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Review

Psychosis Caused by a Somatic Condition: How to Make the Diagnosis? A Systematic Literature Review

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Abstract: Background: First episode of psychosis (FEP) is a clinical condition that usually occurs during adolescence or early adulthood and is often a sign of a future psychiatric disease. However, these symptoms are not specific, and psychosis can be caused by a physical disease in at least 5% of cases. Timely detection of these diseases, the first signs of which may appear in childhood, is of particular importance, as a curable treatment exists in most cases. However, there is no consensus in academic societies to offer recommendations for a comprehensive medical assessment to eliminate somatic causes. Methods: We conducted a systematic literature search using a two-fold research strategy to: (1) identify physical diseases that can be differentially diagnosed for psychosis; and (2) determine the paraclinical exams allowing us to exclude these pathologies. Results: We identified 85 articles describing the autoimmune, metabolic, neurologic, infectious, and genetic differential diagnoses of psychosis. Clinical presentations are described, and a complete list of laboratory and imaging features required to identify and confirm these diseases is provided. Conclusion: This systematic review shows that most differential diagnoses of psychosis should be considered in the case of a FEP and could be identified by providing a systematic checkup with a laboratory test that includes ammonemia, antinuclear and anti-NMDA antibodies, and HIV testing; brain magnetic resonance imaging and lumbar puncture should be considered according to the clinical presentation. Genetic research could be of interest to patients presenting with physical or developmental symptoms associated with psychiatric manifestations.

Keywords: first-episode psychosis; differential diagnosis; initial work-up; systematic review

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1. Introduction

First episode of psychosis (FEP) is a full-threshold clinical condition in which altered perceptions of reality, delusions, disorganized thoughts, and cognitive dysfunctions can be observed by others and are associated with functional decline [1]. FEP affects 3% of the population and typically manifests during adolescence or early adulthood. Additionally, FEP may pose severe consequences, such as suicidal behaviors. Following FEP, the clinical trajectory leads to a diagnosis of schizophrenia in 51% of cases and 32.5% for another non-affective psychotic disorder [2]. However, psychotic-like symptoms have been found to also reveal a somatic affection; for example, it is well-established that auditory hallucinations can be caused by temporal epilepsy [3]. Furthermore, several studies have found that psychotic-like experiences may be associated with physical disorders in ultra-high-risk patients [4] and that at least 5% of FEP could be caused by a physical disease [5,6]. It is thus essential to identify whether these somatic diseases potentially underlie FEP patients, especially since curative treatments for them exist in most cases. Moreover, over the past decade, studies have

shown evidence that early detection of the first signs of psychosis and early intervention with a reduced duration of untreated psychosis (DUP) limit the negative impacts of the disorder [7,8].

Considering these results, guidelines focus on early intervention and treatment of FEP. However, the low specificity of FEP symptoms makes detection highly challenging, and even though guidelines propose a "general medical assessment" [1], no consensus of recommendation has been established for a comprehensive medical assessment to eliminate somatic causes. One of the main challenges in the case of FEP is to dual-diagnose co-occurring somatic diseases. Some pathologies may not be immediately clinically apparent, although a brief test can make the diagnosis. More commonly, there is no specific test, and the clinician will turn to a differential diagnosis based on unstructured clinical and paraclinical inferences. Furthermore, antipsychotics should be used with caution in some clinical conditions, such as encephalitis [9–11], indicating the sensitivity of FEP cases as well as the lethal risks that ill-informed treatment or medical mismanagement may pose. This systematic review aims to determine the physical diseases that can engender psychotic symptoms and the paraclinical exams that allow for the exclusion of these pathologies.

2. Materials and Methods

This review followed the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines [12] to ensure comprehensive and transparent reporting of methods and results. The protocol was registered at the International Prospective Register of Systematic Reviews (PROSPERO) in June 2019 (registration number: CRD42020136243).

2.1. Search Strategy

Two independent authors searched the electronic database PubMed. A two-fold research strategy was used to ensure a complete search. The research terms used were as follows: "Schizophrenia/diagnosis"[MAJR] AND "Diagnosis, Differential"[MeSH Terms] AND review" and psychosis[Title/Abstract] AND diagnosis[Title/Abstract] AND neurologic[Title/Abstract] OR endocrinologic[Title/Abstract] OR metabolic[Title/Abstract] OR infectious[Title/Abstract] OR inflammatory[Title/Abstract] OR autoimmune[Title/Abstract] OR chromosomic[Title/Abstract] OR genetic[Title/Abstract] OR toxic[Title/Abstract].

In addition, a manual literature search was performed on the websites of academic societies to identify publications related to the subject.

Papers published until July 2021 were included.

2.2. Eligibility Criteria

Articles must have included the prerequisite topics, "first-episode psychosis differential diagnosis and/or related paraclinical features", with the language of the publication written in English.

Case reports were excluded to avoid non-relevant data or duplicates with reviews already reporting matching cases. The psychotic presentation of medical conditions such as brain tumors, traumatic brain disorders, substance abuse, and common thyroid disorders has been clearly established for several decades [1,13,14]. These somatic conditions are thus not detailed in this review, as they have not been the subject of active research within the last 15 years.

2.3. Data Collection

All potential studies were first exported into a reference citation manager (Zotero), and duplicates were removed. Two independent researchers separately performed the screening of titles and abstracts, full-text analysis, and selection of paraclinical exams. Disagreements were resolved through discussions until a consensus was reached and a third reviewer was available to resolve the rest of the disagreements.

Next, a narrative-descriptive summary was created for the selected studies.

