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GUIDELINES

Guideline for the cognitive assessment and follow-up in the Guangdong-Hong Kong-Macao Greater Bay Area (2024 edition)

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Abstract

This practice guideline focuses on the cognitive assessment for mild cognitive impairment in the Guangdong-Hong Kong-Macao Greater Bay Area. To achieve the standardization and normalization of its clinical practice and generate individualized intervention, the National Core Cognitive Center of the Second Affiliated Hospital of Guangzhou Medical University, the Cognitive Disorders Branch of Chinese Geriatic Society, the Dementia Group of Neurology Branch of Guangdong Medical Association and specialists from Hong Kong and Macao developed guidelines based on China's actual conditions and efficiency, economic cost and accuracy. The article addresses the significance, background, and the process of the assessment and follow-up to realize the promotion and dissemination of cognitive assessment.

KEYWORDS
Alzheimer's disease, cognitive assessing, cognitive impairment, dementia

1 | BACKGROUND

Dementia is also known as a cognitive disorder or cerebral degeneration. The Diagnosis and Statistical Manual of Mental Disorders (fifth edition) (DSM-5) defines dementia as follows: one or more cognitive domains (such as complex attention, executive function, learning and memory, language, perceptual-motor function, and social cognition) have been impaired, and the cognitive impairment affects the ability to complete daily activities independently.[1] People with dementia in China account for approximately 25% of the global 55 million people with dementia.[2] Of the 249 million Chinese people aged 60 and older, 9.83 million have Alzheimer's disease (AD), and approximately 38.77 million experience mild cognitive impairment.[3] However, according to the Survey Report on the Status of Alzheimer's Disease

in China released by the Alzheimer's Disease Prevention Association in September 2022, the rate of AD patients with treatment in China was only 12.9%.

Most cognitive disorders are irreversible or progressive, with early stages of subjective cognitive decline (SCD) and amnestic mild cognitive impairment (aMCI).[4] SCD refers to decreased self-perceived cognitive function compared to self-prior states, and a soaring number of studies suggest that there might be evidence for AD-related pathological changes in the SCD population.[5] The risk of developing MCI or dementia within 15 years is 4.5 times higher for people with SCD, and more than 60% of the population will develop MCI or dementia within 15 years for people with SCD. Therefore, SCD can be considered a high risk of dementia.[6] Frisoni and other experts have proposed focusing on the primary and secondary prevention of dementia,[7] arguing that individuals without cognitive impairment should undergo cognitive evaluation, and that interventions targeting 12 risk factors for dementia can reduce the incidence of dementia by 40% worldwide.[8] International authoritative organizations have proposed assessing cognitive function in patients with complaints of memory loss or related functional symptoms in order to achieve early identification and early prevention.[9] Nonetheless, the evaluation criteria are still deficient. At the same time, China's National Health Commission and other departments jointly put forward the 14th Five-Year Plan to promote the development of disability (intellectual) prevention and intervention and encourage the pilot work of cognitive function assessment and early intervention for senile dementia. In response to the plan, the Department of the National Core Cognitive Center of the Second Affiliated Hospital of Guangzhou Medical University, the Cognitive Disorders Branch of Chinese Geriatic Society, and the Dementia Group of Neurology Branch of Guangdong Medical Association released the guidelines for cognitive assessment and follow-up in the Guangdong-Hong Kong-Macao Greater Bay Area (2024 edition) based on China's national conditions, especially the characteristics of the Guangdong-Hong Kong-Macao Greater Bay Area. From preparation to completion, we spent over a year perfecting early cognitive assessment, realizing individualized risk mitigation, and offering scientific and consistent direction.

We searched PubMed from 1990 to 2023 using the following terms: (Dementia or Cognitive Disease or Cognition Disorders or Cognitive Dysfunction or Cognitive Decline or Alzheimer Disease) and (Screening or Assessing). The Chinese databases include the CNKI, Wanfang Database and the VIP Chinese journal service platform. We divided the evidence levels of the search results into five grades: I, II, III, IV, and V, according to the evidence grading and recommendation criteria for the University of Oxford (Oxford Centre for Evidence-based Medicine, OCEBM).[10] For each recommendation, the expert group will first summarize, analyze and evaluate the current research evidence, try to select the most reliable evidence and the highest level of evidence that can be retrieved, and then obtain the recommendation levels according to the evidence and the experts' opinions. This guide is applicable to all clinicians, particularly neurologists, psychiatrists, and general practitioners in community hospitals.

