiovascular risk, and the risk of gastrointestinal and major bleeding events must be carefully weighed (14,15).

In summary, aspirin for primary prevention in diabetes and other chronic non-communicable diseases should be individualized, focusing on patients with elevated cardiovascular risk and low bleeding risk, and generally avoided in older adults or those at increased bleeding risk. For secondary prevention, the benefit of aspirin remains well established (10,12,13).

3.3 Blood Pressure Monitoring:
In Qatar and the region, and despite the confidence of primary care physicians in the management of hypertension, a marked deficiency exists in the frequency and target achievement with less than one third achieving the target blood pressure (114–116). The ADA advises measuring blood pressure at every routine visit for patients with diabetes and other chronic non-communicable diseases, confirming elevated readings (systolic 120–129 mmHg and diastolic <80 mmHg) with multiple measurements before diagnosing hypertension (17,18). Home blood pressure monitoring is recommended for patients with diabetes and hypertension to detect white-coat or masked hypertension, while orthostatic measurements should be performed initially and periodically in those with symptoms or autonomic dysfunction risk (17–19). Similar principles apply to other chronic conditions, with routine checks at clinical visits and individualized frequency based on risk factors and treatment changes (17). The American Heart

Association (AHA) and American College of Cardiologists (ACC) recommend annual screening for adults at increased risk and more frequent monitoring for those with hypertension or elevated readings, with office visits every 3–6 months once goals are achieved and monthly follow-up during medication titration (20,117). Home monitoring and ambulatory monitoring are strongly encouraged for high-risk patients,(20,22,25) and accurate technique with standardized protocols remains essential (22,25).

The ACC/AHA recommends a blood pressure target of <130/80 mm Hg for adults with diabetes, CKD, CAD, and hypertension to reduce cardiovascular events, CKD progression, and mortality (20,24). Evidence from trials such as ACCORD and BPROAD supports intensive control, though ACCORD did not show added benefit for a systolic target <120 mm Hg compared to <140 mm Hg, leading to the current recommendation of <130/80 mm Hg as a balance between benefit and risk, with individualization for frailty or intolerance (20). For CKD, SPRINT and meta-analyses support a systolic target <130 mm Hg, and ACE inhibitors or ARBs are preferred in patients with albuminuria ≥30 mg/g (20). For CAD, the same <130/80 mm Hg target applies, with nonpharmacologic strategies as first-line therapy and guideline-directed medications for compelling indications, while caution is advised to avoid diastolic BP <60 mm Hg in older adults or those with diabetes (23,24). Overall, intensive BP lowering reduces stroke, heart failure, coronary events, and mortality, with adverse effects being infrequent when monitored appropriately, and targets should be individualized for patients with limited tolerance or life expectancy (20) .

3.4 Body Mass Index (BMI) Documentation:
BMI should be assessed and documented at least annually in all patients with diabetes and other chronic non-communicable diseases, and more frequently (every 3 months) during active weight management or when clinical concerns arise (26). The ADA emphasizes annual height and weight measurement to calculate BMI, with increased frequency during intervention or instability (26). While BMI is a useful screening tool for overweight and obesity, it should not be the sole measure; additional assessments such as waist circumference are recommended, particularly in populations with varying body composition and cardiometabolic risk (25,27,28). The American Association of Clinical Endocrinology advises confirming BMI through physical examination to exclude confounders like muscle mass or edema and recommends waist circumference as a marker of cardiometabolic risk (25). Documentation should be sensitive and nonjudgmental, using person-first language and explaining the clinical value of BMI assessment (26).

For patients with other chronic non-communicable diseases, routine BMI assessment is similarly recommended to guide weight management and monitor outcomes (24,28). Clinicians should consider racial and ethnic differences in BMI thresholds, as some populations experience increased cardiometabolic risk at lower BMI values (25,29). These recommendations aim to ensure accurate assessment, reduce stigma, and support individualized care.

Comorbidity	Screening Method / Diagnostic Criteria
Asthma / Respiratory Disease	History, physical examination; spirometry as indicated
Diabetes	Fasting plasma glucose ≥126 mg/dL; HbA1c ≥6.5%; 2-hour oral glucose tolerance test
Dyslipidemia	Lipid panel: triglycerides, HDL-C, LDL-C, total cholesterol, non-HDL-C
Gastroesophageal Reflux Disease	History; endoscopy as indicated
Hypertension	Sitting blood pressure ≥130/80 mm Hg
Metabolic Syndrome	≥3 of: waist circumference ≥88 cm (women) or ≥102 cm (men); triglycerides ≥150 mg/dL; fasting glucose ≥100 mg/dL; BP ≥130/85 mm Hg; HDL-C <40 mg/dL (men) or <50 mg/dL (women)
Nonalcoholic Fatty Liver Disease	Liver function tests; Fibrosis-4 Index calculation; imaging
Obstructive Sleep Apnea	Neck circumference: screening questionnaires (e.g., STOP-BANG); polysomnography as indicated
Osteoarthritis	History; physical examination (weight-bearing joints); radiography as indicated
Prediabetes	Fasting plasma glucose 100–125 mg/dL; HbA1c 5.7–6.4%; 2-hour oral glucose tolerance test

3.5 CVD Risk Calculation and Electrocardiogram:
Atherosclerosis cardiovascular disease risk estimation is an essential component of cardiovascular prevention in patients with diabetes mellitus, hypertension, chronic kidney disease, and other chronic non-communicable diseases. Baseline assessment should be performed at the initial evaluation using validated tools such as the PREVENT risk calculator, and repeated every 4–6 years in adults aged 40–75 years, or more frequently if new risk factors develop or clinical status changes (30,31). For adults with diabetes, annual risk factor assessment is recommended, and PREVENT can be used to estimate 10-year risk of ASCVD, heart failure, and total CVD, incorporating kidney function and metabolic parameters when available (30,31). This tool is designed for primary prevention in adults without established CVD and offers a more contemporary and accurate risk estimate than older models (30–32).

PREVENT uses age, sex, blood pressure, cholesterol, smoking status, diabetes, eGFR, and optionally HbA1c and urine albumin-to-creatinine ratio, providing a comprehensive risk profile (30–32). A 10-year predicted CVD risk of ≥7.5% defines increased risk and guides decisions on statin and antihypertensive therapy, consistent with ACC/AHA guidelines (31,33,34). Compared to the Pooled Cohort Equations (PCE), PREVENT generally produces lower risk estimates, which may influence treatment eligibility thresholds (31,33,34).

In Qatar and the GCC region, utilization of ASCVD risk calculators remains low and inconsistent. Most clinicians rely on Western tools such as PCE, SCORE2, WHO/ISH, or QRISK3, but systematic integration into routine practice is limited, and region-specific models are rarely used (35–37). Studies highlight poor agreement between calculators and emphasize the need for locally validated tools. In Qatar, risk calculators are recommended but not consistently implemented, with gaps in clinician awareness and compliance to documentation in the electronic health record (35–37). Improved adoption and regional validation of PREVENT and similar tools are essential to optimize CVD prevention strategies.

Electrocardiogram (ECG) screening plays an important role in chronic disease care particularly those with marked association with cardiovascular complications. For hypertension, the 2025 ACC/AHA guideline recommends obtaining a baseline ECG in all adults as part of the initial diagnostic evaluation, since it provides valuable information on subclinical cardiovascular disease such as left ventricular hypertrophy (20). ECG may then be repeated annually, or sooner if clinical changes occur, to monitor progression of target organ damage and evolving cardiovascular risk (20). In diabetes, a resting ECG is reasonable for CVD risk assessment in asymptomatic adults, but routine annual screening is not supported; instead, ECG should be performed at diagnosis and repeated only if symptoms or abnormal findings arise (17,63,118,119). Advanced cardiac testing, including exercise ECG, may be considered in adults ≥40 years with suspected cardiac or vascular disease (17).

For chronic kidney disease (CKD), while no specific guideline mandates routine baseline ECG, it remains clinically useful for detecting left ventricular hypertrophy, atrial fibrillation, and other arrhythmias, which are common across CKD stages (64,65). The frequency of cardiovascular monitoring in CKD should be guided by disease stage and albuminuria, ranging from once yearly in stage G3 to every 1–3 months in stage G5, with ECG integrated into this framework given the high cardiovascular burden (8). Together, these recommendations highlight the importance of tailoring ECG screening to disease-specific risks, ensuring timely detection of subclinical cardiovascular disease while avoiding unnecessary routine testing.

3.6 Cholesterol Screening:
The ADA recommends that in adults with diabetes mellitus or prediabetes not taking lipid-lowering therapy, a lipid profile should be obtained at diagnosis, at the initial medical evaluation, and annually thereafter, or more frequently if clinically indicated. For those starting statin or other lipid-lowering therapy, a lipid profile should be checked at initiation, 4–12 weeks after starting or changing dose, and then annually to monitor response and adherence (17).

For children and adolescents with diabetes, lipid screening should be performed initially after glycemic optimization and then annually. If lipids are abnormal, initial management focuses on optimizing glycemia and dietary intervention, with statin therapy considered if LDL cholesterol remains >130 mg/dL after 6 months of lifestyle changes. The treatment goal for LDL cholesterol in youth is <100 mg/dL, HDL cholesterol >35 mg/dL, and triglycerides <150 mg/dL (38).

Treatment targets for adults with diabetes and established ASCVD are an LDL cholesterol goal of <70 mg/dL, with high-intensity statin therapy recommended. If this target is not achieved, addition of ezetimibe or a PCSK9 inhibitor may be considered. For primary prevention in adults aged 40–75 years, moderate-intensity statin therapy is recommended, with high-intensity statin preferred for those at higher risk. Statin therapy may also be reasonable in adults <40 or >75 years based on individual risk assessment (39).

For patients with other chronic non-communicable diseases, the ADA and American College of Cardiology/American Heart Association recommend similar screening and treatment targets, emphasizing annual lipid assessment and individualized statin therapy based on ASCVD risk (17,39).

3.7 Depression and Anxiety:
The ADA recommends at least annual screening for depression in individuals with diabetes using validated tools such as the Patient Health Questionnaire-9 (PHQ-9), with more frequent screening for those with a history of depression (40). Anxiety screening is also advised, particularly for patients at risk of hypoglycemia or with

fear of hypoglycemia (40). Screening should occur at the initial visit and annually thereafter, as well as during key transitions such as diabetes diagnosis, hospitalizations, treatment changes, or when glycemic targets are not met (40,41). Additional triggers include the diagnosis of complications or significant changes in medical status. The American Association of Clinical Endocrinology further recommends screening during each clinic encounter when appropriate (25).

Validated instruments for mental health screening include the PHQ-9 for depression, Generalized Anxiety Disorder-7 (GAD-7) for anxiety, WHO Wellbeing Index (WHO-5), and the Problem Areas in Diabetes Scale for diabetes distress (25,40,42). Despite these recommendations, screening rates remain suboptimal globally and regionally. Studies from the GCC region show a prevalence of depression among patients of T2DM ranging between 5.21%-21.1% (46,47,48). However, these studies primarily report prevalence rather than systematic implementation of screening protocols.

