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# Reversible Causes of Cognitive Decline in Young Patients: A Narrative Review

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## Abstract

Young-onset cognitive decline represents a major diagnostic challenge because, unlike age-related neurodegenerative dementias, a considerable proportion of cases are potentially reversible if recognized and treated promptly. This narrative review aimed to summarize current evidence regarding the etiology, pathogenesis, diagnostic approaches, and treatment strategies for reversible causes of cognitive decline in young patients. A literature search was conducted in PubMed, Scopus, Web of Science, and Google Scholar using keywords related to reversible dementia, young-onset cognitive impairment, metabolic encephalopathy, autoimmune encephalitis, and treatable causes of dementia. Original studies, systematic reviews, meta-analyses, and clinical guidelines published in English within the last 10 years were analyzed.

The review demonstrated that metabolic and deficiency-related disorders, including vitamin $B_{12}$, folate, and thiamine deficiency, are among the most frequent reversible causes of cognitive impairment in younger individuals. Infectious conditions such as neurosyphilis, HIV-associated neurocognitive disorder, Lyme neuroborreliosis, viral encephalitis, and tuberculous meningitis may mimic dementia but often respond to etiological therapy. Autoimmune encephalitis, toxic exposures, medication-induced cognitive dysfunction,

structural brain abnormalities, and psychiatric disorders, particularly depression-related pseudodementia, also play an important role. The diagnostic process should include detailed clinical assessment, laboratory screening, neuroimaging, and cerebrospinal fluid analysis when indicated. A multidisciplinary approach involving neurologists, psychiatrists, endocrinologists, infectious disease specialists, and other healthcare professionals is essential for timely diagnosis and effective treatment.

Early identification of reversible causes of cognitive decline significantly improves prognosis and may result in substantial or complete restoration of cognitive function in young patients.

**Keywords: Dementia; Cognitive Dysfunction; Young-Onset Dementia; Vitamin $B_{12}$ deficiency; Encephalitis; Neurosyphilis; HIV-Associated Neurocognitive Disorders; Hydrocephalus, Normal Pressure; Depression; Differential Diagnosis.**

Dementia is traditionally associated with older age; however, its occurrence in young patients represents a distinct clinical and diagnostic challenge [1, 2]. Unlike degenerative processes that predominate in older populations, a substantial proportion of cognitive impairment in younger individuals is potentially reversible [1, 3]. Therefore, the concept of “reversible dementias” is of particular importance in this population, as timely identification of the underlying cause may lead to significant improvement or complete restoration of cognitive function [3, 4].

The main causes of reversible dementias in younger patients include metabolic and endocrine disorders, deficiency states (particularly vitamin $B_{12}$ and folate deficiency), infectious diseases of the central nervous system, autoimmune processes, toxic exposures, adverse drug effects, and structural brain abnormalities, including normal pressure hydrocephalus [3, 4]. Psychiatric conditions also play an important role, especially depression, which may mimic the clinical presentation of dementia (so-called pseudodementia) [5].

Clinical heterogeneity and the nonspecific nature of symptoms complicate early diagnosis, often resulting in delayed treatment. In this context, a multidisciplinary approach involving neurologists, psychiatrists, endocrinologists, and other specialists, as well as the use of modern neuroimaging and laboratory techniques, is of particular importance [6].

The aim of this article is to summarize current knowledge regarding the etiology, pathogenesis, diagnostic algorithms, and treatment options for reversible dementias in young

patients, thereby increasing clinicians' awareness and improving the prognosis of this patient population.

**Materials and Methods**

This narrative literature review was conducted to synthesize current evidence on reversible dementias in young patients. The literature search was performed using the international scientific databases PubMed [7], Scopus [8], Web of Science [9], and Google Scholar [10]. Additionally, the reference lists of relevant publications were screened to identify further potentially significant sources.