2 | METHODS

2.1 | Scale introduction

2.1.1 | The Ascertain Dementia 8-item Questionnaire

The Ascertain Dementia 8-item Questionnaire (AD8) is an early screening scale for dementia that was developed at the University of Washington in 2005. It covers eight aspects of questions that encompasses four cognitive domains, namely memory, endurance, execution, and complex functions. It can be used for patient self-assessment or caregiver assessment, which takes approximately 3 min and can be carried out online or by telephone. It is both straightforward and effective. The AD8 questionnaire has a high correlation and parallel validity with the other screening scales, with high reliability and validity. It is also extremely useful in community hospitals, as well as primary and tertiary diagnosis and treatment institutions.[11]

2.1.2 | The Mini-Mental State Examination

The Mini-Mental State Examination (MMSE) is widely utilized in clinical cognitive screening assessments.[12] It covers multiple cognitive domains and takes 5–10 min, and the scores are influenced by educational levels. The sensitivity for detecting MCI was between 13% and 97%, and the specificity was between 60% and 100%.[13–15] A wide range of sensitivity and specificity indicates middle-level confidence in identifying early MCI and better superiority in identifying moderate cognitive dysfunction.[16]

2.1.3 | The Montreal Cognitive Assessment

The Montreal Cognitive Assessment (MoCA) is also a commonly utilized cognitive assessment scale in clinical practice. The MoCA covers multiple cognitive domains, including visual–spatial, executive, language, abstraction, attention, calculation, memory and orientation.[17] It takes 10 min and must be corrected according to education levels, thus limiting its application in community screening.[16] As the MoCA is more rigorous in evaluating the execution and visual–spatial capabilities, it exhibits greater sensitivity than the MMSE in distinguishing MCI (67%–100%).[18] It can identify cognitive impairment in a population with normal MMSE scores.[17,19] It can detect cognitive decline early before patients experience impaired living ability.[20] However, the MMSE exhibits a slight advantage in specificity (50%–95%).[17,18]

2.1.4 | Cardiovascular Risk Factors, Age and Dementia Disease Scale

The Cardiovascular Risk Factors, Age and Dementia Disease Scale (CAIDE) is the first tool to predict the risk of dementia after 20 years based on lifestyle and cardiovascular risk factors in middle-aged people.[21] A score above 9 can have a sensitivity of 0.77, a specificity of 0.63 and a negative predictive value of 0.98.[21] Increasing the APOE e4 has little impact on the accuracy of the scale predictions.[22] Higher CAIDE scores are associated with smaller whole-brain volumes, hippocampal volumes, and cortical thickness.[23,24] CAIDE can also provide a predictive value of the life span and quality of life in the subject population.[25] CAIDE is suitable for predicting the risk of dementia after 20 years in people aged 39–64 years. In people under 39 years of age, the risk of dementia after 20 years is low. Low accuracy was found after a short follow-up of the elderly population,[26] so it is not first intended for people aged 65 and above.[22] The scale is presented in Table 1.

TABLE 1 The CAIDE assessment.

Items	CAIDE Score	CAIDE Score	CAIDE (including APOE ε4 status) Score	CAIDE (including APOE ε4 status) Score
1. Age				
<47 Years old	0		0	
47–53 Years old	3		3	
>53 Years old	4		5	
2. Education				
≥10 years	0		0	
7–9 Years	2		3	
0–6 Years	3		4	
3. Gender				
Female	0		0	
Male	1		1	
4. Systolic blood pressure				
≤140 mm Hg	0		0	
>140 mm Hg	2		2	
5. BMI				
≤30 kg/m^2	0		0	
>30 kg/m^2	2		2	
6. Total cholesterol levels				
≤6.5 mmol/L	0		0	
>6.5 mmol/L	2		1	
7. Exercise[a]				
Acive	0		0	
Inactive	1		1	
8. APOEε4 status				
Non-ε4			0	
ε4			2	
Scores				

[a]Active participants should do recreational exercise at least twice a week; inactive participants should exercise less than twice a week.

2.1.5 | The Brief Screening Scale for Dementia

The Brief Screening Scale for Dementia (BDSI) was published by Deborah E Barnes in *Alzheimers Dement* in 2014. It was designed to assess the risk of dementia within 6 years in older adults without complaints or manifestations of cognitive impairment.[27] In the four cohort studies, including the Cardiovascular Health Study (cardiovascular health study, CHS), the Framingham Heart Study (Framingham Heart Study, FHS), the Health and Retirement Study (Health and Retirement Study, HRS), and the Latino Aging Study in the Sacramento Region (Sacramento Area Latino Study on Aging, SALSA), high accuracy was obtained after testing (AUC was between 0.68 and 0.78).[27] Those who scored ≥22 are considered at high risk. However, high-risk individuals aged 65–79 and normal-risk individuals aged 80–84 have an equal risk of developing dementia within 6 years, so the BDSI is recommended for dementia risk prediction in people aged 65–79 years. Specific items of the scale are shown in Table 2.