Globally, depression affects approximately 35% of individuals with diabetes, impacting nearly one in four patients with type 1 or type 2 diabetes (40,42,46). Anxiety and diabetes distress are also common and associated with poor glycemic control, reduced adherence, and increased complications. These findings underscore the importance of embedding routine mental health screening into chronic disease management pathways. Early identification and intervention can improve quality of life, treatment adherence, and clinical outcomes, making mental health care a critical component of comprehensive diabetes and chronic disease management.

3.8 Dental assessment:

Dental screening is essential in chronic diseases because periodontitis and other oral complications are strongly linked to systemic outcomes. In diabetes, the ADA recommends at least annual dental exams, as periodontal disease is more severe and prevalent, worsens glycemic control, and increases A1C levels; semiannual screening may be warranted in poorly controlled patients (41,47,50). In hypertension, periodontitis is independently associated with uncontrolled blood pressure, and annual screening is advised, with more frequent exams for those with poorly controlled hypertension(51–53). In cardiovascular disease, periodontal disease approximately doubles myocardial infarction risk, and consensus guidelines recommend routine dental exams every 6–12 months, plus mandatory screening before elective cardiothoracic or interventional procedures to prevent complications such as infective endocarditis (54,55,120,121).

In CKD, KDIGO advises dental evaluation before transplantation, with annual screening for adults and semiannual exams for dialysis or post-transplant patients due to risks such as enamel defects, calculus accumulation, candidiasis, and gingival overgrowth (48,49,56,57,122). Overall, integrating regular dental screening, annual for most patients and semiannual for high-risk groups, into chronic disease management is critical to reduce systemic complications and improve outcomes.

3.9 Sexual Health and Urinary Dysfunction:

Routine screening for erectile dysfunction (ED) and genitourinary symptoms in men with diabetes hypertension, and CKD is essential, as these conditions are frequently underreported. Expert consensus and recent evidence emphasize the need for proactive physician inquiry, with the American Urological Association recommending clinicians initiate discussions about ED since many men hesitate to raise sexual concerns. Validated questionnaires, such as the Sexual Health Inventory for Men, provide a structured approach to assessment and help quantify symptom severity and treatment response (58). A comprehensive evaluation should include medical, sexual, and psychosocial history, physical examination, and selective laboratory testing. For men presenting with ED, assessment should address cardiovascular risk factors, diabetes control, medication history, and signs of hypogonadism, with morning serum testosterone, glucose, and lipid profiles recommended as part of the initial workup (59,60). ED is also recognized as a marker for underlying cardiovascular disease, warranting further evaluation for occult vascular pathology (58,60).

Recent meta-analyses confirm that ED in diabetes is strongly associated with poor glycemic control, longer disease duration, diabetic neuropathy, retinopathy, nephropathy, cardiovascular disease, hypertension, metabolic syndrome, and depression (61,62). These findings highlight the importance of targeted screening in patients with these risk factors, as early identification and management can improve sexual function and overall health outcomes. Beyond ED, clinicians should assess for bladder dysfunction and urinary incontinence, which may indicate diabetic autonomic neuropathy or other complications, particularly in those with recurrent urinary tract infections or neurogenic bladder (61,62). Multidisciplinary management involving sexual medicine, endocrinology, and mental health support is often required. In summary, structured, proactive screening using validated tools and targeted laboratory evaluation is recommended for men with diabetes and other chronic diseases, as early detection facilitates timely intervention and improves quality of life (58–62)

3.10 Diabetic Foot Examination:

The ADA recommends that all patients with diabetes mellitus undergo a comprehensive foot examination at least annually to identify risk factors for ulcers and amputations. This evaluation should include inspection of the skin, assessment of foot deformities, neurological testing using the 10-g monofilament combined with at least one other method (such as pinprick, temperature, or vibration), and vascular assessment of pedal pulses and signs of peripheral artery disease (PAD) (66). Individuals with sensory loss, prior ulceration, or amputation should have their feet inspected at every clinical visit (66) . Initial PAD screening should include assessment of lower-extremity pulses and symptoms, with further vascular

vascular testing such as ankle-brachial index when indicated (66). Screening frequency is stratified by risk: very low risk (no loss of protective sensation [LOPS], no PAD) annually; low risk (LOPS or PAD) every 6–12 months; moderate risk (LOPS plus PAD or foot deformity) every 3–6 months; and high risk (LOPS or PAD plus history of ulcer, amputation, or end-stage renal disease) every 1–3 months (66). All patients should receive education on daily foot self-examination and preventive care, and those at high risk should be referred to foot care specialists and consider specialized footwear(66).

These recommendations are based on the high prevalence and morbidity of diabetic foot complications. Diabetic foot ulcers affect approximately 2–6% of people with diabetes annually, and the lifetime risk of ulceration is up to 25%, with amputation rates significantly higher than in the general population (66). Up to 50% of diabetic peripheral neuropathy cases are asymptomatic, making early recognition critical to prevent progression to ulceration and amputation (66). Major risk factors include poor glycemic control, neuropathy, PAD, foot deformities, prior ulceration or amputation, smoking, retinopathy, and nephropathy(66). For other chronic non-communicable diseases, routine foot screening is not specifically recommended unless diabetes or PAD is present(66). Early detection and intervention can substantially reduce morbidity and improve outcomes.

Recent epidemiological studies and meta-analyses provide further insight into the burden and risk factors for diabetic foot complications. Large-scale data indicate that the lifetime risk of developing a diabetic foot ulcer (DFU) in patients with diabetes is at least 19–34%, with annual prevalence rates ranging from 2.2% to 6.3% and incidence rates around 4–6% depending on the population and setting (67–69). Recurrence after healing is common, with rates up to 65% at 3–5 years, and the risk of lower-extremity amputation following a DFU is approximately 20% over a lifetime (67,69). Notably, the presence of a DFU more than doubles mortality risk compared to diabetic patients without foot ulcers (68). PAD is present in about half of patients with DFU and is a major predictor of non-healing and amputation (68,70). Socioeconomic disparities and comorbid cardiovascular disease further increase the risk and worsen outcomes, with recent trends showing rising amputation rates, particularly among younger and minority populations (67,68).

Validated screening tools such as the Michigan Neuropathy Screening Instrument, Semmes-Weinstein monofilament, and Ipswich Touch Test are widely used for objective assessment of neuropathy and ulcer risk (25,71). In addition to neuropathy screening, comprehensive foot exams should include evaluation for vascular insufficiency, musculoskeletal deformities, and skin breakdown, as these factors are integral to risk stratification and prevention (25). For patients with other chronic non-communicable diseases, the incidence of foot complications is substantially lower unless diabetes or PAD is present. However, multidisciplinary monitoring, patient education, and periodic foot examination remain essential strategies to reduce ulceration and amputation risk in high-risk groups (69).

3.11 Retinopathy Screening:

The ADA recommends that adults with type 1 diabetes undergo an initial dilated and comprehensive fundus examination within five years of diagnosis, while those with type 2 diabetes should have this exam at the time of diagnosis (38,66,72). If no retinopathy is detected and glycemic control is optimal, screening may be repeated every 1–2 years; otherwise, annual exams are advised. More frequent assessments are required if retinopathy is present, progressing, or sight-threatening, and during pregnancy in patients with preexisting diabetes, with exams before conception, in the first trimester, and as indicated throughout pregnancy and postpartum (38,66,72). Screening methods include dilated fundus examination and validated retinal photography, which may be interpreted by trained professionals or FDA-approved artificial intelligence systems. Digital retinal imaging improves access, but abnormal or poor-quality images require referral for in-person examination, and all results should be documented and communicated to the referring provider (38,66).

Diabetic retinopathy (DR) is often asymptomatic until advanced stages and remains a leading cause of vision loss. Epidemiological studies show that up to 20% of patients with newly diagnosed type 2 diabetes have retinopathy, and the lifetime risk exceeds 30% (25,73). Vision-threatening DR is less common initially but increases with longer diabetes duration, poor glycemic control, and comorbid hypertension, and can progress rapidly during pregnancy or puberty (73). Despite clear guidelines, only about 60% of people with diabetes receive annual retinal screenings, highlighting gaps in preventive care and the need for improved access (73). While the gold standard remains a dilated fundus examination, validated digital retinal imaging interpreted by trained readers or AI systems offers an effective alternative, particularly in resource-limited settings (25,73). For patients without retinopathy and well-controlled diabetes, screening intervals may be safely extended to every 2–3 years, whereas those with any degree of retinopathy or additional risk factors require at least annual assessments (25,74). Comprehensive eye exams also detect other ocular conditions such as cataracts, glaucoma, and age-related macular degeneration, which are more prevalent in diabetes and other chronic diseases (25,75).

3.12 FIB-4 Score for Liver Fibrosis:

The ADA recommends that adults with type 2 diabetes, prediabetes, or other cardiometabolic risk factors, including obesity and established cardiovascular disease, be screened for risk of cirrhosis related to metabolic dysfunction–associated steatotic liver disease (MASLD) using the FIB-4 score, even when liver enzymes are normal (41,76). Initial FIB-4 assessment should be performed at diagnosis and as part of the comprehensive

medical evaluation, with repeat surveillance every 1–2 years if initial risk is low and no new risk factors develop. If FIB-4 is ≥1.3, further risk stratification with liver stiffness measurement (transient elastography) or the enhanced liver fibrosis (ELF) test is recommended; if FIB-4 exceeds 2.67, direct referral to hepatology is warranted (41,76). For patients with persistently elevated aminotransferases (>6 months) and low FIB-4, evaluation for other causes of liver disease is advised. In type 1 diabetes, routine FIB-4 screening is not recommended unless additional risk factors such as obesity, abnormal liver imaging, or elevated aminotransferases are present (41). FIB-4 is the most cost-effective initial screening tool in primary care and diabetes clinics, as reliance on liver enzymes alone would miss most cases of clinically significant fibrosis (41,76).

MASLD is highly prevalent in diabetes and other chronic non-communicable diseases, affecting up to 68% of individuals with type 2 diabetes and 20% of those with type 1 diabetes, largely driven by obesity (41,76). "At-risk" MASH (steatohepatitis with significant fibrosis, ≥F2) occurs in 12–20% of people with type 2 diabetes and 5% of those with type 1 diabetes (41,76). Recent multicenter studies show that 17.6% of patients with type 2 diabetes and/or obesity have intermediate or high risk of advanced fibrosis by FIB-4, and 9.3% have high risk, underscoring the burden of advanced liver disease in these populations (77). Consensus statements recommend FIB-4 assessment at diagnosis and repeat testing every 1–3 years if initial risk is low, with second-line elastography or serum fibrosis tests for indeterminate scores (1.3–2.7) and hepatology referral for high scores (>2.7) (78,79). MASLD is the most common chronic liver disease globally, affecting 30–40% of adults and up to 70–80% of those with obesity, with rising mortality linked to cirrhosis, hepatocellular carcinoma, cardiovascular disease, and chronic kidney disease (80). These findings reinforce the importance of regular FIB-4 screening and sequential noninvasive testing in high-risk populations to enable early detection and intervention (78–80).