The search strategy included combinations of English-language keywords and their synonyms: "reversible dementia," "young-onset dementia," "early-onset cognitive impairment," "treatable causes of dementia," "secondary cognitive decline," "metabolic encephalopathy," "vitamin deficiency," "autoimmune encephalitis," and "normal pressure hydrocephalus." Boolean operators (AND, OR) were used to enhance search sensitivity, along with filters for age (young adults, <65 years), study type, and publication language [11]. The depth of search was 10 years.

The inclusion criteria were: (1) original studies, systematic reviews, and meta-analyses addressing reversible or potentially treatable causes of dementia; (2) studies involving young or middle-aged patients (under 65 years); (3) publications in English; and (4) studies covering clinical, diagnostic, or therapeutic aspects of the topic. Key clinical guidelines and consensus statements were also included.

The exclusion criteria were: (1) studies focusing exclusively on irreversible neurodegenerative dementias without consideration of potentially reversible conditions; (2) isolated case reports lacking generalizable value; (3) publications with insufficiently described methodology; and (4) duplicate publications or earlier versions of the same studies.

Selected sources were subjected to qualitative analysis with a focus on etiological factors, clinical manifestations, diagnostic approaches, and treatment effectiveness. Due to the narrative nature of the review, a formal assessment of the risk of bias was not performed; however, priority was given to recent, peer-reviewed, and highly cited studies. Totally 40 sources were selected for further analysis.

**Results.** Metabolic and deficiency-related disorders occupy a leading position among the causes of potentially reversible cognitive decline in young patients, which determines their particular clinical importance in the context of early diagnosis and timely therapy [12-14]. Unlike neurodegenerative processes, these conditions are often characterized by functional or biochemical changes that, if corrected, may lead to complete or substantial

restoration of cognitive function [15]. One of the most extensively studied causes is vitamin $B_{12}$ (cobalamin) deficiency, which disrupts methylation processes, myelin synthesis, and leads to the accumulation of neurotoxic metabolites, including homocysteine and methylmalonic acid. Clinically, this manifests as a combination of cognitive impairment and neurological symptoms, including paresthesia, ataxia, and peripheral neuropathy [12, 13, 16]. Importantly, neurological manifestations may precede cognitive deficits, and early replacement therapy can completely reverse symptoms.

A similar, though less pronounced, effect is observed in folate deficiency, which is also associated with impaired homocysteine metabolism and neurotransmitter synthesis [12, 17]. Particular attention should be paid to the fact that isolated folate supplementation may mask the hematological manifestations of vitamin $B_{12}$ deficiency while failing to prevent the progression of neurological impairment [16, 17]. Thiamine (vitamin $B_1$) deficiency, characteristic not only of chronic alcohol use but also of malnutrition or post-bariatric surgery states, underlies Wernicke encephalopathy and Korsakoff syndrome, which present with severe memory impairment, disorientation, and confabulations. Early parenteral thiamine therapy is a key condition for the reversibility of these changes [12, 13, 18-20].

Electrolyte disturbances, particularly hyponatremia and hypercalcemia, make a substantial contribution to the development of cognitive dysfunction by affecting neuronal excitability and synaptic transmission, leading to confusion, reduced attention, and slowed thinking [21, 22]. Systemic metabolic encephalopathies, such as hepatic and uremic encephalopathy, are also of major importance; they result from the accumulation of toxic metabolites (including ammonia and uremic toxins) and are accompanied by fluctuations in the level of consciousness and varying degrees of cognitive impairment [23-25]. Disturbances of glucose homeostasis should be highlighted separately: both acute hypoglycemia and chronic glycemic variability may lead to transient or persistent cognitive changes [16-28]. It is important to emphasize that most of these conditions are highly reversible when detected early and treated appropriately, which necessitates their mandatory exclusion in young patients presenting with cognitive decline.