2.2 | Biological markers and imaging techniques

In 2018, the National Institute on Aging and the Alzheimer's Association (National Institute on Aging and Alzheimer's Association, NIA-AA) established an AD survey framework, defined as "ATN". "A" represents evidence of amyloid (Aβ, beta-amyloid) deposition; "T" represents evidence of fibrous tau; and "N" represents the neurodegeneration or neuronal damage. Aβ deposition represents the earliest evidence of pathological changes in AD neuropathy, and Aβ plaque deposition alone can serve as an indication of AD.[28] However, to meet the neuropathological criteria for AD, evidence of both Aβ plaque deposition and phosphorylated tau (P-tau) deposition is required.[29] The evidence of neuronal damage is not a specific pathological change in AD but it can provide insight into the course and severity of the disease. However, despite the theoretical classification of biomarkers, there are limitations in clinical application. They are more applicable to the research framework than the clinical diagnostic criteria. Moreover, it is difficult to apply CSF detection due to the low acceptance of lumbar puncture. Recent results from Shen Yong's team at the First Affiliated Hospital of University of Science and Technology of China have demonstrated that during the progression of brain atrophy, CSF and plasma p-tau showed the same accuracy in distinguishing between AD and the normal population. Furthermore, plasma p-tau and p-tau/t-tau had higher accuracy in distinguishing non-AD dementia and AD, as well as in predicting Aβ deposition in the brain. Therefore, plasma p-tau is more indicative of brain atrophy than Aβ42/Aβ40.[30] The International Working Group (IWG) believes that the diagnosis of AD requires not only biomarkers but also the

TABLE 2 The BDSI assessment.

BDSI		
Do you think that your patient may have a cognitive impairment, because of:		
☐ Your observation		
☐ Patient concerns		
☐ Concerns of family members or others		
If you answered yes: your patient should be screened for cognitive impairment.		
Is your patient aged 80 or older?		
☐ Yes		
☐ No		
If Yes: your patient should be screened for cognitive impairment.		
If No: Use Cognitive Disorder (Dementia Screening Indicator)		
Screening indicators for cognitive impairment (Dementia Screening Indicator)		Score
1. How old is your patient?[a]		
2. Does your patient have less than 12 years of education?[b]	No (0)	Yes (9)
3. The BMI of your patient was <18.5 kg/m^2?[c]	No (0)	Yes(8)
4. Does your patient have a history of type 2 diabetes?	No (0)	Yes (3)
5. Has your patient ever had a stroke?	No (0)	Yes (6)
6. Does your patient need help to manage money or medication?[d]	No (0)	Yes (10)
7. Is your patient currently taking antidepressant medication, or complainting that it was "difficult to do everything" 3 days a week for the past week?[e]	No (0)	Yes (6)
Total score: If ≥ 22, your patient should be screened for cognitive impairment		

[a]If aged 65–79, aged 65 gets 0 points, and add 1 point for each year older.

[b]Never graduated from high school or never passed the General Education Development Certificate (GED) exam.

[c]http://www.nhlbi.nih.gov/guidelines/obesity/BMI/bmicalc.htm.

[d]Ask your patient and/or family members (if possible): Do you need help to manage money or medication?

[e]Ask your patient: How many days in the past week have you found yourself struggling with doing everything? Few times (<1 day); Sometimes (1–2 days); Half time (3–4 days); And most times (5–7 days); If the answer is "half time" or "most times", 6 points on point 7 in the Cognitive Disorder Screening Index (Dementia Screening Indicator).

following criteria: (1) Progressive clinical manifestations and aggravated memory decline; objective evidence of memory decline; (2) In vitro evidence of pathological changes in AD, such as decreased CSF Aβ1-42 and increased T-tau or P-tau; Aβ-PET is positive; mutations in AD-related genes on the chromosome, such as presenilin 1 (PSEN 1), presenilin 2 (PSEN 2), and amyloid precursor protein (APP); (3) Dementia caused by cerebrovascular diseases, toxicity, inflammatory diseases, metabolic diseases, and other diseases unrelated to AD was excluded.[31] Combining the NIA-AA and IWG, it is easy to summarize that cognitive assessment for dementia requires a combination of clinical considerations and biomarkers.