As the selected studies use heterogeneous methods, we identified commonalities from them through a two-part analysis: (1) by study features (number of studies, theme, populations, bias, etc.); and (2) by studies' contributions to clinical psychiatry and implications

for future research. Studies were classified according to the nosographic category they addressed (neurology, infectious diseases, etc.), the type of exam performed, and the type of study that was conducted (meta-analysis, literature review, etc.).

3. Results

3.1. Search Results

The electronic search returned 970 articles, and five recommendations were also identified through the hand-searched literature of publications in academic societies. A full-text evaluation was conducted for 130 articles, out of which 87 met the inclusion criteria and were included in the final synthesis (details in Appendix A). From these studies, autoimmune, metabolic, neurologic, infectious, and genetic differential diagnoses were identified for FEP, and the complete list of laboratory or imaging features required to confirm these diseases was drawn (detail in Appendix B).

Details of the search results are summarized in Figure 1.

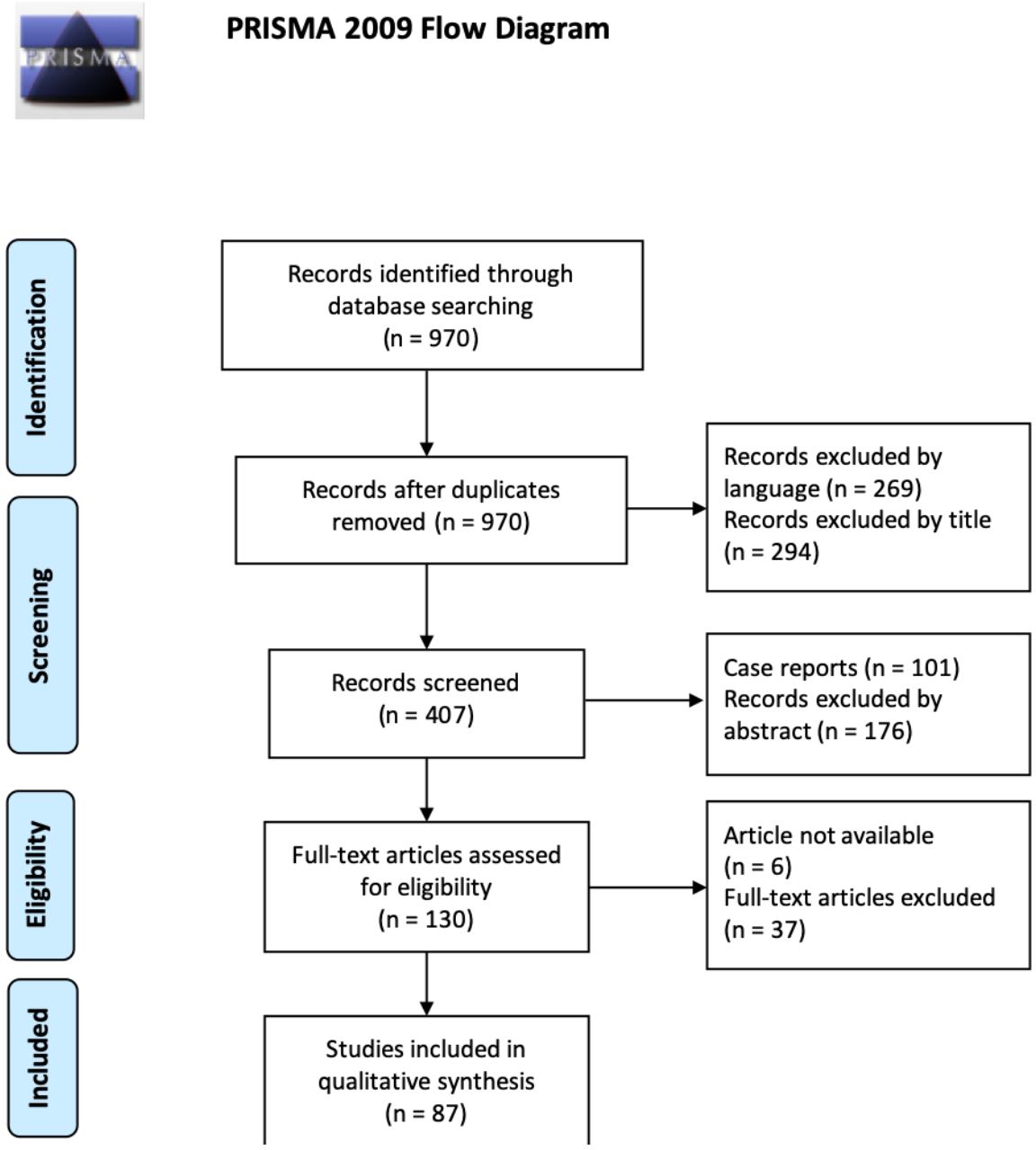


Figure 1. Prisma flow diagram for the literature search.

3.2. Autoimmune Diseases

Of the 87 articles, 38 concerned autoimmune diseases, of which systemic lupus erythematosus and limbic encephalitis were the most frequently described.