Infectious lesions of the central nervous system constitute an important and potentially reversible group of causes of cognitive decline in young patients, characterized by significant clinical variability and often subacute or chronic courses [3, 29-39]. Unlike acute meningitis and encephalitis, which present with pronounced neurological symptoms, some neuroinfections may initially manifest predominantly with cognitive and behavioral disturbances that mimic dementia. Among the most significant and potentially treatable

etiological factors is neurosyphilis, which in its late form presents with progressive memory impairment, personality changes, psychotic symptoms, and executive dysfunction [29, 30]. Timely diagnosis using serological tests and cerebrospinal fluid analysis allows for significant clinical improvement with appropriate antibacterial therapy.

HIV-associated neurocognitive disorder also occupies a prominent place, ranging from mild cognitive impairment to severe dementia [31, 32]. Its pathogenesis is associated with chronic neuroinflammatory activation, direct viral effects, and secondary opportunistic infections. Importantly, early initiation of antiretroviral therapy can stabilize or even improve cognitive function. Among opportunistic infections, central nervous system toxoplasmosis deserves special attention, as it may be accompanied by focal neurological symptoms and cognitive impairment, particularly in immunocompromised individuals, and responds well to specific therapy [31, 33].

A separate group is formed by viral encephalitides, particularly those caused by Herpes simplex encephalitis, which may leave persistent cognitive deficits even after the acute phase, predominantly affecting the medial temporal structures [34]. With early initiation of antiviral therapy, partial or substantial functional recovery is possible. At the same time, a number of infections have a subacute course with gradual progression of cognitive impairment, which complicates their recognition. Among these is Lyme neuroborreliosis, which may manifest with impaired concentration, memory deficits, and asthenic symptoms, and responds well to antibacterial treatment [35, 36].

Tuberculous meningitis is also an important differential diagnostic consideration, as it may present subacutely with predominance of cognitive and behavioral changes, especially in the early stages, and requires prolonged specific therapy [37]. Overall, infectious causes of cognitive decline in young patients are characterized by potential reversibility when detected early and treated with appropriate etiological therapy, which justifies their active search within the framework of differential diagnosis [38, 39].

Among other causes of potentially reversible cognitive decline in young patients, autoimmune, toxic, medication-induced, structural, and psychiatric factors play a leading role and are often underestimated due to clinical polymorphism and the lack of specific symptoms at early stages [1-5, 40-44]. Of particular importance are autoimmune disorders of the central nervous system, including autoimmune encephalitis, such as anti-NMDA receptor encephalitis, which may manifest with subacute onset of cognitive impairment, psychotic symptoms, seizures, and behavioral changes [40]. Pathogenetically, these conditions are associated with the production of antibodies against neuronal antigens, leading to synaptic

dysfunction. Importantly, early immunotherapy (glucocorticoids, plasmapheresis, intravenous immunoglobulins) can result in significant or complete recovery of cognitive functions [3, 45].

Toxic influences, including chronic intoxication with alcohol, drugs, heavy metals (lead, mercury), organic solvents, and carbon monoxide, also play a substantial role [41]. Toxic brain injury is characterized by a diffuse impact on neuronal networks, manifesting as impairment of memory, attention, and executive functions. In many cases, cessation of toxin exposure and detoxification therapy are accompanied by regression of symptoms, although prolonged exposure may result in persistent deficits [45].

Medication-induced cognitive impairment is another important and often overlooked cause [42, 45]. The most significant agents include drugs with anticholinergic activity, benzodiazepines, certain antipsychotics, anticonvulsants, and opioids, which may cause confusion, memory impairment, and slowed thinking. In such cases, revision of pharmacotherapy or dose reduction often leads to improvement in cognitive status .

Structural causes include conditions associated with impaired cerebrospinal fluid dynamics or compression of brain structures, such as normal pressure hydrocephalus, which classically presents with the triad of cognitive decline, gait disturbance, and urinary incontinence [43, 46]. Surgical treatment (shunting) can significantly improve the patient’s condition. Similarly, brain tumors, subdural hematomas, or other space-occupying lesions may manifest with cognitive impairment that partially or completely regresses after removal of the underlying cause [45].