Structural magnetic resonance imaging (sMRI) can provide information about on the structure of the brain tissue.[32] It is widely used clinically for the noninvasive diagnosis and prediction of dementia.[33] The hippocampus is the first region on sMRI to reflect dementia-related brain atrophy.[34] An over 0.395% decrease in hippocampal volume can be regarded as positive. The AD resemblance atrophy index (AD-RAI) is an index based on whole-brain MRI and can be used to distinguish normal populations from AD patients and to determine whether MCI is at risk of conversion to AD.[35,36] AD-RAI >0.5 for distinguishing AD from the normal population can achieve a sensitivity of 88% and a specificity of 96%.[36] The AD-RAI is an appropriate assessment index for the elderly in AD or the preclinical period.[37] The accuracy of sMRI for AD, dementia with Lewy bodies (DLB), and frontotemporal dementia (FTD) can be 87%, 95%, and 90%, respectively.[38] The affected regions of AD are mainly in the medial temporal lobe, left temporal lobe, parietal lobe, and insula. More subcortical distribution can be observed in DLB, including the amygdala, dorsal midbrain, anterior temporal lobe, and left middle temporal lobe. The affected regions of the behavioral variant FTD were mainly in the frontal, anterior cingulate, anterior and medial temporal, inferior temporal, and parietal lobes.[38] Therefore, sMRI can be used for screening different dementia etiologies.

PET/SPECT is used to portray the function of the synapses and the deposition of Aβ.[39] ^{18}FFDG (fluorodeoxyglucose)-PET can measure the glycometabolism capacity of the brain and reflect the neurologic changes before sMRI changes. Thus, it is more suitable for the early assessment of neurodegeneration. Compared to CSF or MRI, ^{18}FFDGPET is superior in predicting the transition from mild cognitive impairment (especially short-term progression) to Alzheimer's dementia.[40] The negative predictive value of MCI due to AD can be up to 77% to 95%.[41,42] In addition, the cerebral metabolism of ^{18}FFDGPET varies in different neurodegenerative diseases. AD mainly shows hypometabolism in the posterior cingulate gyrus and frontal parietal lobe, whereas FTD hypometabolism is predominantly found in the dominant lateral frontal lobe (behavioral variability), around the lateral fissure (nonfluent aphasia), and the anterior temporal lobe (semantic dementia). Therefore, ^{18}FFDGPET is used for the various diagnosis of unexplained dementia or AD[43] and is included in the diagnostic criteria for different neurodegenerative diseases, including FTD,[44] primary and progressive aphasia,[45] DLB,[46] and progressive supranuclear palsy.[47] However, the ^{18}FFDGPET does not reflect the pathological changes. Aβ-PET can identify the deposition of Aβ plaques, which is considered the classic pathological change of AD but not the core change of FTD. Thus, there is high confidence in the negative signal of Aβ-PET to exclude AD, and Aβ-PET can be widely used to distinguish AD from FTD. However, for diseases with pathological changes in the same Aβ deposits, the diagnostic value of Aβ-PET is low. Therefore, the

population meeting the appropriate criteria may benefit, including (1) uncertainty of dementia in patients with MCI; (2) AD-related dementia symptoms with atypical clinical manifestations or possible confounding causes; and (3) patients with early-onset cognitive decline.[48] DAT-SPECT imaging (dopamine transporter-single photon emission CT) can be used to identify dopaminergic deficits and is recommended for DLB and AD discrimination, especially with core symptoms of DLB, such as fluctuating cognitive decline, visual hallucinations, or REM sleep behavior disorders.[49] Different combinations of clinical markers and PET imaging techniques can be used for the different aetiologies of dementia. However, the cost limits the development of functional brain PET imaging, which is currently used more for population screening in clinical studies. PET imaging technology can support diagnosis when etiology diagnosis is difficult or when atypical clinical manifestations are present.

3 | RESULTS

3.1 | Cognitive assessment and the follow-up process

After conducting a thorough examination of the guidelines and literature in relevant fields, taking into account the sensitivity, specificity, and time efficacy of the assessment methods, we made the subsequent recommendations after discussion by the expert group.

3.1.1 | People presenting complaints of cognitive decline

Assessment process

The AD8 scale should be used for people with complaints of cognitive decline, whether subjectively or by caregiver observation. If the AD8 is normal, a risk assessment should be performed. Patients with AD8 ≥ 2 could be considered to have a high risk of dementia and should be assessed further. Identification for dementia requires first excluding the presence of consciousness or depression, and those with moderate or severe depression need to be reassessed after treatment of depressive symptoms. A high suspicion of dementia should be made in individuals who have experienced memory loss or other cognitive decline for a duration of up to 6 months.[50] Further cognitive assessments, such as the MMSE and MoCA, must be completed with the help of a general practitioner or specialist.