3.2.1. Limbic Encephalitis

Since the first description of limbic encephalitis in 1960, several antibodies responsible for these autoimmune brain inflammations have been discovered. The most frequent kind of limbic encephalitis is anti-N-Methyl-D-Aspartate (NMDA) receptor encephalitis, which predominantly affects children (35–40%) [9,15]. Other antibodies that are found in limbic encephalitis include anti-LG1, anti-GAD, anti-VGKC-Ab, anti-AMPA-r, and anti-GABA-r. Encephalitis with anti-NMDA-r, anti-AMPA-r, and anti-GABA-r antibodies has been strongly identified as being responsible for the manifestation of psychiatric symptoms [16–20]. It appears that psychotic symptoms are more frequent in limbic encephalitis with anti-NMDA-r antibodies in the form of drastic personality change [9], abnormal behavior (agitation, aggression, or catatonia), and delusions and hallucinations [15,21]. These symptoms can also be associated with irritability, insomnia, mood troubles [22,23], and a high level of anxiety [10,24]. Anti-NMDA receptor encephalitis is associated with cerebrospinal fluid (CSF) IgG antibodies against the GluN1 subunit of the NMDA receptor. Since antibody tests are not accessible in many institutions and it can take several weeks to obtain the results, most recent studies have aimed to improve knowledge of psychopathology and the clinical specificities of these diseases [25–30]. The clinical and paraclinical criteria have been defined to identify a probable anti-NMDA receptor encephalitis as soon as possible [31], but may not be sufficient in cases of autoimmune encephalitis without neurologic symptoms, and a systematic serum anti-NMDA-r antibodies test, electroencephalogram (EEG), and magnetic resonance imaging (MRI) of the brain are recommended [32]. For patients with psychotic symptoms presenting with clinical criteria or MRI or EEG signs, routine CSF should be mandatory [33,34].

3.2.2. Systemic Lupus Erythematosus

Systemic Lupus Erythematosus (SLE) is a prototype of a chronic, inflammatory, systemic autoimmune disease with unknown etiology, preferentially affecting females in their childbearing years [35,36], but two-thirds of pediatric patients develop neuropsychiatric symptoms [37]. Neuropsychiatric SLE (NPSLE) includes the neurologic syndromes of the central, peripheral, and autonomic nervous systems and psychiatric symptoms including depression, psychosis, and anxiety [37–39]. Delusions and hallucinations are the two main symptoms of SLE psychosis; however, they can also occur after administration of corticosteroids, and a differential diagnosis between NPSLE and cortico-induced neuropsychiatric disorders must further be considered [40]. The diagnosis of SLE is based on clinical and paraclinical criteria, but the lack of pathognomonic symptoms or test findings makes the diagnosis of NPSLE difficult to confirm. According to the last classification criteria for SLE, positive antinuclear antibodies are an obligatory criterion [41].

3.2.3. Hashimoto Encephalopathy

Two articles concerned Hashimoto encephalopathy (HE), a rare neuropsychiatric syndrome associated with Hashimoto thyroiditis, an autoimmune-mediated chronic inflammation of the thyroid. HE is more common in women and is associated with serologic evidence of antithyroid antibodies when other causes of encephalopathy are excluded [42]. In these cases, clinical manifestations include neurologic symptoms such as seizures or abnormal movements, impaired consciousness, and behavioral changes. Additionally, psychiatric symptoms including aggression, severe irritability, hallucinations, insomnia, and agitation have been described as dominant clinical features for all pediatric patients examined [43]. Brain MRI and EEG are recommended in all patients to account for the differential diagnosis of other possible encephalopathies, but the results are not specific to HE. The diagnosis is based on the identification of clinical manifestations that are associated

with increased antithyroid antibodies, and all suspected patients presenting with acute or subacute encephalopathy or cognitive decline of unknown etiology should be tested for antithyroid antibodies in serum or CSF [44]. When the antibody titer is normal at presentation with persistent symptoms of HE and evidence for another diagnosis, follow-up determinations of antithyroid antibody titers can lead to a confirmed diagnosis [43].

3.2.4. Acute Disseminated Encephalomyelitis

Acute disseminated encephalomyelitis (ADEM) is an immune-mediated inflammatory process involving central nervous system white matter that mainly occurs in children [45]. ADEM typically follows a viral infection, but it can occasionally occur without any defined preceding trigger. The classical presentation is characterized by the acute onset of neurologic abnormalities. Several cases of initial presentation with acute psychosis have been reported. In these cases, fever and focal neurologic signs were absent on presentation [46]. Early MRI facilitates the diagnosis [47] and often reveals diffuse, symmetric white matter demyelinating lesions [45,48].

3.3. Metabolic Diseases

Inborn errors of metabolism (IEMs) are physiological defects or malfunctions due to full or partial loss of gene function, usually caused by autosomal recessively inherited enzyme defects [13]. Although metabolic diseases are not frequent, the number of articles concerning these pathologies has greatly increased over the last ten years. Their impact on the central nervous system can be responsible for psychiatric syndromes such as psychosis, depression, anxiety, or mania [49,50]. Niemann-Pick disease and Wilson's disease were the two main metabolic disorders described in the literature.

3.3.1. Niemann–Pick Disease

Niemann–Pick disease type C (NP–C) is a rare (<1/100,000 births) [51], autosomal recessive neurodegenerative disease caused by mutations in the NPC1 or NPC2 gene that lead to impaired intracellular lipid trafficking and excess storage of cholesterol and glycophospholids in the brain, liver, and other tissues [52]. Patients with NP–C show extreme clinical heterogeneity in their symptom profiles, ages at disease onset, and rates of disease progression [53]. Neurological disease onset typically occurs during childhood [53], and psychiatric manifestations are frequently reported with a high incidence of psychotic symptoms, comprising paranoid delusions, auditory hallucinations, and disorganized thoughts [54,55]. Psychiatric manifestations may remain isolated for several years, and most patients who initially present with psychotic symptoms do not have obvious abnormalities on neurological examination [56,57]. Although several biochemical markers have been evaluated to provide a low-invasive and inexpensive diagnostic method [58], the definite diagnosis of NP–C disease remains based on the demonstration of specific mutations in the genes NPC1 and NPC2 [59].