A separate category is represented by psychiatric disorders, primarily depression, which may mimic dementia (pseudodementia) [5, 44]. In such cases, complaints of memory impairment, decreased concentration, and psychomotor retardation predominate; however, with adequate antidepressant therapy, cognitive functions are largely restored. Thus, the wide spectrum of non-infectious and non-metabolic factors necessitates a comprehensive and multidisciplinary approach to the assessment of cognitive decline in young patients, with mandatory exclusion of potentially reversible causes .

The diagnostic algorithm for cognitive decline in young patients should be stepwise, etiologically oriented, and primarily aimed at identifying potentially reversible causes [3, 45, 47]. The first stage involves detailed clinical screening, including history taking with emphasis on the rate of symptom progression, the presence of comorbid somatic, psychiatric, or neurological diseases, medication and toxicological history, as well as assessment of risk factors for infectious and autoimmune processes. Family history should be уточнений to

exclude hereditary neurodegenerative diseases. In parallel, standardized assessment of cognitive function (MMSE, MoCA), psychoemotional status, and level of daily functioning is performed .

The second stage includes basic laboratory evaluation aimed at excluding metabolic, deficiency, and endocrine disorders. Mandatory tests include complete blood count, biochemical analysis, assessment of glucose levels, electrolytes, liver and kidney function parameters, thyroid-stimulating hormone, vitamin $B_{12}$, and folate levels. According to clinical indications, serological screening for HIV, syphilis, and other infections is performed. Detection of abnormalities at this stage allows identification of a significant proportion of reversible causes of cognitive decline .

The third stage involves neuroimaging, primarily magnetic resonance imaging of the brain, which enables detection of structural changes such as space-occupying lesions, demyelinating processes, vascular pathology, or signs of normal pressure hydrocephalus. In cases of suspected functional or metabolic disorders, additional methods, including positron emission tomography, may be used [45, 47].

The next stage involves cerebrospinal fluid (CSF) examination, which is indicated when infectious, autoimmune, or inflammatory processes of the central nervous system are suspected. CSF analysis allows for the detection of inflammatory markers, antibodies to neuronal antigens, and the confirmation or exclusion of conditions such as autoimmune encephalitis or neuroinfections.

The fifth stage includes extended evaluation as clinically indicated, including electroencephalography, advanced neuropsychological testing, genetic studies, and consultations with related specialists. Particular attention should be paid to the exclusion of psychiatric causes, primarily depression, which may mimic cognitive impairment [5, 44, 45].

A multidisciplinary approach is a key element of effective diagnosis and treatment, as the spectrum of causes of cognitive decline in young patients involves multiple organ systems [6, 45, 47]. The diagnostic work-up and management of the patient involve a neurologist, psychiatrist, endocrinologist, infectious disease specialist, and general physician, as well as, when necessary, a neurosurgeon and a geneticist. Such coordination not only improves diagnostic accuracy but also ensures timely initiation of etiological treatment. Coordinated interdisciplinary interaction is especially important in conditions of clinical uncertainty, when symptoms are nonspecific and may correspond to several nosological categories. Ultimately, it is the comprehensive algorithmic approach involving multiple specialists that determines the ability to identify reversible causes and improve prognosis in this patient population.

**Conclusions:**

1. In young patients, cognitive decline is significantly more likely to be potentially reversible than in older individuals, with the leading etiological factors being metabolic, deficiency-related, infectious, autoimmune, and toxic disorders, the timely correction of which may result in complete or substantial recovery of cognitive functions.

2. Clinical polymorphism and the nonspecific nature of symptoms necessitate a stepwise, algorithm-based diagnostic approach, including mandatory laboratory screening, neuroimaging, and, when indicated, cerebrospinal fluid analysis to identify potentially reversible causes.

3. A multidisciplinary approach involving various specialists is the key factor in improving prognosis, as it ensures early diagnosis, timely etiological treatment, and maximal restoration of cognitive function in this patient population.

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