At the same time, it is extremely important to collect clinical history, complete physical examination, and auxiliary examination in order to clarify the causes of cognitive decline and possible comorbidities, exclude reversible causes, and reverse them in time. Clarify the characteristics of the onset, including the speed of onset, the course of the disease, the impairment of the cognitive domains, the impairment of life ability, and whether the disease is accompanied by mental symptoms, the existence of hallucinations, neurological positioning signs, and other accompanying symptoms. The abuse of addictive substances such as alcohol, psychotropic drugs like sleeping pills and anxiolytic drugs can cause a progressive decline in cognitive function, hence it is imperative to establish the pertinent medication history.[51] Family history is critical to identify whether dementia is inherited. For neurological examinations, we must consider the objective existence of nervous system damage and clarify whether there are mental symptoms with obvious executive function abnormalities (FTD), whether there are nervous system positioning signs (e.g., vascular dementia, VaD), whether supranuclear eye movement disorder (progressive supranuclear paralysis), tremor, festinating gait (Parkinson's disease dementia, DLB), and involuntary jitter (Huntingdon's disease) are conducive to clarifying the possible causes of cognitive dysfunction. At the same time, the necessary systemic physical examination will help to exclude some rare causes of cognitive dysfunction, such as the Kayser-Fleischer ring around the cornea in Wilson's disease. Infection, chronic anemia, vitamin B1/B12 deficiency, liver and kidney insufficiency, hypothyroidism, sexually transmitted diseases, tumors, and heavy metal poisoning can cause chronic cognitive decline, so routine blood, urine, and stool examinations should be performed. The utilization of blood biochemical, vitamin B1/B12, thyroid function, sexually transmitted disease-related antibodies, tumors, heavy metals, poisons, and other laboratory tests will also help to eliminate chronic metabolic, toxicity, and infectious encephalopathy. The MRI can detect infarcts or cerebral white matter lesions, and microhemorrhage lesions in SWI will suggest the possibility of VaD. The “morning glory sign” or “hummingbird sign” highly indicates the presence of progressive supranuclear palsy, while MRI can clarify the presence of hydrocephalus, brain tumors and other abnormalities. EEG is helpful in judging changes in brain function and clarifying whether there are seizures, and periodic synchronous distribution is important in the diagnosis of K-Jakob disease. The trophy of the frontal or anterior temporal lobe on sMRI strongly suggested the possibility of FTD.

The diagnosis of AD requires an emphasis on biomarker changes, including ① sMRI imaging results of AD-RAI≥0.5 or hippocampal volume reduction (≤0.395%) and ② abnormal AD biological markers such as plasma p-tau181 and p-tau217. Identifying the causes of cognitive dysfunction requires a medical history, physical examination, and auxiliary examination. Then, we can determine the cause of cognitive dysfunction and decide the corresponding intervention or follow-up. However, for the identification of prodromal disease or atypical symptoms, especially between AD and other cognitive dysfunctions caused by frontotemporal lobe degeneration or neuropathy, we should perform special examinations, which may provide additional evidence. However, the examinations are only available in tertiary hospitals or cognitive centers. A decrease in CSF Aβ1-42 and increased tau or p-tau levels can support the diagnosis of AD. When there are confounding causes, brain functional imaging such as ^{18}FFDGPET can provide some clues for the diagnosis. For patients who refuse lumbar puncture, those who are contraindicated for CSF collection, and those with uncertain CSF results due to technical problems or the levels of biomarkers are near-threshold values,

brain function PET imaging is recommended for the identification of dementia causes, especially for those with mild functional impairment, which has guiding significance for clinical prognosis. Given the elevated suspicion of AD, it is highly recommended to utilize precise CSF markers and Aβ-PET. DAT-SPECT imaging is recommended for cognitive impairment with motor symptoms of Parkinson's syndrome. If the reasons for cognitive impairment are still not clear, ^{18}FFDGPET is recommended.[48] See Figure 1 for specific procedures.

Recommendation: (1) Perform AD8 scale assessment in people with cognitive decline, whether subjective or observed. For AD8 ≥ 2, it is necessary to further complete the cognitive function assessment (Evidence level: I; Recommendation level: A); (2) MMSE can be used to further improve the cognitive function assessment