3.3.2. Wilson's Disease

Wilson's disease is an autosomal recessive disease that is more common than NP–C disease (6/100,000) [57] and commonly affects children or young adults [59,60]. It is caused by a mutation of a gene [ATP7B] encoding for a copper transportation protein, leading to copper accumulation in the liver, kidney, bones, and brain. Psychiatric symptoms appear early, and it has been estimated that one in two patients present psychiatric signs in the absence of other organic signs [61]. The most frequently reported psychiatric manifestations are mood disorders, including both depressive and manic elements [62–66], hallucinations [67–69], and personality changes with the emergence of irritability and aggressive behavior [70,71]. A meta-analysis found a 2.4% frequency of "frank psychosis" resembling schizophrenia [60]. Blood copper measurement and the existence of a conventional Kayser–Fleisher ophthalmic ring are important diagnostic indicators of Wilson's disease.

The current diagnosis is made through MRI findings indicating thalamic and lenticular nucleus hyper-signals [57].

3.3.3. Disorders of Homocysteine Metabolism

Homocysteine is an essential amino acid and is metabolized along pathways of remethylation or transsulfuration. Disorders of homocysteine metabolism (DHMs) are usually caused by the absence of an enzyme in one of these pathways. Two distinct enzymes are concerned: (1) methyltetrahydrofolate reductase deficiency in the remethylation pathway and (2) cystathionine beta synthase (CbS) deficiency in the transsulfuration pathway. These deficiencies lead to a functional deficiency of folate or B12 despite normal circulating levels [57,59]. DHMs are responsible for highly variable clinical features such as skeletal abnormalities, neurologic signs, and psychiatric problems that can essentially be characterized as psychotic symptoms [57,72]. Psychotic-like symptoms can be an isolated clinical feature [73] and often produce visual hallucinations [74]. Brain imaging sometimes shows demyelination but may also present as completely normal. An MRI scan of the spinal cord may show high signal intensity in the dorsal columns of the spinal cord, similar to that seen in pernicious anemia [59]. The diagnosis is based on amino acid chromatography and the determination of homocysteine [57].

3.3.4. Acute Intermittent Porphyria

Acute intermittent porphyria (AIP) is an autosomal dominant disease with variable penetrance that is linked to the deficiency of porphobilinogen deaminase, an enzyme that is involved in the biosynthesis of heme [59]. Clinical signs of AIP usually appear in adults, but cases of childhood onset have been reported [57,75,76]. The "classical triad" of symptoms consists of abdominal pains, psychiatric disturbances, and peripheral neuropathies. Psychiatric symptoms such as delusions, hallucinations, and the development of thought disorders [57] are frequent [75,77] and can be the single presenting feature [49,78]. Acute attacks are often triggered by porphyrinogenic treatments (contraceptives, barbiturates, sulfonamides, antiepileptics), sepsis, or alcohol ingestion [76]. The diagnosis of AIP should be considered in any psychiatric syndrome with unexplained cyclical pain [79]. This diagnosis is based on the measurement of delta-aminolevulinic acid in blood testing and porphibilinogene in urinalysis [57], which may be normal between attacks [59].

3.3.5. Cerebrotendinous Xanthomasis

Cerebrotendinous xanthomatosis (CTX) is a hereditary metabolic disorder characterized biochemically by sterol 27-hydroxylase deficiency, an enzyme involved in the degradation of cholesterol. This metabolic deficiency causes the formation of xanthomatous lesions (gradual buildup of cholestanol, a cholesterol metabolite) in various tissues, including the brain and tendons [59]. The estimated prevalence of CTX is <5 in 100,000, and it varies by country and ethnic group [80,81]. Clinically, patients usually have juvenile cataracts, tendinous xanthomata, and xanthomas (lipid accumulation in the superficial dermis) as visible signs [57]. Neurological signs and psychiatric disorders (psychotic manifestations, hallucinations) are often associated with CTX [82] and appear during adolescence. Acute psychotic-like episodes have been described, even though other behavioral disorders, especially attention-deficit hyperactivity disorders, are more frequent in childhood and adolescence [82]. In CTX cases, MRI shows a typical dendritic hyper-signal in the cerebellar nuclei regions. The diagnosis is confirmed by plasma cholestanol measurements and gene sequencing [57].

3.3.6. Arylsulfatase A Deficiency

Arylsulfatase A Deficiency, also known as metachromatic leukodystrophy (MLD), is an autosomal recessive disorder in which the partial or complete absence of arylsulfatase A leads to demyelination of the central and peripheral nervous systems [83]. The clinical presentation is heterogeneous with respect to the age of onset, the rate of progression,

and the initial symptoms [84–86]. The neurological signs of MLD are often preceded by precursor symptoms in childhood, mainly behavioral disturbances and psychotic symptoms such as auditory hallucinations, bizarre delusions, and catatonic posturing [87]. The diagnosis should be suspected in individuals with progressive neurologic dysfunction, MRI evidence of leukodystrophy, or low arylsulfatase A activity in leukocytes [84]. The presence of sulfatides in urine is a useful indicator of the enzyme block [56]. The diagnosis of MLD can also be confirmed by molecular testing or after the identification of metachromatic lipid deposits in a nerve or brain biopsy [84].