3.12 Heart Failure Screening:

The ADA recommends that adults with diabetes mellitus undergo initial screening for heart failure using NT-proBNP (or BNP) at diagnosis or as part of the comprehensive cardiovascular risk assessment, even if asymptomatic (17,81). For ongoing surveillance, annual NT-proBNP measurement is advised to detect early (stage B) heart failure and guide preventive management. If NT-proBNP is ≥125 pg/mL (or BNP ≥50 pg/mL), further evaluation with echocardiography is recommended to assess for structural heart disease and diastolic dysfunction, and multidisciplinary management should be considered (17,81). Abnormal NT-proBNP results should be interpreted in clinical context, as levels may be elevated due to renal insufficiency, pulmonary disease, or other comorbidities, and may be lower in individuals with obesity (17). These recommendations are based on evidence that diabetes substantially increases the risk of heart failure, which may be the first manifestation of cardiovascular disease in this population. Elevated NT-proBNP identifies individuals at highest risk for progression to symptomatic heart failure or death, and serial measurements (every 6–12 months) can improve sensitivity for detecting incident heart failure (81).

Recent consensus and large cohort studies further support NT-proBNP screening in diabetes and in other high-risk populations. Expert panels advise initiating NT-proBNP testing at the time of type 2 diabetes diagnosis and five years after onset of type 1 diabetes, with annual testing thereafter, beginning no earlier than age 30 for type 1 diabetes and at any age for type 2 diabetes (82). An abnormal NT-proBNP result (≥125 pg/mL) defines asymptomatic preclinical heart failure (Stage B), warranting echocardiography for risk stratification and management (82). Pooled analyses show that selective NT-proBNP testing, especially when combined with risk scores such as WATCH-DM, efficiently identifies high-risk individuals who benefit from preventive therapies like SGLT2 inhibitors, reducing heart failure incidence (83). Elevated NT-proBNP is highly prognostic for incident heart failure and mortality, with hazard ratios 2–4.5 times higher for levels above 125 pg/mL (84). Guidance from the Heart Failure Society of America and the American Society for Preventive Cardiology supports annual NT-proBNP measurement in asymptomatic individuals with type 2 diabetes, recognizing its predictive value and role in risk-based prevention (85). For other chronic non-communicable diseases, NT-proBNP screening is most valuable in those with multiple comorbidities or elevated cardiovascular risk rather than universal application (86). Overall, integrating NT-proBNP screening into routine diabetes care enables early identification of preclinical heart failure and supports timely preventive intervention.

3.13 Microalbuminuria Testing:

The ADA recommends annual measurement of the urine albumin-to-creatinine ratio (uACR) in all adults with type 2 diabetes starting at diagnosis and in individuals with type 1 diabetes beginning five years after diagnosis, using a spot urine sample (7,8,87). For those with established CKD, defined as persistent eGFR <60 mL/min/1.73 m² or albuminuria ≥30 mg/g, monitoring of uACR and eGFR should be performed 1–4 times per year, with frequency determined by CKD stage, progression risk, and comorbidities (7,8,87). If an abnormal uACR (≥30 mg/g) is detected, confirmation with a repeat sample within 3–6 months is recommended due to biological variability. More frequent monitoring (every 3–6 months) is advised for patients with worsening albuminuria, declining eGFR, or after initiation or adjustment of therapies targeting kidney protection, such as ACE inhibitors, ARBs, or SGLT2 inhibitors (7,8,87). These recommendations reflect the role of albuminuria as a sensitive marker for early diabetic kidney disease and cardiovascular risk, often preceding changes in eGFR. Early detection and regular monitoring enable timely intervention to slow CKD progression and reduce cardiovascular events (7,8,87).

Recent evidence highlights that albuminuria is present in up to 40% of individuals with type 2 diabetes, and diabetic kidney disease remains the leading cause of CKD and end-stage renal disease globally, as well as an independent risk factor for cardiovascular events and mortality (88). Persistent albuminuria, confirmed by two abnormal uACR results 3–6 months apart, signals progressive renal decline and heightened cardiorenal risk. Early identification allows for timely initiation of renoprotective therapies, including SGLT2 inhibitors, ACE inhibitors, ARBs, and finerenone, which has demonstrated benefit in reducing CKD progression and cardiovascular events in patients with diabetes and CKD .(123). Despite clear guidelines, many patients remain underdiagnosed and undertreated, underscoring the need for improved screening and adherence at all levels of care (88). For other chronic non-communicable diseases, uACR monitoring is most valuable in those with hypertension, cardiovascular disease, or other CKD risk factors, where albuminuria remains a strong predictor of adverse outcomes. Overall, integrating regular uACR assessment into diabetes and high-risk chronic disease care pathways is essential for early detection, risk stratification, and optimal management of CKD and its complications.

3.14 Vaccinations

The ADA recommends that adults with diabetes mellitus and other chronic non-communicable diseases receive age-appropriate vaccinations based on guidance from the Advisory Committee on Immunization Practices and the Centers for Disease Control and Prevention (41). Annual influenza vaccination with an inactivated (not live attenuated) vaccine is advised for all people with diabetes due to their increased risk of severe influenza-related complications and hospitalizations (41). Pneumococcal vaccination is recommended for adults aged 19–64 years with a single dose of PCV15 or PCV20; if PCV15 is used, PPSV23 should follow at least one year later, while PPSV23 is not needed after PCV20. Adults ≥65 years should receive one dose of PCV15 or PCV20, with similar follow-up recommendations. Hepatitis B vaccination is universally recommended for adults with diabetes under 60 years and may be considered for those ≥60 years based on individual risk (41). Additional vaccines include Tdap boosters every 10 years, herpes zoster vaccination (two-dose Shingrix) for adults ≥50 years, COVID-19 vaccination for all individuals ≥6 months, and RSV vaccination for adults ≥60 years after shared decision-making (41).

These recommendations are supported by strong evidence demonstrating increased susceptibility to infections and higher rates of complications, hospitalizations, and mortality in patients with diabetes and other chronic diseases. Meta-analyses confirm that influenza vaccination significantly reduces all-cause hospitalization, pneumonia/influenza-specific hospitalization, and mortality in people with diabetes, with a number needed to treat (NNT) of 60 for hospitalization and 250 for mortality (89). Pneumococcal vaccination is similarly justified by the elevated risk of invasive pneumococcal disease in diabetes, and newer conjugate vaccines (PCV15, PCV20) have shown comparable immunogenicity and safety (25,90). Hepatitis B vaccination remains critical, as diabetes increases susceptibility to infection and complications; newer two-dose vaccines (HBsAg-1018) provide higher seroprotection than traditional three-dose regimens, however, are not yet available in the Middle East (25). Herpes zoster vaccination is recommended for adults ≥50 years because diabetes and other chronic conditions increase the risk and severity of shingles and its complications (25,90). Tdap boosters and COVID-19 vaccination are essential preventive measures, with COVID-19 vaccines reducing hospitalization and mortality in high-risk populations (25,91).

The ACC highlights the importance of RSV vaccination in adults with chronic medical conditions such as congestive heart failure, coronary heart disease, diabetes, and chronic obstructive pulmonary disease, given their elevated risk for severe RSV disease and the substantial burden of RSV-related hospitalizations and deaths among older adults in the United States (92). Clinical trial evidence, including the AReSVi-006 study, demonstrates that a single dose of RSVPreF3 (Arexvy) provides sustained efficacy against RSV-related lower respiratory tract disease across multiple seasons in adults ≥60 years, with peak efficacy of 88% and durable protection extending beyond two years despite gradual waning (93,94). Real-world data further confirm reductions in RSV-associated hospitalizations and severe in-hospital outcomes among vaccinated adults, supporting its clinical utility (95,96). Safety evaluations show that adverse events are generally mild and transient, though rare inflammatory neurologic events have been reported (97). RSV vaccines can also be co-administered with other adult vaccines, including influenza, without compromising immune responses, facilitating efficient preventive care during routine visits in late summer or early fall (94,95).

Despite clear guidelines, vaccination rates remain suboptimal among individuals with diabetes and other chronic diseases, underscoring the need for systematic assessment of immunization status at every clinical encounter and proactive strategies to overcome barriers to vaccine uptake (91). Clinical practice should also incorporate other age-appropriate vaccines such as MMR, varicella, HPV, and polio based on individual risk and travel history (25,90). Overall, integrating comprehensive immunization into routine diabetes and chronic disease care pathways is a cornerstone of preventive medicine, reducing infection-related morbidity and mortality and improving long-term health outcomes.

3.15 Cancer Screening (Category S)

The ADA recommends that patients with diabetes mellitus and other chronic non-communicable diseases follow standard, age- and sex-appropriate cancer screening guidelines for colorectal, breast, and cervical cancer in coordination with their primary care provider (41). Diabetes is associated with increased risk for several cancers,

including colorectal and breast cancer, due to shared risk factors such as older age, obesity, and physical inactivity, as well as possible diabetes-related mechanisms (41). There is no recommendation for routine pancreatic cancer screening unless symptoms or atypical diabetes presentation are present (41). For osteoporosis, the ADA advises bone mineral density (BMD) testing using dual-energy X-ray absorptiometry (DXA) in all adults aged ≥65 years, and in postmenopausal women and men aged ≥50 years with diabetes-specific risk factors (e.g., long disease duration, frequent hypoglycemia, A1C >8%, neuropathy, retinopathy, nephropathy, falls, or glucocorticoid use). BMD should be monitored every 2–3 years in older adults and younger individuals with multiple risk factors, and fracture risk should be routinely assessed, with pharmacologic therapy considered for those at high risk (41). The Qatar national guidelines recommend the use of the locally calibrated FRAX tool (Q-FRAX) for osteoporosis screening in postmenopausal women and men aged ≥50 years, with particular emphasis on identifying high fracture risk among patients with chronic conditions such as diabetes, hypertension, and chronic kidney disease, where DXA scanning is reserved for those classified as high-risk by Q-FRAX (108).

Current guidelines from the U.S. Preventive Services Task Force (USPSTF), National Comprehensive Cancer Network (NCCN), American Cancer Society (ACS), and American College of Physicians (ACP) reinforce that patients with diabetes should follow standard population-based screening intervals for colorectal, breast, and cervical cancer (98–102). For colorectal cancer, adults aged 45–75 years should undergo regular screening using one of several modalities: annual fecal immunochemical test (FIT) or high-sensitivity guaiac-based fecal occult blood test (gFOBT), multi-target stool DNA test every 3 years, colonoscopy every 10 years, CT colonography every 5 years, or flexible sigmoidoscopy every 5 years (98–102). Screening for ages 76–85 should be individualized based on comorbidities and life expectancy, and is not recommended after age 85 (98–102). Although diabetes confers a higher risk and earlier onset of colorectal cancer, current guidelines do not recommend earlier or more frequent screening solely based on diabetes status; however, emerging data suggest that individuals with diabetes may reach threshold risk for CRC earlier, supporting vigilant adherence to screening schedules (98–102).