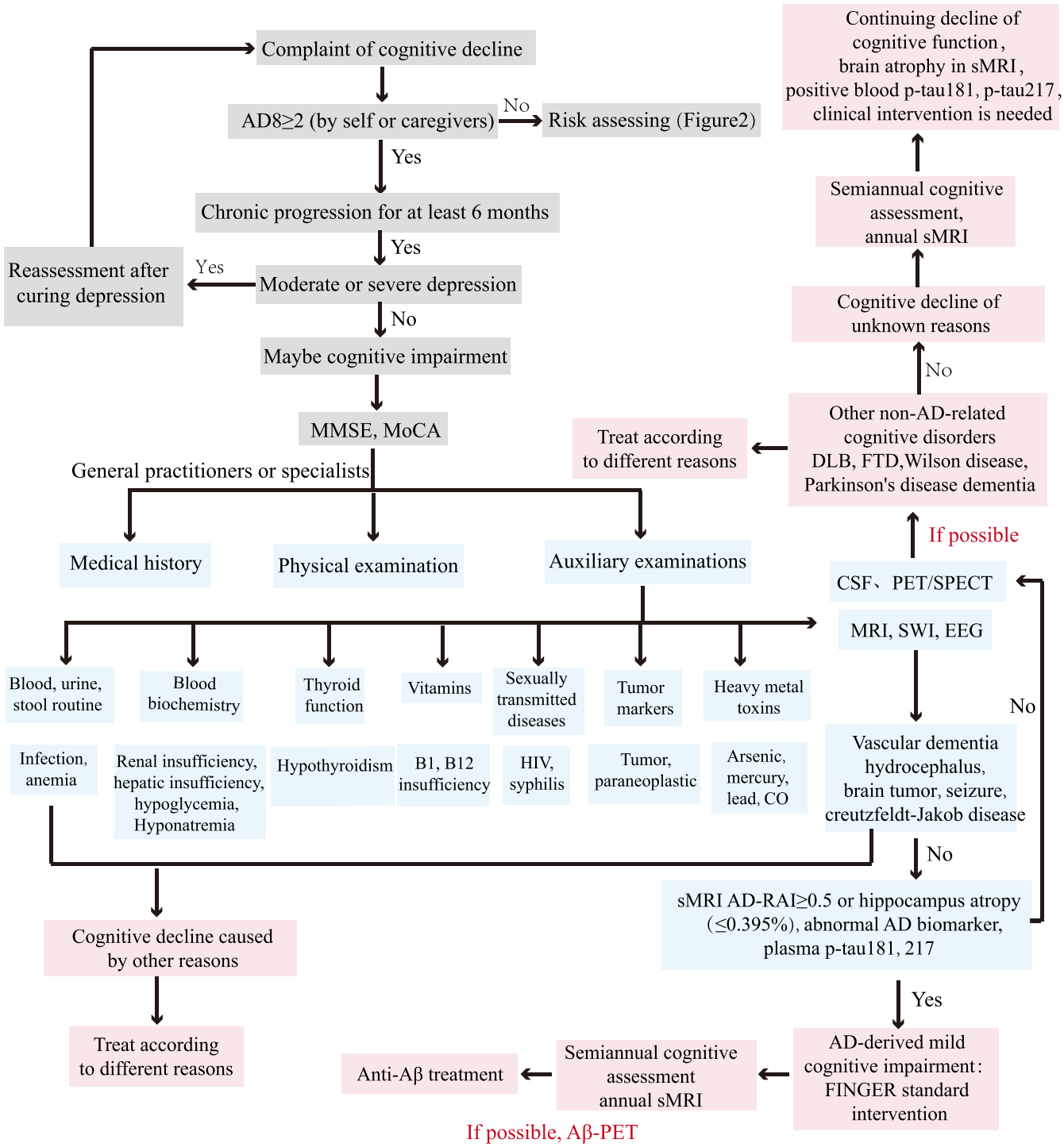

FIGURE 1 Cognitive assessment process in people with complaints of cognitive decline. The parts in gray can be performed in primary hospitals or community hospitals; the parts in blue can be performed in general or specialized departments; and the parts in red can be performed in Class A tertiary hospitals or cognitive centers.

(Evidence level: I; Recommendation level: A), MoCA (Evidence level: II; Recommendation level: B) can be used to identify mild cognitive impairment; (3) For those with abnormal MMSE or MoCA scores, further general practitioners or specialists are required to improve medical history collection, physical examination and auxiliary examination (Level of evidence: II; Recommendation grade: B); (4) sMRI imaging results AD-RAI ≥ 0.5 or relative hippocampal volume reduction (≤0.395%), high probability of AD-related cognitive dysfunction (Level of evidence: II; Recommended grade: B).

3.1.2 | The follow-up process

(1) Cognitive decline caused by other reasons: there are clear causes or non-AD-related evidence of cognitive dysfunction, such as infection, anemia, liver and kidney insufficiency, hyponatremia, hypothyroidism, B1/B12 lack, and cerebral small vessel disease. We need further guidance according to the specific causes and treatment. (2) Other non-AD-related cognitive disorders, such as DLB, FTD, Parkinson's disease dementia, Wilson disease, Huntington's disease, corticobasal ganglia degeneration, and progressive supranuclear paralysis, which are further managed and treated according to different causes. (3) AD-derived mild cognitive impairment: the intervention can be conducted according to the multidomain cognitive intervention protocol (The Finnish Geriatric Intervention Study to Prevent Cognitive Impairment and Disability, FINGER study).[52] These include nutritional counseling and eating brain-friendly foods (such as leafy greens, berries, fish, olive oil, nuts, whole grains, and beans, etc.).[53] Exercise (such as gradual muscle strength training, balance improvement, regular aerobic exercise, etc.), cognitive training (including enhanced executive functions such as planning and organizing tasks, processing speed, memory, etc.), management of cardiovascular and metabolic factors to prevent and delay cognitive decline, and supplementation of brain nutrients.[54,55] Ask them to complete their personal life planning in the future, such as personal life or financial planning, arrangement, attorney authorization, etc. Also, the education and support for the caregiver. Cognitive follow-up assessments were conducted every 6 months, and sMRI was performed annually. In such a population, further Aβ-PET examination is recommended, and patients with positive signals should be treated with anti-Aβ drugs. (4) Cognitive decline of unknown causes: There were no known causes of cognitive impairment, the sMRI imaging was normal, AD-RAI <0.5, and hippocampus >0.395%; AD biomarkers such as blood p-tau181 and p-tau217 results were normal; and there were no other known causes of cognitive decline. For such populations, a semiannual cognitive assessment and an annual sMRI are both needed. If cognitive function continues to decline, sMRI reflects the severity of brain atrophy, or blood p-tau181 and p-tau217 results are abnormal, clinical intervention is needed according to the reasons.