3.3.7. Hereditary Transthyretin Amyloidosis

Hereditary transthyretin amyloidosis (ATTR) is a systemic disorder characterized by the extracellular deposition of misfolded transthyretin protein [88]. The clinical presentation is typically a slowly progressive sensorimotor and/or autonomic neuropathy [89] that is frequently accompanied by non-neuropathic features such as cardiomyopathy, nephropathy, vitreous opacities, and central nervous system (CNS) amyloidosis [90]. Individuals with leptomeningeal amyloidosis (LA) show CNS signs, including psychotic symptoms. The diagnosis of ATTR is established with the above clinical features, a biopsy showing amyloid deposits that bind to anti-TTR antibodies, and the identification of a heterozygous pathogenic variant in TTR by molecular testing [90]. In LA, the protein concentration in the CSF is usually high, and gadolinium-enhanced MRI typically shows an extensive increase in the surface of the brain, ventricles, and spinal cord [91].

3.3.8. Urea Cycle Disorders

Urea cycle disorders are caused by inherited enzyme deficiencies. Ornithine transcarbamylase enzyme deficiency is the most common of these diseases (1 in 8000 births) [92]. These disorders may evolve chronically during the neonatal period with mild symptoms until a first acute hyperammonemia episode appears. Patients present with symptoms related to the accumulation of ammonium in the body, including digestive, neurological, and psychotic symptoms [93]. These pathologies can easily be revealed by a measurement of plasma ammonemia.

3.3.9. Tay-Sachs Disease

Tay-Sachs disease (TSD) is an autosomal recessive lipid storage disorder caused by ß-hexosaminidase A deficiency. The prevalence of TSD is estimated to be 1 in 200,000 live births [94]. Three clinical variants of TSD, based on the age of onset, have been described in the literature [56]. Psychiatric signs may be the only manifestations of the chronic form, which usually presents in late childhood or adolescence; acute psychosis is reported in 30% to 50% of cases [95–97]. The presence of speech disturbances, gait abnormalities, movement disorders, and cognitive decline may indicate an underlying metabolic disorder. Most reports suggest that neuroleptic medications are rarely efficacious and may produce an unacceptably high risk/benefit ratio, whereas benzodiazepine may ameliorate the psychiatric and neurologic abnormalities in these patients [56,95,96].

3.4. Neurologic Diseases

3.4.1. Huntington's Disease

Huntington's disease (HD) is an incurable, autosomal dominant, neurodegenerative disease caused by an expanded CAG repeat in the huntingtin gene [98]. In HD, the production of a mutant protein leads to progressive motor impairment, psychiatric disorders, and cognitive decline [98,99]. The first manifestations begin around midlife. Neuropsychiatric symptoms, including psychosis, are highly prevalent and appear in the premotor phase [100]. According to the current criteria, the diagnosis is centered around the presence of involuntary choreiform movements and a positive genetic test for the CAG-expanded allele gene or a family history of HD [101].

3.4.2. Frontotemporal Dementia

Frontotemporal dementia (FTD) is a neurodegenerative disorder indicating a focal clinical syndrome; it is characterized by changes in one's personality and social conduct with circumscribed degeneration of the prefrontal and anterior temporal lobes [102]. Clinical and clinicopathological observations have found that people with younger-onset FTD frequently present with symptoms of schizophrenia, bipolar disorder, and affective disorders. When patients who are diagnosed with schizophrenia have an insidious and evolving cognitive deficiency, the probability of FTD is higher [103]. The characterization of FTD remains challenging, and, in the absence of definitive biomarkers, the diagnosis is based on clinical criteria including early behavioral disinhibition, apathy, perseverative behaviors, hyperorality, and executive deficits [104–106].

3.4.3. Wernicke Encephalopathy and Korsakoff's Psychosis

Wernicke encephalopathy (WE) is an acute neuropsychiatric condition caused by thiamine (vitamin B1) intracellular depletion in brain cells [107]. Alcohol misuse is the first cause of thiamine deficiency in industrialized countries but can also be seen in depleted teenage mothers in areas of poverty [108,109]. Korsakoff's psychosis results from inadequate treatment or a failure to diagnose WE in a timely manner and is characterized by severe short-term memory loss and hallucinations [110]. MRI investigations show thalamic, mamillary body, and frontal lobe atrophy, but the diagnosis remains based on clinical features and can be supported by a history of alcohol abuse [111,112].

3.4.4. Kleine–Levin Syndrome

Kleine–Levin syndrome (KLS) is a rare disease with an estimated prevalence of 1–2 per million people in France [113,114]. The triad of hypersomnia, megaphagia, and hypersexuality is classically reported in KLS cases, but the clinical presentation is rather hypersomnia with prolonged sleep times accompanied by a combination of cognitive, behavioral, and/or psychiatric disturbances. These symptoms also include depression, anxiety, and, rarely, suicidality [115]. Derealization, dissociation (feelings of mind-body disconnection), and altered perceptions are frequently described by patients with KLS [116]. The symptomatic periods alternate with intermitting periods of normalcy. Sleep monitoring and functional brain imaging during and between episodes are useful to support the diagnosis, which is based on clinical features [117,118].

3.4.5. Narcolepsy

Narcolepsy is a chronic sleep disorder that affects the regulation of sleep-wake cycles. It occurs in approximately 1 in 2000 individuals [119] and usually begins during adolescence. The classical symptoms of narcolepsy are excessive daytime sleepiness and sleep attacks, and the diagnosis is based on physiological testing, including nocturnal polysomnography and daytime EEG measurement of sleep latency. Hypnagogic/hypnopompic hallucinations are also reported, and the distinction between this presentation and the hallucinations of schizophrenia may be challenging in some clinical settings [119,120]. Most studies suggest that these psychotic symptoms are worsened by antipsychotic drugs; by contrast, they abate when narcolepsy is identified and treated with psychostimulants [119].