For breast cancer, women aged 45–54 years should have annual mammography, transitioning to biennial screening at age ≥55, with the option to continue annual screening (103–106). Screening should continue as long as overall health is good and life expectancy is ≥10 years. (103–106). For cervical cancer, screening should begin at age 21 with Pap testing every 3 years for women aged 21–29, and for women aged 30–65, preferred screening is HPV testing plus Pap every 5 years, or Pap alone every 3 years (103–106). Screening may be discontinued after age 65 if prior results have been consistently negative (103–106). While diabetes does not alter recommended intervals, adherence to breast and cervical cancer screening is consistently lower in patients with diabetes, highlighting the need for targeted interventions to improve uptake (105,106).

For osteoporosis, population-based guidelines recommend DXA screening for women ≥65 and men ≥70, or younger adults with risk factors (41). In diabetes, additional risk factors such as long disease duration, complications, and medication use may prompt earlier or more frequent screening, but intervals of every 2–3 years are generally appropriate unless clinical changes occur (41). Overall, the absolute benefit of cancer and osteoporosis screening may be greater in diabetes due to higher baseline risk, but screening intervals and modalities should be individualized based on comorbidities, life expectancy, and patient preferences (102,124). Integrating these preventive strategies into routine diabetes care is essential to reduce morbidity and mortality associated with cancer and fractures.

3.16 Obstructive sleep apnea

The American Academy of Sleep Medicine recommends annual screening for obstructive sleep apnea (OSA) in adults aged 18 years and older who are at high risk, including patients with type 2 diabetes, impaired glucose tolerance, and treatment-resistant hypertension (109). Screening should be performed at least every 12 months using a standardized tool, with documentation of a follow-up plan based on results (109). Other high-risk conditions identified in the 2024 quality measure include obesity (BMI ≥30 kg/m²), congestive heart failure, atrial fibrillation, nocturnal dysrhythmias, stroke, pulmonary hypertension, preoperative evaluation for bariatric surgery, and coronary artery disease (109). Although chronic kidney disease is not explicitly listed, its association with cardiovascular risk suggests that clinicians should maintain vigilance in these patients (109).

The ADA similarly emphasizes screening for OSA in individuals with diabetes, recommending evaluation in those with symptoms such as excessive daytime sleepiness, snoring, and witnessed apnea (41). The American Association of Clinical Endocrinology advises incorporating routine screening for sleep disorders either clinically through symptom-based questions or using validated tools such as the STOP-Bang questionnaire (25,91). These recommendations are supported by the high prevalence of OSA in type 2 diabetes, estimated at 23% to 58% for any sleep-disordered breathing, with rates exceeding 80% in patients with both diabetes and obesity (25,41). Untreated OSA is linked to poor glycemic control, increased cardiovascular risk, and reduced quality of life, making early detection critical.

Despite these guidelines, screening rates remain low, and underdiagnosis is common. The American Heart Association notes that 86% to 95% of individuals with clinically significant OSA report no prior diagnosis (112). While the U.S. Preventive Services Task Force concluded in 2022 that evidence is insufficient to recommend universal screening in asymptomatic adults (110,111),

recent studies highlight the gap between prevalence and diagnosis. For example, a 2025 study of 164 patients with type 2 diabetes in primary care found that 75% had OSA, with 31% requiring treatment for moderate to severe cases; central fat distribution and albuminuria were significant predictors of severe OSA (113). These findings underscore the need for targeted screening in high-risk populations using validated tools such as STOP-Bang and Epworth Sleepiness Scale, despite limitations in sensitivity and specificity (112). Integrating OSA screening into diabetes and cardiovascular care pathways can improve patient-centered outcomes and reduce long-term complications.

Conclusion

The *"ABCDEF Ultra Violet S: Family Medicine Guide"* offers a practical, evidence-based framework to close persistent gaps between guideline recommendations and real-world practice in chronic disease management. By standardizing preventive care and empowering both clinicians and patients, the guide addresses critical domains from metabolic monitoring and cardiovascular risk assessment to mental health, cancer screening, and vaccination, within a single, streamlined approach. Its dual-purpose design reduces cognitive load for providers while enhancing patient engagement and health literacy, fostering shared decision-making and continuity of care. Expected benefits include improved clinical outcomes through earlier detection and better disease control, enhanced patient satisfaction, and cost savings from complication prevention. Successful implementation will require provider training, stakeholder engagement, and iterative refinement based on feedback and data. Future quality audits, adherence monitoring, and structured quality improvement projects are essential to evaluate impact, identify gaps, and sustain progress. These efforts will not only strengthen compliance with preventive care standards but also contribute valuable evidence to inform policy and practice.

Ultimately, this initiative reflects the core values of family medicine, comprehensive, patient-centered, and quality-driven care and demonstrates that systematic, checklist-based approaches can transform chronic disease management, improving outcomes for every patient, every time.

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Parkinson's Disease Dementia: Etiology, Mechanisms, Diagnosis, Management and Future Directions

A Abyad

Correspondence:
A. Abyad, MD, MPH, MBA, DBA, AGSF
Consultant, Internal Medicine and Geriatric, Dar Al Shifa Hospital, Kuwait
Chairman, Middle-East Academy for Medicine of Aging.
President, Middle East & North Africa Association on Aging & Alzheimer's
Coordinator, Middle-East Primary Care Research Network
Coordinator, Middle-East Network on Aging
Email: aabyad@cyberia.net.lb

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Abstract

Parkinson's disease dementia (PDD) is a common, disabling, and prognostically important neurocognitive syndrome arising in the context of established Parkinson's disease (PD). It represents one of the major late-stage manifestations of synucleinopathy and reflects the convergence of cortical Lewy body pathology, cholinergic degeneration, dopaminergic network dysfunction, Alzheimer-type co-pathology, neuroinflammation, vascular injury, and age-related vulnerability. Clinically, PDD is characterized by progressive impairment in attention, executive function, visuospatial processing, memory retrieval, and behavioural regulation, typically accompanied by neuropsychiatric symptoms such as visual hallucinations, apathy, depression, anxiety, delusions, REM sleep behaviour disorder, and fluctuating cognition. The diagnostic distinction between PDD and dementia with Lewy bodies remains anchored in the one-year rule, although biological and clinicopathological evidence increasingly supports their conceptualization as overlapping Lewy body dementias. Diagnosis remains primarily clinical, supported by neuropsychological testing, structural and functional imaging, exclusion of reversible contributors, and emerging biomarkers including α-synuclein seed amplification assays, amyloid and tau biomarkers, and neurodegeneration markers. Rivastigmine remains the best-supported symptomatic pharmacologic therapy, while management requires systematic rationalization of dopaminergic and anticholinergic medication, treatment of neuropsychiatric complications, sleep optimization, rehabilitation, caregiver support, and advanced-care planning. Disease-modifying therapies remain investigational, but future directions include biological staging, precision phenotyping, synuclein-targeted immunotherapy, lysosomal enhancement, neuroinflammation modulation, digital biomarkers, and integrated trials across the Lewy body disease spectrum.

Keywords: Parkinson's disease dementia; Lewy body dementia; α-synuclein; cognitive impairment; rivastigmine; dementia with Lewy bodies; neurodegeneration; synucleinopathy

Overview

Parkinson's disease dementia (PDD) is a progressive major neurocognitive disorder that develops in patients with established Parkinson's disease (PD). It is clinically defined by cognitive decline severe enough to impair activities of daily living and occurring in the setting of previously diagnosed idiopathic PD. The central diagnostic premise is temporal: motor Parkinsonism precedes dementia by more than one year. When dementia precedes or emerges within one year of Parkinsonism, the syndrome is conventionally classified as dementia with Lewy bodies (DLB), although both disorders share α-synuclein pathology and are increasingly viewed as related clinical expressions within a Lewy body disease continuum (Emre et al., 2007; McKeith et al., 2017; Aarsland et al., 2021).

PDD is not merely an extension of motor PD. It represents widespread failure of distributed neural systems, particularly frontostriatal, limbic, cholinergic, attentional, and posterior cortical networks. The cognitive profile differs from typical Alzheimer's disease in that early impairments often involve attention, executive function, processing speed, visuospatial ability, and retrieval-based memory rather than isolated amnestic encoding failure (Emre et al., 2007; Litvan et al., 2012; Aarsland et al., 2021). Nevertheless, Alzheimer-type amyloid and tau pathology frequently coexist and influence phenotype, progression, and prognosis (Irwin et al., 2013; Compta et al., 2011; Halliday et al., 2014).

The clinical importance of PDD is substantial. Cognitive impairment in PD predicts loss of independence, medication complexity, psychosis, falls, institutionalization, caregiver burden, and mortality (Hely et al., 2008; Lawson et al., 2016; Aarsland et al., 2021). PDD also complicates treatment of motor symptoms because dopaminergic escalation can aggravate hallucinations, impulse-control symptoms, confusion, and orthostatic hypotension, while reduction of dopaminergic therapy may worsen mobility and quality of life. Thus, clinical reasoning in PDD requires balancing cognition, psychosis, sleep, autonomic function, mobility, swallowing, falls risk, caregiver capacity, and patient preferences.

From a neuropathological standpoint, PDD is usually associated with cortical and limbic Lewy body-type α-synuclein pathology, frequently accompanied by degeneration of cholinergic nuclei, noradrenergic and serotonergic systems, dopaminergic mesocortical pathways, and variable amyloid-β and tau co-pathology (Braak et al., 2003; Halliday et al., 2014; Irwin et al., 2013). The heterogeneity of PDD arises from differential involvement of these systems. A patient with severe cholinergic and posterior cortical involvement may present with hallucinations, attentional fluctuation, and visuospatial impairment, whereas another with prominent frontostriatal dysfunction may show dysexecutive slowing, apathy, and impaired planning.

Etiology

The etiology of PDD is multifactorial and cannot be attributed to a single lesion, neurotransmitter deficit, or proteinopathy. Rather, PDD emerges when neurodegenerative burden crosses a threshold sufficient to impair large-scale cognitive networks. The dominant etiological substrate is Lewy body disease, but dementia risk is shaped by age, disease duration, genetic susceptibility, motor phenotype, neuropsychiatric manifestations, sleep disorders, autonomic dysfunction, vascular injury, systemic illness, and medication exposure.

The primary pathological process is aggregation of misfolded α-synuclein into Lewy bodies and Lewy neurites. In PD, α-synuclein pathology classically involves the substantia nigra and other brainstem nuclei, but progression to limbic and neocortical regions is strongly associated with dementia (Braak et al., 2003; Dickson, 2018). Cortical Lewy body pathology is particularly relevant when it affects temporal, parietal, cingulate, and frontal association cortices. However, the relationship between Lewy body counts and cognition is imperfect, suggesting that synaptic dysfunction, soluble α-synuclein species, network disconnection, and co-pathologies may be as important as visible inclusions (Kramer & Schulz-Schaeffer, 2007; Halliday et al., 2014).