Recommendation: For AD-derived mild cognitive impairment, the FINGER standard intervention program is recommended (Evidence level: I; Recommendation level: A).

3.2 | People without cognitive decline complaints or with complaints but normal AD8 scores

For this type of population, especially those with a family history of dementia or constipation, olfactory loss, sleep disturbance, hearing impairment, and other non-specific symptoms, we need to be alert to the possibility of progression to cognitive impairment. Therefore, we recommend improving risk assessment for this population (Figure 2). Age is one of the risk factors for dementia, and different risk factors could be found in different age groups. To achieve an accurate individualized intervention, we divided the participants into three groups: age < 65 years, 65 ≤ age < 80 years, and ≥ 80 years.

3.2.1 | People under 65 years

Perform the CAIDE assessment and assess for other reversible cognitive risk factors other than those in CAIDE, such as diabetes, smoking, alcohol consumption, depression, hearing impairment, unhealthy eating habits or inadequate fish intake, lack of social activity or loneliness.[56] ① Those with CAIDE<10 can be considered a low-risk population, and an annual CAIDE assessment and follow-up should be performed. Reversible risk factors, such as lack of physical exercise, should be reversed. ② Those with CAIDE≥10 points and normal head sMRI results should dynamically detect and treat any reversible cognitive risk factors, perform annual CAIDE assessment and AD8 scale score, and perform sMRI every 2–3 years. ③ Those with CAIDE≥10 points and abnormal sMRI results need to be given a comprehensive examinations by general practitioners or specialists. If they are identified as AD-derived mild cognitive impairment, they should be treated according to the recommendations in the "AD-derived mild cognitive impairment" above.

3.2.2 | 65 ≤ age < 80 years

Perform the BDSI assessment and assess for any other reversible cognitive risk factors, such as smoking, drinking, hearing impairment, unhealthy eating habits or inadequate fish intake, lack of social activity or loneliness. ① Those with BDSI <22 are considered at low risk, and we should undergo an annual BDSI assessment and treat any modifiable risk factors, such as hypertension, diabetes, obesity, and physical inactivity. ② People with BDSI≥22 and normal sMRI should dynamically screen and treat any reversible cognitive risk factors, perform DSI and AD8 annually, and perform sMRI every 2 years. ③ BDSI≥22 and abnormal sMRI should be transferred to the general practitioner or specialist for a comprehensive examination. If they are recognized as AD-derived mild cognitive impairment, they should be treated according to the recommendations in the "AD-derived mild cognitive impairment" above.