3.5. Infectious Diseases

A viral hypothesis for schizophrenia was seriously considered at the beginning of the 20th century [121]. More recent data show that some infectious agents, like toxoplasma gondii or CMV, could explain psychosis pathophysiology; however, a clear mechanism and a chronology are still difficult to establish. However, the human immunodeficiency virus appears to take a central role in these infectious etiologies [122], which are caused by well-known opportunistic agents [123].

3.5.1. Human Immunodeficiency Virus

Human Immunodeficiency Virus (HIV) is estimated to be associated with psychotic symptoms in 0.23% to 15% of cases [14]. These symptoms, characterized by persecutory, grandiose, and somatic delusions with hallucinations, present generally either in late-stage HIV or when patients have transitioned to acquired immunodeficiency syndrome (AIDS) [124,125]. One complicating factor in assessing this association is the fact that side effects of antiretrovirals include hallucinations [125]. HIV is diagnosed by the detection of antibodies (anti-HIV) in serum and confirmed by a Western blot test. In addition to a specific link between HIV and psychosis, some opportunistic infections related to HIV also trigger psychotic manifestations. Moreover, patients with HIV are at increased risk of developing extrapyramidal symptoms and tardive dyskinesia, particularly with the use of atypical antipsychotics [124,126]. Systematic testing (detection of anti-HIV antibodies in serum) is recommended in the case of FEP, but consent from each patient may be required, according to some state laws, before testing [127].

3.5.2. Toxoplasmosis

Toxoplasma gondii is an intracellular protozoan whose definitive host is the cat. Humans are infested by ingestion of contaminated food or water, or by congenital transmission to the fetus [128]. In immunocompetent hosts, primary infection typically remains asymptomatic. Toxoplasma is the most common infection in HIV-seropositive immunocompromised hosts, and in some cases, patients present with neurological or psychotic symptoms [129,130]. An MRI may show specific cerebral granuloma lesions, and the diagnosis is confirmed by serologic testing [129,131].

3.5.3. Cytomegalovirus

Cytomegalovirus (CMV) is a beta-herpesvirus that spreads by personal contact or congenital transmission. Most initial CMV infections are asymptomatic in immune-competent individuals, but reactivation may occur in cases of immunodeficiency (transplantation, HIV infection) and lead to psychiatric symptoms [130]. Clinical symptoms include deficits in concentration, memory, manipulation of knowledge, humor, and emotional expression [132]. A case of auditory hallucinations, delusions, and tangential thinking has also been described in the literature [133]. The diagnosis of CMV is confirmed by serologic testing.

3.5.4. Syphilis

Syphilis is a chronic, sexually transmitted disease caused by Treponema pallidum [134]. In the absence of adequate treatment, the disease follows several stages: primary, secondary, and tertiary syphilis. Neurosyphilis can occur at any stage of the disease but is classically associated with tertiary syphilis [134]. The clinical manifestations of neurosyphilis are not specific, and patients may present with psychotic symptoms mimicking psychiatric illness [14,135]. The diagnosis can be suspected based on clinical findings. The demonstration of T. pallidum in infected tissues or fluid confirms the diagnosis, but serologic testing remains the mainstay of laboratory diagnosis [135,136].

3.5.5. Lyme Borreliosis

Lyme borreliosis is a bacterial disease caused by a spirochete, Borrelia burgdoferi, which is transmitted by ticks. Its prevalence is correlated with the presence of forest areas [137]. If not treated at the initial stage, characterized by the apparition of erythema migrans, spirochetes spread in the organism and infect various organs, especially the nervous system. Neurological symptoms can occur several years after the initial infection, as spirochetes can remain quiescent for a long time [138]. The symptoms of Lyme neuroborreliosis (LNB) include encephalopathies and encephalomyelitis, but mood disturbances and psychotic symptoms are also described [139,140]. The CSF analysis is a key test for

the diagnosis of LNB, which is based on several criteria defined in the article by Mygland et al. [141].

3.6. Genetic Diseases

The relationship between some genetic disorders and psychotic-like symptoms has been noted since the 1980s [142]. Because of the multiplicity of possible gene alterations and the low prevalence of these diseases, however, systematic genetic screening is not yet conceivable. However, genetic screening should be discussed in the case of a clinical presentation evocative of a genetic disorder. In most of these diseases, patients suffer from several physical disorders and may present with an intellectual disability, dysmorphia, or resistance to antipsychotic treatments. The genetic syndromes reported in the literature are 22q11 deletion syndrome, Prader–Willi syndrome, C9orf72 mutations, Fragile X premutation disorders [143,144], BSC1L mutation [145], 3q29 recurrent deletion [146], 15q duplication syndrome [147], and genetic mitochondrial disorders [148]. The syndromes that are most frequently found in cases of FEP are described below.

3.6.1. 22q11 Deletion Syndrome

22q11 deletion syndrome is a common (1 in 4000 births) genetic disorder. Patients suffer from several physical disorders, such as congenital heart defects, palate defects, hypoparathyroidism, and immunologic abnormalities. This syndrome is also associated with psychiatric manifestations [149,150], and 23–32% of adults present with psychotic disorders [151,152]. This deletion is estimated to account for 1–2% of schizophrenia cases overall [153].