Cholinergic degeneration is another central etiological contributor. The nucleus basalis of Meynert, pedunculopontine nucleus, and other cholinergic structures are affected in PD and PDD. Cholinergic dysfunction correlates with attentional deficits, visual hallucinations, fluctuations, gait impairment, and falls (Bohnen & Albin, 2011; Bohnen et al., 2015). The therapeutic benefit of cholinesterase inhibitors, particularly rivastigmine, supports the clinical relevance of cholinergic loss (Emre et al., 2004).

Alzheimer-type pathology contributes variably to PDD. Amyloid-β plaques and tau neurofibrillary tangles are more frequent in cognitively impaired PD than in cognitively normal PD and may accelerate cognitive decline (Compta et al., 2011; Irwin et al., 2013). The presence of mixed Lewy and Alzheimer pathology may shift the phenotype toward more prominent memory impairment and faster progression.

Medication effects are not usually the primary etiology of PDD, but they can unmask or exacerbate cognitive impairment. Anticholinergics, dopamine agonists, amantadine, benzodiazepines, opioids, sedative hypnotics, and polypharmacy may contribute to delirium, hallucinations, orthostasis, and attentional impairment. The clinician must therefore distinguish progressive dementia from medication-induced encephalopathy or delirium, particularly when symptoms evolve abruptly.

Epidemiology

Cognitive impairment is common across the PD disease course. Mild cognitive impairment in PD may occur early, including near diagnosis, whereas dementia typically increases with age and disease duration (Aarsland et al., 2021). Longitudinal studies indicate that a substantial proportion of patients with PD develop dementia over time, with cumulative risk rising markedly after 10–20 years of disease (Hely et al., 2008; Buter et al., 2008; Aarsland et al., 2021).

The prevalence of PDD varies by diagnostic criteria, population age, disease duration, ascertainment method, and survival bias. Clinic-based cohorts tend to over-represent advanced disease, whereas community-based studies may capture broader severity. Older age at PD onset, greater motor severity, postural instability-gait disorder phenotype, hallucinations, REM sleep behaviour disorder, autonomic dysfunction, depression, and baseline mild cognitive impairment consistently predict dementia (Aarsland et al., 2003; Williams-Gray et al., 2009; Lawson et al., 2016).

The epidemiology of PDD is also shaped by survival. Patients with tremor-dominant PD and younger onset may live for many years before dementia, while patients with older-onset akinetic-rigid or postural instability phenotypes may progress more quickly. Male sex has often been associated with higher PD incidence and may influence dementia risk indirectly through phenotype, comorbidity, and survival, although sex-specific risk estimates vary across cohorts.

Table 1. Epidemiologic and clinical risk factors for PDD

Risk factor	Clinical implication	Mechanistic interpretation
Older age	Strongest demographic predictor	Reduced cognitive reserve, higher mixed pathology burden
Longer PD duration	Dementia risk increases over time	Progressive cortical and limbic spread of synucleinopathy
Older age at PD onset	Higher early dementia risk	Greater baseline vulnerability and co-pathology
Postural instability-gait disorder phenotype	Higher dementia and falls risk	Non-dopaminergic network involvement
Visual hallucinations	Predictor and marker of Lewy body dementia phenotype	Cholinergic dysfunction and posterior cortical involvement
REM sleep behaviour disorder	Associated with diffuse synucleinopathy	Brainstem-limbic network involvement
Orthostatic hypotension/autonomic dysfunction	Associated with cognitive decline	Diffuse α-synuclein burden and vascular vulnerability
PD-MCI	Major prodromal state	Early network-level cognitive involvement
GBA mutation	Increased risk and faster decline	Lysosomal dysfunction and α-synuclein accumulation
Alzheimer co-pathology	Faster decline and memory impairment	Amyloid-tau-synuclein interaction

Genetic Factors

PDD is usually sporadic, but genetic factors influence susceptibility, age at onset, phenotype, and rate of cognitive decline. The strongest and most clinically relevant genetic association is with glucocerebrosidase gene variants. GBA encodes the lysosomal enzyme glucocerebrosidase, and heterozygous pathogenic variants increase risk for PD and are associated with earlier onset, more rapid progression, autonomic dysfunction, hallucinations, and dementia (Sidransky et al., 2009; Gan-Or et al., 2015; Liu et al., 2016). The mechanistic link is biologically plausible: impaired lysosomal function may reduce α-synuclein clearance, while α-synuclein accumulation may further impair glucocerebrosidase trafficking, creating a pathogenic feedback loop.

SNCA mutations and multiplications are associated with autosomal dominant PD and may produce prominent cognitive impairment, psychiatric symptoms, and dementia, especially with gene multiplications that increase α-synuclein dosage (Singleton et al., 2003; Fuchs et al., 2007). These families illustrate the principle that α-synuclein burden is sufficient to drive a diffuse Lewy body phenotype.

MAPT haplotypes have been associated with cognitive outcomes in PD. Tau biology may influence network vulnerability, especially where Alzheimer-type pathology coexists. APOE ε4 is associated with Alzheimer's disease risk and may also influence cognitive decline in PD, likely through amyloid and tau pathways rather than synuclein-specific mechanisms (Irwin et al., 2013; Tsuang et al., 2013).

LRRK2-associated PD is heterogeneous. Some LRRK2 patients resemble idiopathic PD, while others show less cognitive impairment and less Lewy body pathology, depending on mutation, ancestry, and pathological substrate. This variability emphasizes that clinical PD is etiologically heterogeneous and that dementia risk is not uniform across genetic forms.

Table 2. Genetic contributors to cognitive impairment and PDD

Gene/locus	Biological pathway	PDD relevance	Clinical interpretation
GBA	Lysosomal function	Strong association with cognitive decline and dementia	High-risk genotype; consider closer cognitive surveillance
SNCA	α-synuclein production/aggregation	Multiplications associated with dementia and psychiatric features	Supports dose-dependent synuclein toxicity
MAPT	Tau biology	Modifies cognitive phenotype and risk	Suggests tau-synuclein interaction
APOE	Lipid transport, amyloid risk	ε4 may increase cognitive decline risk	Often indicates mixed Alzheimer-Lewy vulnerability
LRRK2	Kinase signalling, vesicle trafficking	Variable dementia risk	Mutation-specific and pathology-dependent
PRKN/PINK1/DJ-1	Mitochondrial quality control	Often young-onset; dementia less typical early	Cognitive risk depends on duration and phenotype

Pathophysiology

The pathophysiology of PDD is best understood as a multi-level process involving protein aggregation, synaptic failure, neurotransmitter depletion, circuit disruption, neuroinflammation, mitochondrial dysfunction, lysosomal impairment, and co-pathology.

1 α-Synuclein aggregation and propagation
α-Synuclein is a presynaptic protein involved in vesicular trafficking and synaptic function. In disease states, misfolded α-synuclein aggregates into oligomers, fibrils, Lewy neurites, and Lewy bodies. The distribution of Lewy pathology correlates broadly with clinical stage. Brainstem-predominant pathology produces motor and autonomic features; limbic and neocortical involvement is more closely associated with hallucinations, fluctuations, and dementia (Braak et al., 2003; Dickson, 2018).

The prion-like propagation hypothesis proposes that pathological α-synuclein spreads through connected neural systems. Although this model remains debated, it explains the stereotyped involvement of olfactory, autonomic, brainstem, limbic, and cortical regions in many patients. The cognitive phenotype depends not simply on the presence of α-synuclein but on where it accumulates, which neurons are vulnerable, and how it interacts with aging and co-pathologies.

2 Cholinergic dysfunction
Cholinergic loss is one of the most important neurochemical correlates of PDD. Degeneration of the Nucleus Basalis of Meynert (NBM) reduces cortical acetylcholine, compromising attention, arousal, visual processing, and memory retrieval. Cholinergic deficits may be more severe in PDD than in Alzheimer's disease in certain cortical regions, which helps explain the responsiveness of some patients to cholinesterase inhibitors (Bohnen & Albin, 2011; Emre et al., 2004).
Clinically, cholinergic dysfunction helps unify several PDD features: fluctuating cognition, visual hallucinations, attentional lapses, falls, gait instability, and sleep-wake dysregulation. This also explains why anticholinergic drugs can be disproportionately harmful in PD patients at risk for dementia.

3 Dopaminergic and frontostriatal dysfunction
The dopaminergic model of PD cognition emphasizes frontostriatal loops. Dopamine depletion in dorsal striatum and mesocortical pathways impairs working memory, cognitive flexibility, set-shifting, and planning. Early PD-MCI often manifests as dysexecutive slowing related to frontostriatal dysfunction. However, dementia usually requires additional posterior cortical, cholinergic, limbic, or Alzheimer-type pathology. This distinction is clinically important: dopaminergic optimization may improve some executive functions in selected patients but can worsen hallucinations, impulsivity, and confusion in PDD.

4 Posterior cortical involvement
Visuospatial dysfunction is a major predictor of dementia in PD. Impairment in pentagon copying, clock drawing, visual discrimination, and spatial orientation often reflects posterior cortical involvement. Occipital and parietal hypometabolism on FDG-PET, posterior cortical atrophy, and cholinergic denervation have been associated with Lewy body dementia phenotypes (McKeith et al., 2017; Aarsland et al., 2021).

5 Alzheimer-type co-pathology
Amyloid-β and tau co-pathology are common in older patients with Lewy body disease and may accelerate cognitive decline. Patients with both Lewy and Alzheimer pathology often show more severe memory impairment, faster progression, and reduced survival compared with patients with relatively pure synucleinopathy (Compta et al., 2011; Irwin et al., 2013). This co-pathology complicates biomarker interpretation and may eventually justify biologically stratified treatment trials.

6 Neuroinflammation, mitochondrial dysfunction, and lysosomal impairment
Microglial activation, oxidative stress, mitochondrial dysfunction, and impaired autophagy-lysosomal pathways contribute to neuronal injury in PD and PDD. GBA-associated disease illustrates the importance of lysosomal biology. Mitochondrial impairment may contribute to neuronal vulnerability in substantia nigra and cortical networks. Neuroinflammation may amplify synaptic dysfunction and protein aggregation, although it remains uncertain whether inflammatory changes are primary drivers or secondary responses.

Differential Diagnosis

The differential diagnosis of cognitive decline in PD is broad. The most important distinction is between PDD and DLB. The one-year rule remains the practical clinical convention: dementia developing after more than one year of established PD supports PDD; dementia preceding or occurring within one year of Parkinsonism supports DLB (McKeith et al., 2017). This rule is imperfect biologically but useful clinically and in research.