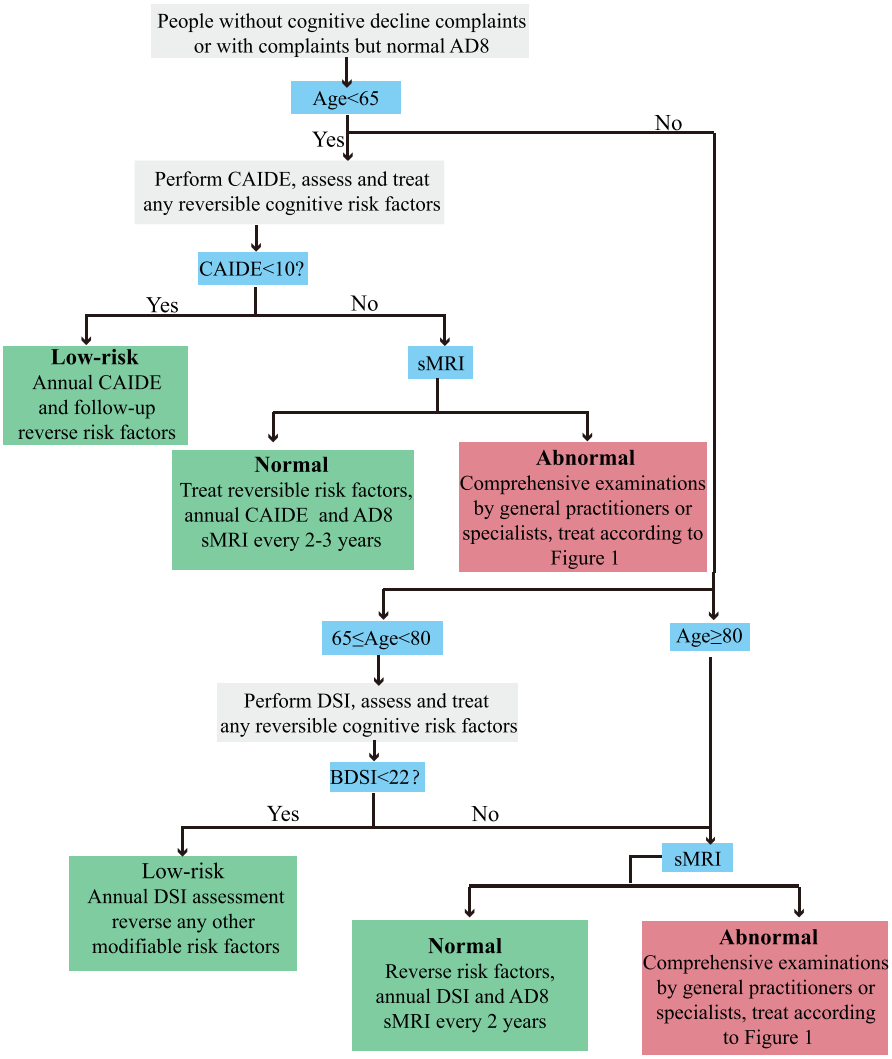

FIGURE 2 The process of risk assessment in people without cognitive decline complaints or with complaints but normal AD8 scores.

3.2.3 | Age ≥ 80 years

sMRI is recommended first. ① For those with normal results, we need to manage any reversible cognitive risk factors and undergo annual BDSI and AD8 assessments and head sMRI every 2 years. ② Those with abnormal results need to be given comprehensive examination by the a general practitioner or specialist. If they are recognized as AD-derived mild cognitive impairment, they should be treated according to the recommendations in the "AD-derived mild cognitive impairment" above.

Recommendation: Different risk screening scales should be applied to different age groups. The CAIDE scale was chosen for risk assessment in people under the age of 65 (level of evidence: I; level of recommendation: A); for people 65 ≤ age < 80 years, the BDSI scale was chosen (level of evidence: I; level of recommendation: A).

4 | DISCUSSION

Although studies on dementia are emerging and the mechanisms of mild dementia are uncovering, early assessments of SCD and MCI are still needed. At present, the challenge of aging has emerged in China, and dementia has become a major public health problems. Cognitive assessment, early intervention, education of patients and their families, and dealing with the patients' social relations would help to greatly reduce the economic burden on patients, families and society. The Guangdong–Hong Kong–Macao Greater Bay Area has unique geographical advantages and rapid economic development, keeping up with the world frontier and rapid medical-related technologies. Cognitive centers have been established, and the hierarchical diagnosis has been enhanced, providing a solid foundation for the development of cognitive assessment. Compared to the other screening guidelines,[57,58] our process has the following characteristics: (1) Most screening solely focuses on scales, which are too intricate to implement in the community hospital. This guideline recommends utilizing a straightforward AD8 scale in conjunction with MMSE and MoCA scales. It is easy to complete and has a high degree of applicability. We combined medical history, physical examination, imaging and tests to realize individualized intervention and follow-up, which reflects the concept of "precision". (2) This guideline includes a risk assessment to achieve age-stratified prevention, reflecting the concept of "early prevention", which is different from

the majority of existing screening. (3) This guideline classifies the assessment process into three parts: primary hospitals or community hospitals, general or specialties, and tertiary hospitals or cognitive centers, and puts forward the concept of "graded screening", which is beneficial for promoting the construction of cognitive assessments and work networks of intervention. It is critical to balance the individual, economic, and social benefits and burdens when promoting cognitive assessment. We consider the efficiency, economic cost, and accuracy, multidimensional optimization of methods, standardization of the assessment of dementia, promotion of the early detection of dementia, and realization of precise individualized intervention. However, future practice is still needed.

AUTHOR CONTRIBUTIONS

PJL, retrieval, visualization and original draft; MYR, review and Editing; CDBCGS and DGNBGMA, ideas and development of guidelines; LJ, ideas and development of guidelines, supervision and funding acquisition. All authors read and approved the final manuscript.

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CONFLICT OF INTEREST STATEMENT

All of the authors declare that there are no conflicts of interest.

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