3.6.2. Prader–Willi Syndrome

Prader–Willi syndrome (PWS) is a neurodevelopmental disorder resulting from a genetic anomaly on chromosome 15, with two main subtypes identified. The birth incidence is estimated at around 1 in 22,000 [154]. Patients with PWS usually present with an association of clinical features such as hypotonia, hypogonadism, hyperphagia, mild learning disabilities, and small hands and feet. A comorbid association of PWS with psychiatric disorders has been suggested in several studies [155,156]. In both genetic subtypes, psychiatric manifestations are mainly atypical affective disorders with or without psychotic symptoms [157,158].

3.6.3. C9orf72 Mutations

C9orf72 mutations have been found in almost 12% of patients with FTD and are also associated with some forms of amyotrophic lateral sclerosis (ALS) [159]. In patients with FTD or ALS, late-onset psychotic disorders as the initial presentation have been reported to be more frequent when these diseases are secondary to the C9orf72 mutation [160–162].

4. Discussion

This systematic review reveals that many somatic illnesses, most of which may occur or begin during childhood or adolescence, can be responsible for psychotic symptoms. Determining the appropriate initial work-up implies that the prevalence of these pathologies and screening test features are to be taken into account [127].

4.1. Limitations

Studies published for the past fifteen years were selected to illustrate less-investigated, actual, and contemporary concerns about FEP. Well-known differential diagnoses (i.e., blood sugar troubles) do not require active academic research. As a consequence, these pathologies did not appear in this systematic review as they are systematically screened in current practice. As this literature review focused on clinical perspectives, the bibliographic search was restricted to medical databases. However, the probability that our pathology

list will be representative of current research concerns is increased by the inclusion of the only review that has been published to date.

4.2. Prevalence

Diseases in which psychosis is a typical presentation (i.e., Wilson's disease, thyroid diseases, etc.) should be systematically screened for FEP [127]. Many of the diseases found in this review can potentially influence psychotic symptoms even if they are not the usual presentation, especially as some of these pathologies are very rare. It would be arduous, if not infeasible, to systematically screen every disease in which psychotic symptoms have been reported; however, it seems necessary to propose an initial work-up that will help physicians who suspect a somatic origin of a given disorder.

4.3. Causality

When a somatic disease is found in a patient with FEP, the question is whether this pathology is the cause of psychosis or a comorbidity. The stress-diathesis model of schizophrenia shows this distinction is difficult to make: a somatic disorder may act as an external stress factor in individuals with pre-existing vulnerability factors [163,164]. However, models have been proposed to assess whether an association can causally contribute to a pathological phenomenon [165,166], and three key principles (atypicality, temporality, and explicability) have been suggested to help establish a cause-and-effect relationship between a somatic condition and psychotic symptoms [14]. In an atypical presentation of FEP, psychotic symptoms that appear after the onset of a medical condition and the possibility of establishing a physiological link between a somatic lesion and psychotic symptoms may provide evidence of a secondary psychotic disorder.

4.4. Exams

4.4.1. Laboratory Tests

A consensus exists on the performance of laboratory screening, but opinions differ on how broad this screening should be [1,127,167,168]. Many diseases reported in this review may produce inflammation that can be detected with a C-reactive protein test.

An ammonemia measurement, which is a low-cost exam, should be systematically performed to eliminate hyperammonemia. Positive antinuclear and anti-NMDA antibodies associated with an inflammatory syndrome may reveal an autoimmune disorder such as SLE or limbic encephalitis. A negative syphilis serology allows for ruling out neurosyphilis. This serologic test may also show an underlying autoimmune disease in the case of false-positive results [169].

A TSH test is necessary to reveal thyroid dysfunction. It may also lend support in detecting HE [170]. Suspicion of Wilson's disease, which is the most frequent metabolic disorder responsible for psychotic symptoms, can be formally excluded with a negative result of the copper measurement in the blood.

All infectious diseases outlined in this review, except syphilis and LNB, appeared in cases of immunodeficiency in HIV-seropositive hosts. As the HIV-seropositive condition can itself be associated with psychotic manifestations, systematic detection is recommended in cases of FEP after recording consent from patients [14,129,130,171].

Additionally, this review highlights the role of genetic diseases, particularly the relationship between psychotic disorders and 22q11 deletion syndrome. Clinicians should systematically seek this genetic mutation when patients present with clinical manifestations such as a developmental delay or facial dysmorphology [149,172].

The main function of an electrocardiogram (ECG) is to identify a cardiac disease, which is of particular importance before introducing an antipsychotic treatment [1]. Conduction disorders detected in young patients should also lead clinicians to suspect lupus [173].

EEG is an inexpensive, easily available, and noninvasive exam that is helpful for the diagnosis of limbic encephalitis when specific signs are detected (extreme delta brush waves have been reported in 30% of cases) [16,31,174,175].

Most of the diseases reported in this review can be revealed by magnetic resonance imaging (MRI): signs of encephalitis [43,44], leukodystrophy [56], white matter lesions [173], diffuse cortical atrophy [162], or specific lesions of toxoplasmosis [129]. Even though cerebral computed tomography (CT) is considerably more feasible, this exam uses ionizing radiation. CT is also less sensitive than MRI in the diagnosis of cerebral lesions [3]. Moreover, studies report good patient compliance, and an economic analysis suggested that MRI could be cost-saving when the prevalence of organic causes is around 1%, while the prevalence of imaging findings that would influence clinical management is estimated at around 5% in the case of FEP [3,10,168,170,176].

4.4.2. Lumbar Puncture

This review emphasizes the increasing number of limbic encephalitis cases reported and the high probability of underdiagnosis of this pathology [10,177,178]. Considering these observations, lumbar puncture would be indicated in the FEP initial work-up [179,180]. The invasive aspect of this exam is highly counterbalanced by the opportunity to diagnose a curable disease and consider the risk of a paradoxical reaction to antipsychotics in the case of encephalopathy.