Alzheimer's disease should be considered when early memory encoding failure, prominent hippocampal atrophy, aphasia, or biomarker evidence of amyloid and tau pathology is present. Vascular cognitive impairment is suggested by stepwise decline, focal neurological signs, extensive white matter disease, lacunes, strategic infarcts, or vascular risk burden. Normal pressure hydrocephalus may mimic gait disorder and cognitive impairment but typically presents with magnetic gait, urinary symptoms, ventriculomegaly, and a different motor pattern.

Medication-induced cognitive impairment is common and must be actively excluded. Anticholinergics, dopamine agonists, amantadine, sedatives, opioids, and polypharmacy can produce confusion, hallucinations, and attentional impairment. Delirium must be considered when cognitive change is acute or fluctuates dramatically over hours to days.

Depression may produce cognitive symptoms, but true PDD often shows objective visuospatial and executive deficits, hallucinations, functional decline, and progressive course. Sleep disorders, hypothyroidism, vitamin B12 deficiency, infection, dehydration, renal or hepatic dysfunction, and sensory impairment may worsen cognition and should be addressed.

Table 4. Differential diagnosis of dementia in a patient with Parkinsonism

Diagnosis	Key distinguishing features	Clinical reasoning point
PDD	Dementia develops after established PD, usually >1 year after motor onset	Diagnosis requires cognitive functional decline beyond motor disability
DLB	Dementia before or within 1 year of Parkinsonism; early hallucinations/fluctuations	Biological overlap with PDD; temporal rule remains conventional
Alzheimer's disease	Early amnestic encoding deficit, hippocampal atrophy, amyloid/tau biomarkers	Common co-pathology in PDD
Vascular cognitive impairment	Stepwise decline, focal signs, infarcts, severe small vessel disease	May coexist and lower cognitive reserve
Drug-induced cognitive impairment	Temporal relationship to medication change	Review anticholinergics, dopamine agonists, amantadine, sedatives
Delirium	Acute onset, altered arousal, medical trigger	Must be excluded before diagnosing progression
Normal pressure hydrocephalus	Ventriculomegaly, gait apraxia, urinary symptoms	Parkinsonism can coexist
Frontotemporal dementia	Early disinhibition, compulsions, aphasia	Parkinsonism may occur in atypical syndromes
Progressive supranuclear palsy	Early falls, vertical gaze palsy, axial rigidity	Cognitive syndrome often frontal
Multiple system atrophy	Severe autonomic failure, cerebellar signs	Dementia less prominent early

Diagnostic Approach

Diagnosis of PDD is clinical, supported by structured cognitive assessment and exclusion of alternative causes. The Movement Disorder Society criteria define probable PDD by established PD, dementia syndrome with impairment in more than one cognitive domain, decline from premorbid level, functional impairment, and absence of features suggesting another primary cause (Emre et al., 2007; Dubois et al., 2007).

Table 5. Diagnostic criteria for Parkinson's disease dementia

Domain	Requirement
Established PD	Diagnosis of idiopathic PD precedes dementia
Dementia syndrome	Cognitive decline from previous level
Cognitive domains	Impairment in more than one domain, commonly attention, executive function, visuospatial ability, and memory
Functional impairment	Cognitive deficits impair daily living independent of motor symptoms
Associated features	Apathy, hallucinations, delusions, depression, anxiety, excessive daytime sleepiness, fluctuations
Exclusion	Delirium, major depression alone, medication toxicity, vascular event, metabolic disease, other dementia syndrome
Temporal distinction	Dementia occurs after at least one year of established Parkinsonism

1 Clinical history

The clinician should establish the chronology of motor symptoms, cognitive decline, hallucinations, fluctuations, sleep disturbance, medication changes, and functional loss. Collateral history is essential because insight is often impaired. The history should identify whether deficits interfere with instrumental activities of daily living such as medication administration, finances, shopping, cooking, driving, communication, and appointment management.

2 Cognitive screening

The Montreal Cognitive Assessment is generally more sensitive than the Mini-Mental State Examination for PD-related executive and visuospatial deficits. However, screening tests are insufficient for complex cases. Neuropsychological testing should assess attention, processing speed, executive function, visuospatial ability, memory, language, mood, and performance validity. In advanced PD, motor slowing, tremor, dysarthria, fatigue, and visual impairment may confound testing; therefore, interpretation should be individualized.

3 Functional assessment

Functional impairment should be attributed carefully. Cognitive impairment may be masked by caregiver compensation. Conversely, motor disability may mimic cognitive dependence. Structured informant scales, medication management review, financial capacity assessment, and occupational therapy evaluation can help determine whether cognition independently affects daily living.

4 Clinical algorithm

Step	Action	Clinical purpose
1	Confirm idiopathic PD and chronology	Distinguish PDD from DLB and atypical Parkinsonism
2	Obtain collateral cognitive and functional history	Identify decline and real-world impairment
3	Review medications and recent medical events	Exclude reversible cognitive worsening
4	Screen cognition with MoCA or comparable instrument	Establish objective impairment
5	Perform neuropsychological testing when diagnosis uncertain	Define domains and severity
6	Screen mood, psychosis, sleep, autonomic symptoms	Identify treatable contributors
7	Order laboratory tests and structural imaging	Exclude metabolic, vascular, mass, hydrocephalus causes
8	Consider biomarkers when phenotype is atypical or trials are considered	Detect synuclein, amyloid, tau, or neurodegeneration signatures
9	Apply PDD criteria	Assign probable or possible diagnosis
10	Stage severity and create management plan	Align treatment with goals and safety

Laboratory and Imaging Studies

Laboratory tests do not diagnose PDD but are necessary to identify reversible or contributory causes. A reasonable baseline evaluation includes complete blood count, electrolytes, renal and liver function, thyroid-stimulating hormone, vitamin B12, folate when indicated, glucose or HbA1c, inflammatory or infectious tests when clinically suspected, and medication/toxicology review when relevant.

Structural MRI is recommended in most patients with new or worsening cognitive impairment. MRI may show generalized atrophy, posterior cortical atrophy, medial temporal atrophy if Alzheimer co-pathology is present, white matter disease, lacunes, infarcts, microbleeds, hydrocephalus, mass lesions, or subdural collections. Imaging is particularly important when decline is rapid, focal signs are present, gait changes are disproportionate, or vascular disease is suspected.

FDG-PET can show posterior cortical hypometabolism, including occipital and parietotemporal regions, in Lewy body dementia. Dopamine transporter imaging supports presynaptic dopaminergic degeneration but does not distinguish PDD from DLB and is less useful when PD is already established. Cardiac MIBG scintigraphy may support Lewy body disease in some settings but is affected by cardiac disease and medications.

Amyloid and tau PET or CSF biomarkers may be useful when Alzheimer co-pathology is suspected, when phenotype is atypically amnestic, or when disease-modifying Alzheimer therapies are being considered. α-Synuclein seed amplification assays are among the most important emerging biomarkers because they detect misfolded α-synuclein seeding activity and may enable biological classification of synucleinopathies. Their role in routine PDD diagnosis is still evolving, but they are increasingly important for research stratification and future disease-modifying trials.

Table 6. Laboratory and imaging studies in suspected PDD

Investigation	Role	Interpretation
CBC, CMP	Detect anaemia, infection clues, renal/hepatic dysfunction	Abnormalities may cause delirium or worsen cognition
TSH, vitamin B12	Detect reversible cognitive contributors	Treat deficiencies but do not assume they fully explain syndrome
MRI brain	Exclude structural lesions and assess vascular/atrophy burden	Mixed vascular or Alzheimer pathology may be suggested
FDG-PET	Characterize metabolic pattern	Posterior cortical hypometabolism supports Lewy body dementia phenotype
DAT-SPECT	Demonstrate dopaminergic deficit	Useful if Parkinsonism diagnosis uncertain
Amyloid/tau biomarkers	Detect Alzheimer co-pathology	Helps explain amnestic phenotype or rapid decline
α-Synuclein SAA	Detect synuclein seeding activity	Emerging biological marker; strongest research role currently
Polysomnography	Confirm REM sleep behaviour disorder or sleep apnea	Treatable sleep disorders affect cognition and safety

Histopathology

Histopathology is not required for clinical diagnosis but remains the reference standard for understanding disease mechanisms. The defining pathological feature is Lewy body-type α-synuclein pathology involving brainstem, limbic, and neocortical regions. Lewy bodies are eosinophilic intraneuronal inclusions containing α-synuclein, ubiquitin, neurofilament proteins, and other components. Lewy neurites represent abnormal α-synuclein accumulation in neuronal processes and may be more closely related to synaptic dysfunction than Lewy bodies themselves.

In PDD, cortical Lewy pathology is typically more extensive than in PD without dementia. Limbic structures, cingulate cortex, temporal association cortex, parietal cortex, and frontal cortex may be involved. Neuronal loss and gliosis occur in affected regions, although cognitive impairment often reflects synaptic failure before frank neuronal loss.

Co-pathology is common. Amyloid plaques and tau neurofibrillary tangles may coexist, particularly in older patients. Cerebrovascular disease, TDP-43 pathology, hippocampal sclerosis, and age-related tau astrogliopathy may further modify clinical expression. This explains why two patients with similar motor PD duration can have dramatically different cognitive trajectories.

Table 7. Histopathological contributors to PDD

Pathology	Typical substrate	Clinical relevance
Cortical Lewy bodies and Lewy neurites	Misfolded α-synuclein	Core substrate of PDD
Limbic Lewy pathology	Amygdala, cingulate, hippocampal regions	Hallucinations, mood, memory, fluctuations
Cholinergic neuronal loss	Nucleus Basalis of Meynert	Attention, hallucinations, treatment response
Alzheimer-type pathology	Amyloid plaques and tau tangles	Faster decline, amnestic phenotype
Vascular pathology	Lacunes, infarcts, small vessel disease	Stepwise decline, gait worsening, reduced reserve
Synaptic degeneration	Cortical and subcortical networks	May correlate strongly with cognition
Neuroinflammation	Microglial activation	Potential amplifier of neurodegeneration

Management

1 General principles

The first step is to identify reversible contributors. Infection, dehydration, constipation, urinary retention, sleep deprivation, pain, medication toxicity, orthostatic hypotension, sensory impairment, and depression can all worsen cognition. Treating these contributors may improve function even when underlying dementia remains.

Medication review is central. Anticholinergics should generally be discontinued. Dopamine agonists, amantadine, monoamine oxidase-B inhibitors, catechol-O-methyltransferase inhibitors, sedatives, and opioids should be reviewed carefully. In patients with hallucinations or confusion, simplification often proceeds from anticholinergics and sedatives first, then amantadine, dopamine agonists, MAO-B inhibitors, COMT inhibitors, and finally levodopa adjustments if necessary. Levodopa is usually preserved as the most effective and often best-tolerated motor therapy.

2 Cognitive pharmacotherapy

Rivastigmine has the strongest evidence and is approved for PDD in many jurisdictions. It improves global cognition, attention, executive symptoms, and neuropsychiatric symptoms modestly in some patients. Gastrointestinal adverse effects, tremor worsening, bradycardia, syncope, weight loss, and sleep disturbance require monitoring. Transdermal rivastigmine may improve tolerability.

Donepezil and galantamine have less robust evidence but may be considered when rivastigmine is not tolerated. Memantine has mixed evidence; it may provide modest global or behavioural benefit in selected patients but is not consistently effective for cognition.

Table 8. Pharmacologic treatment comparison in PDD

Treatment	Main indication	Evidence strength	Advantages	Limitations
Rivastigmine oral/transdermal	Cognitive symptoms in PDD	Strongest among cognitive agents	Approved; may help attention and hallucinations	GI effects, tremor, bradycardia, weight loss
Donepezil	Cognitive symptoms	Moderate/limited	Once daily; often tolerated	Less definitive PDD evidence
Galantamine	Cognitive symptoms	Limited	Cholinergic mechanism	Less studied in PDD
Memantine	Global/behavioural symptoms	Mixed	Generally tolerated	Modest and inconsistent cognitive benefit
Pimavanserin	Parkinson disease psychosis	Evidence for psychosis, not cognition	Less dopamine blockade	QT prolongation, cost/access
Quetiapine	Psychosis/agitation	Commonly used; limited trial evidence	Low motor worsening risk	Sedation, orthostasis, metabolic effects
Clozapine	Refractory psychosis	Strong efficacy for PD psychosis	Effective with minimal motor worsening	Agranulocytosis monitoring burden
SSRIs/SNRIs	Depression/anxiety	Symptom-based	Treats comorbid mood	Hyponatremia, falls, REM sleep effects
Melatonin/ clonazepam	REM sleep behaviour disorder	Symptom-based	Reduces dream enactment	Clonazepam worsens cognition/falls

3 Management of psychosis

Psychosis management begins with determining whether symptoms are distressing or dangerous. Benign hallucinations with preserved insight may not require antipsychotic therapy. When treatment is necessary, clinicians should first treat triggers and reduce offending medications. Pimavanserin is used for Parkinson disease psychosis in some regions and has the advantage of avoiding dopamine receptor blockade. Quetiapine is widely used because it is practical and usually has limited motor worsening, although controlled evidence is weaker. Clozapine is the most effective antipsychotic for PD psychosis but requires blood monitoring.

Typical antipsychotics and potent dopamine-blocking atypical antipsychotics such as risperidone and olanzapine should generally be avoided because they can worsen Parkinsonism and precipitate severe sensitivity reactions.

4 Motor management

Motor treatment in PDD requires conservative optimization. The goal is not maximal motor control at all costs, but the best balance between mobility, cognition, psychosis, orthostasis, dyskinesia, and caregiver feasibility. Levodopa remains the cornerstone. Dopamine agonists are often poorly tolerated in older cognitively impaired patients because they increase hallucinations, sleep attacks, edema, orthostasis, and impulse-control disorders.

Deep brain stimulation is generally not appropriate for patients with dementia. Cognitive impairment is a major contraindication because DBS may worsen cognition and does not treat axial, cognitive, or neuropsychiatric progression.

5 Non-pharmacologic management

Non-pharmacologic interventions are essential. These include structured routines, environmental cueing, medication dispensing systems, caregiver education, exercise adapted to fall risk, occupational therapy, physical therapy, speech-language therapy, swallowing evaluation, nutrition support, driving assessment, home safety assessment, and legal/financial planning.

Cognitive rehabilitation should be practical rather than restorative. Patients benefit from external memory aids, simplified choices, visual contrast enhancement, lighting optimization, reduction of clutter, sleep regularity, and caregiver-mediated routines. Exercise may support mobility, mood, sleep, and general health, although it is not a proven disease-modifying therapy for PDD.

6 Clinical management algorithm

Clinical problem	First step	Second step	Escalation
New cognitive decline	Exclude delirium, medication toxicity, metabolic causes	Cognitive testing and MRI	Biomarkers/neuropsychology if atypical
Hallucinations	Assess distress and insight; treat triggers	Reduce anticholinergics/dopamine agonists/amantadine	Pimavanserin, quetiapine, or clozapine
Cognitive symptoms	Start rivastigmine if appropriate	Switch formulation or alternative ChEI if intolerant	Consider memantine selectively
Falls	Review orthostasis, sedatives, vision, gait	PT, home safety, assistive devices	Specialist falls clinic
Orthostatic hypotension	Hydration, salt, compression, medication review	Midodrine/droxidopa/fludrocortisone when appropriate	Autonomic specialist
REM sleep behaviour disorder	Safety measures, remove hazards	Melatonin	Clonazepam cautiously
Depression/anxiety	Confirm syndrome, psychotherapy/support	SSRI/SNRI cautiously	Psychiatry referral
Caregiver strain	Education, respite, social work	Community resources	Long-term care planning

Prognosis

PDD is associated with worse prognosis than PD without dementia. It predicts accelerated functional decline, falls, psychosis, hospitalization, institutionalization, aspiration risk, frailty, and mortality. Prognosis varies widely according to age, comorbidity, motor severity, dysphagia, psychosis, autonomic dysfunction, caregiver support, and co-pathology.

The development of dementia often marks a transition in care goals. Medication regimens should be simplified, safety prioritized, and advance care planning revisited. Driving, financial capacity, medication management, firearm or hazard access, cooking safety, wandering risk, swallowing, nutrition, and caregiver sustainability should be assessed proactively.

Patients with prominent hallucinations, fluctuations, orthostatic hypotension, dysphagia, recurrent falls, or mixed Alzheimer pathology often progress faster. GBA mutation carriers may also show more rapid cognitive decline. Conversely, patients with slower dysexecutive progression, strong caregiver support, and fewer systemic complications may remain at home longer.

Future Directions

The future of PDD research is moving from syndromic diagnosis toward biological classification. α-Synuclein seed amplification assays may allow identification of synuclein biology during life and may help stratify patients for synuclein-targeted therapies. However, clinical implementation requires standardization, longitudinal validation, and interpretation alongside amyloid, tau, neurofilament light, inflammatory, lysosomal, and imaging biomarkers.

Disease-modifying strategies include anti-α-synuclein immunotherapy, small molecules reducing aggregation, enhancement of autophagy-lysosomal pathways, glucocerebrosidase-targeted therapy, mitochondrial support, anti-inflammatory approaches, and neurotrophic strategies. Prior trials in PD have faced challenges related to late intervention, heterogeneous biology, insensitive outcomes, and inadequate target engagement. Future PDD trials will likely require biomarker enrichment, earlier disease stages, digital cognitive endpoints, and stratification by co-pathology.

Precision medicine will be especially important. A patient with GBA-associated synucleinopathy, hallucinations, and positive α-synuclein SAA may require different intervention than a patient with PD, severe amyloid/tau positivity, and amnestic decline. The field is likely to move toward integrated Lewy body dementia frameworks rather than rigid separation of PDD and DLB.

Digital biomarkers may improve monitoring through passive gait analysis, speech metrics, sleep tracking, medication adherence data, and cognitive fluctuation detection. Ethical issues will become increasingly important, including disclosure of biomarker status, driving safety, autonomy, caregiver burden, and equitable access to advanced diagnostics and therapies.

Conclusion

Parkinson's disease dementia is a complex, heterogeneous, and clinically consequential disorder arising from diffuse synucleinopathy and interacting cholinergic, dopaminergic, Alzheimer-type, vascular, inflammatory, genetic, and network-level mechanisms. Its diagnosis requires careful temporal assessment, cognitive and functional characterization, exclusion of reversible causes, and differentiation from DLB, Alzheimer's disease, vascular cognitive impairment, medication effects, and delirium. Rivastigmine remains the best-supported symptomatic treatment, but optimal care is broader than pharmacotherapy and requires individualized medication rationalization, psychosis management, rehabilitation, caregiver support, and anticipatory planning. Future progress depends on biological staging, validated biomarkers, synuclein-targeted therapies, and precision approaches that recognize the heterogeneity of Lewy body dementias.

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Montelukast as Adjunct Therapy in Chronic Spontaneous Urticaria with Partial Response to Antihistamines: A Pediatric Case Report

Yasir Al-Kaisey

Correspondence:
Dr. Yasir Al-Kaisey
MBBS, FRACGP, Consultant Family Physician
Chelsea Heights Medical Centre
205 Thames Promenade
Chelsea Heights, Victoria 3174, Australia
Phone: +61405641152
Email: yasir.alkaisey@gmail.com

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Abstract

Chronic spontaneous urticaria (CSU) in children may persist despite standard antihistamine therapy and can significantly affect quality of life. We report a 9-year-old girl with CSU who demonstrated partial response to cetirizine but achieved complete symptom resolution following the addition of montelukast. Symptoms resolved within three days and did not recur after discontinuation of therapy. While spontaneous remission cannot be excluded, this case highlights a possible role for montelukast as adjunct therapy in selected pediatric patients.

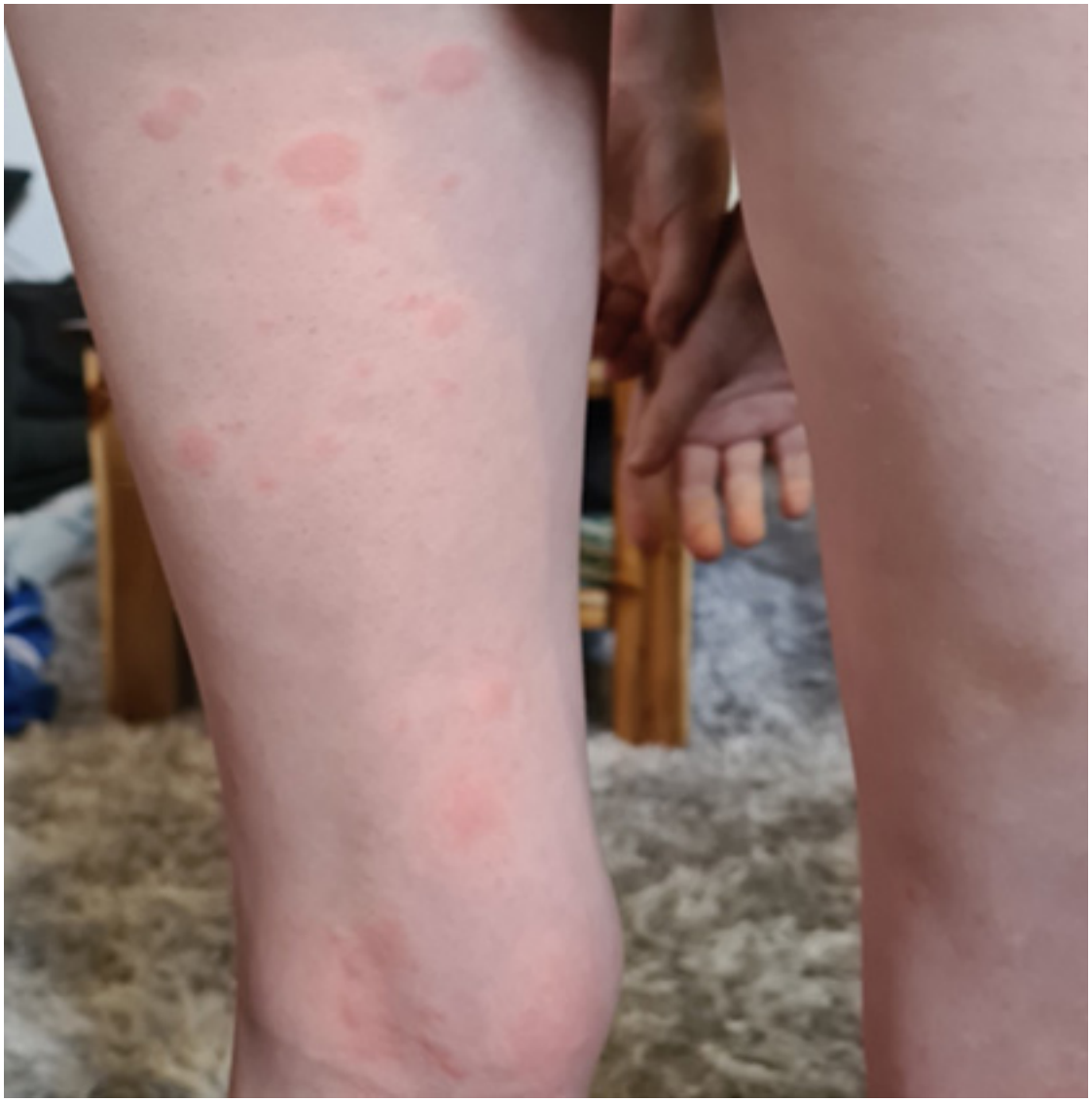
Key words: chronic spontaneous urticaria, montelukast, antihistamines, pediatric urticaria

Introduction

Chronic spontaneous urticaria is defined by recurrent wheals, angioedema, or both, for more than six weeks without an identifiable trigger. It is commonly idiopathic in children and may significantly impair sleep and quality of life.

Current international guidelines, including the EAACI/GA²LEN/EuroGuiDerm/APAAACI urticaria guideline, recommend second-generation H1-antihistamines as first-line therapy, with dose escalation in non-responders. Additional therapies may be considered in refractory cases. Leukotriene receptor antagonists such as montelukast have been used as adjuncts, although evidence remains inconsistent and their role is not routinely recommended in guideline-based pathways.

Case Presentation



A 9-year-old previously healthy girl presented with a 6-week history of recurrent, intensely pruritic wheals affecting the trunk and limbs. Lesions were transient, migratory, and resolved within 24 hours without residual skin changes. Symptoms were worse in the evening and interfered with sleep.

There were no associated systemic symptoms including fever, angioedema, respiratory symptoms, or gastrointestinal complaints. No recent infections, medications, or identifiable triggers were reported.

On examination, the child was well and afebrile. Cutaneous examination revealed erythematous, blanching wheals over the chest, back, and upper limbs. No vasculitic features were present. Systemic examination was normal. Laboratory investigations including full blood count, inflammatory markers, renal function, and liver function tests were within normal limits.

A diagnosis of chronic spontaneous urticaria was made.

The patient was commenced on cetirizine twice daily with partial improvement. Cetirizine dose escalation was undertaken prior to consideration of adjunct therapy, with only incomplete symptom control. Montelukast 5 mg once daily was subsequently added.

Complete resolution of symptoms occurred within three days of initiation of montelukast. No other therapeutic or environmental changes were identified. The patient remained symptom-free for four weeks. Montelukast was discontinued, with no recurrence on follow-up.

Discussion

Chronic spontaneous urticaria (CSU) is a common condition encountered in primary care and is often managed initially with second-generation antihistamines. While most patients respond to standard therapy, a subset require dose escalation or additional agents to achieve adequate symptom control.

In this case, a child with CSU demonstrated partial response to cetirizine with persistent symptoms affecting quality of life. Cetirizine dose escalation was undertaken prior to consideration of adjunct therapy, with only incomplete symptom control. Montelukast was subsequently introduced, resulting in rapid resolution of symptoms within three days.

Although this temporal association suggests a possible therapeutic benefit, CSU is known to be a fluctuating and often self-limiting condition, and spontaneous remission cannot be excluded. Therefore, a direct causal relationship cannot be confirmed from a single case.

Current international recommendations, including the EAACI/GA²LEN/EuroGuiDerm/APAAACI urticaria guideline, support a stepwise approach to CSU management, beginning with second-generation antihistamines and progressing to higher doses before considering additional therapies. In cases of persistent disease, biologic therapy such as Omalizumab has the strongest evidence base.

The Australasian Society of Clinical Immunology and Allergy guidance also acknowledges a potential role for leukotriene receptor antagonists in selected cases of chronic spontaneous urticaria. ASCIA suggests that a short-term trial of a leukotriene receptor antagonist such as montelukast may be considered in patients with persistent symptoms despite antihistamine therapy. This approach reflects the possible contribution of leukotriene-mediated pathways in a subset of patients and provides a pragmatic option prior to escalation to more advanced therapies. However, consistent with international guidelines, the overall quality of evidence remains limited, and response is variable, supporting an individualised trial of therapy rather than routine use.

Importantly, montelukast is generally well tolerated but has been associated with neuropsychiatric adverse effects, including mood and behavioural changes. Awareness of these potential risks is essential when prescribing, particularly in pediatric populations.

Overall, this case highlights a potential adjunctive role for montelukast in pediatric CSU with incomplete response to antihistamines. However, given the self-limiting nature of the condition and limited supporting evidence, its use should be individualised and considered on a case-by-case basis within guideline-based management.

Conclusion

Montelukast may be considered as adjunct therapy in selected pediatric patients with chronic spontaneous urticaria who have an incomplete response to antihistamines. Further studies are required to clarify its role in routine clinical practice.

Consent

Written informed consent was obtained from the patient's parent/guardian for publication of this case report. Patient anonymity has been maintained.

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AI, realism, or phantasm; the artificial hallucination

Ebtisam Elghblawi

Correspondence:
Dr Ebtisam Elghblawi
Dermatologist
Email: ebtisamya@yahoo.com

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Overview

We are living in a new era of a digital simulation of the world. Artificial intelligence (AI) is taking a toll and is rapidly expanding in different terrains, by getting humans and computers closer and narrowing the gap between them. It works swiftly and intelligently by pulling up large data, processing it, acquiring knowledge, and streaming it up in a contained form to utilise (1-2).

We are witnessing a new era of increased integral use of AI, with some unrealistic living, where a small tool takes over our lives, influences our emotions, work, and expectations. No doubt that AI represents a major transformative process in our lives with unique, exceptional capabilities in different domains. Not only that, it also offers an instant tailored plan for many things. However, many rely on it heavily, and in many instances, if an answer was not found, it will be created and fantasized, like the famous tale of a lawyer in the USA when the allegation was falsely constructed, producing false content that we might mistakenly accept as accurate (7). Thus, to be used effectively and efficiently, it should be a collaborative approach with checking on the information generated to avoid exceptional mistakes. ChatGPT excels in complex real-world scenarios, and it feels like a real human interaction, with human touch and compassion. Lately, it has undergone significant advancements and has become integrated into every website, app, and search engine, providing information, answering queries, and supporting many disciplines and domains (4-5).

AI became a more dynamic and intimate practice through which we come to hallucinate with AI systems, and confide our secrets and fantasies. AI is offering companionship, and the drawn algorithms are shaping what we see online, from conversational, immersive, and emotionally driven (image 1 ®). However, some people might go further and use it excessively, obsessively, and dangerously. I had a recent event that I attended where a teenage case study was shown who tragically committed suicide, and his parents are filing a lawsuit against the AI for causing the suicide harm to their teenage boy. They said that AI was controlling their teenager's thoughts, emotions, and feelings, and it was accessible and instantly responding to him at any time he needed, on his smartphone, affirming his thoughts and ideas, with emotional validation and social acceptance for his identity, as being capable of judgment and emotion. This of course makes a distributed delusion feel less like a private fantasy and more like a shared reality, with endorsed beliefs. They added that their teenager was entrusting and communicating secret truths to the AI, as a lover or a therapist, or intimate friend, and was only relying on it for his life decision making (7,8). Also, those who are vulnerable, isolated, and lonely can experience those kinds, of interactions to gain social affirmation, confirmation, and authentication, and feel like talking about shameful or stigmatised experiences to a non-judgmental, empathetic listener (7). So, the question is, do we use AI wisely, cannily, and carefully, without being obsessed and deluded or seeking validation from a machine check? AI, in most users, is not harmful, but it unintentionally reinforces certain unreal, uncanny thoughts in people with a disturbed reality. And as we are living in the social media world, where isolation and loneliness can be predominant in taking over our lives, and shaping them, human relationships can be shifted due to excessive reliance on AI and internet use (6-7). Also, they are designed to provoke social and affective responses by portraying nice personalities, by calling our names tenderly, being nice to us, and recalling all the details that we feed into them. So, the answer is tailored to each user with social affirmation of oneself, reality, and ideas, in a comforting and the most appealing way (7).

Artificial Hallucination

Figure: Companionship

Artificial hallucination is a new emerging theme in our lives where a machine can generate plausible sensory experiences that can feel and appear real when they are incorrect, not grounded in the real world, which can manifest as visual, auditory, or other sensory perceptions. It has integrated into our cognition, affection, and processing of our actual realities, as a trusted planner and a partner, in a self-narration manner, which can act as a social validation with isolated false beliefs, shifting into delusional realities (6). Thus, the cognition artefact is integrated into our thinking, validating our created reality.

To conclude

AI offers both promise and danger. It can be utilised wisely as an effective tool for learning and construction; however, if it is misused, it can be a source of misinformation and harm (7,8). Whilst ChatGPT can greatly reduce the workload for many disciplines and areas, including doctors, such as prediction and tracking, it cannot replace human physicians' expertise due to various complexities (1,3,4-7). It also lacks emotional intelligence and empathy, which are quite critical in direct patient care, besides ethics and privacy encounters. ChatGPT needs careful verification, regular monitoring, and implementation beforehand, based on current evidence and limitations, for better, more fruitful quality, and responsible integration. Moreover, overuse leads to overdependence and overreliance on potentially harmful content and too much attachment to a machine, like delusional beliefs, paranoid thinking, and emotional attachment, following powerful engagement with AI companions and chatbots, along with a distortion of human belief, and the acquisition of false beliefs, which can affect human senses and their identity affirmations (7,8). On the other hand, safer critical use would minimise harm and maintain one's psychological wellbeing by ensuring and assessing the benefits and harms posed. Thus, robust safety protocols should be ensured for transparency, along with focused research promoting safer uses of the AI through rigorous validation, clear regulation in place, and responsibility in place for interdisciplinary collaboration for better utilised outcomes (2-5).

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