5. Conclusions

This review highlights that the timely diagnosis of a physical disease is fundamental when a patient presents with psychotic symptoms in order to (1) administer the appropriate therapy and (2) avoid paradoxical reactions to antipsychotics. This differential diagnosis is possible only if the appropriate exams are performed. The initial exams recommended are presented in Table 1. Specifically, our results indicate that laboratory tests, including the antinuclear antibody test, the test of anti-NMDA antibodies, and HIV testing, must be performed systematically. The benefit-risk ratio indicates that brain MRI should be considered in cases of FEP. Finally, lumbar puncture (which is an invasive exam) could be of interest for some patients, considering the clinical presentation. Future studies should provide additional data on the feasibility and acceptability of these exams, particularly lumbar punctures.

Table 1. Initial exams recommended in the case of a first episode of psychosis.

Laboratory tests	C-reactive protein test TSH Ammonemia Ceruloplasmin FTA-ABS (fluorescent treponemal antibody absorbed) HIV test (after obtaining consent from patients) Antinuclear and anti-NMDA antibodies
Imaging	Brain MRI
Genetic tests	Karyotype and research of specific mutations, such as a 22q11 deletion, for patients presenting physical or developmental features associated with psychiatric manifestations
Others	ECG EEG Lumbar puncture in search of pleocytosis or oligoclonal bands in the CSF

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Appendix A

Author/Date	Article Title	Article Characteristics	Type of Pathology	Disease
Ford B, McDonald A, Srinivasan S. 2019 [21]	Anti-NMDA receptor encephalitis: a case study and illness overview.	Case report (40-year-old female with first episode of psychotic symptoms) and review of anti-NMDA differential diagnoses.	Autoimmune	Limbic encephalitis
Wang HY, Li T, Li XL, Zhang XX, Yan ZR, Xu Y. 2019 [11]	Anti-N-methyl-D-aspartate receptor encephalitis mimics neuroleptic malignant syndrome: case report and literature review.	Case report (24-year-old male presenting with FEP and Neuroleptic Malignant Syndrome-like symptoms) and review of diagnosis criteria for anti-NMDAr encephalitis and NMS.	Autoimmune	Limbic encephalitis
Sarkis RA, Coffey MJ, Cooper JJ, Hassan I, Lennox B. 2019 [27]	Anti-N-Methyl-D-Aspartate Receptor Encephalitis: A Review of Psychiatric Phenotypes and Management Considerations: A Report of the American Neuropsychiatric Association Committee on Research.	Review of case reports and case series (283 articles included) with patients presenting with acute or subacute (<3 months) psychiatric symptoms and anti-NMDAr antibodies. 54% of the 544 case subjects had psychotic symptoms.	Autoimmune	Limbic encephalitis
Steiner J, Prüss H, Köhler S, Frodl T, Hasan A, Falkai P. 2018 [34]	Autoimmune encephalitis with psychosis: Warning signs, step-by-step diagnostics and treatment.	Overview of clinical signs of autoimmune encephalitis in patients with FEP.	Autoimmune	Limbic encephalitis
Endres D, Leypoldt F, Bechter K, Hasan A, Steiner J, Domschke K, Wandinger KP, Falkai P, Arolt V, Stich O, Rauer S, Prüss H, van Elst LT. 2020 [33]	Autoimmune encephalitis as a differential diagnosis of schizophreniform psychosis: clinical symptomatology, pathophysiology, diagnostic approach, and therapeutic considerations.	Overview of clinical signs and diagnostic strategy in autoimmune encephalitis as differential diagnoses of FEP.	Autoimmune	Limbic encephalitis
Guasp M, Giné-Servén E, Maudes E, Rosa-Justicia M, Martínez-Hernández E, Boix-Quintana E, Bioque M, Casado V, Módena-Ouarzi Y, Guanyabens N, Muriana D, Sugranyes G, Pacchiarotti I, Davi-Loscos E, Torres-Rivas C, Ríos J, Sabater L, Saiz A, Graus F, Castro-Fornieles J, Parellada E, Dalmau J. 2021 [32]	Clinical, Neuroimmunologic, and CSF Investigations in First Episode Psychosis.	Overview of clinical signs and diagnostic strategy in autoimmune encephalitis as differential diagnoses of FEP.	Autoimmune	Limbic encephalitis
Khajezadeh MA, Zamani G, Moazzami B, Nagahi Z, Mousavi-Torshizi M, Ziaee V. 2018 [37]	Neuropsychiatric Involvement in Juvenile-Onset Systemic Lupus Erythematosus.	Cross-sectional study including patients under 18 years old with a diagnosis of SLE. 3 of the 41 patients with neuropsychiatric signs had psychotic symptoms.	Autoimmune	Systemic lupus erythematosus
Honnorat J, Plazat LO. 2018 [15]	Autoimmune encephalitis and psychiatric disorders.	Review of psychiatric symptoms of autoimmune encephalitis. 4% of isolated psychotic symptoms in anti-NMDAR encephalitis.	Autoimmune	Limbic encephalitis
Lancaster E. 2015 [177]	Paraneoplastic Disorders.	Review of the clinical specificities of paraneoplastic disorders.	Autoimmune	Limbic encephalitis
Wang D, Hao Q, He L, He L, Wang Q. 2018 [181]	LGI1 antibody encephalitis—Detailed clinical, laboratory and radiological description of 13 cases in China.	Retrospective analysis of clinical presentation for 13 patients (ages 18–63) diagnosed with LGI1 antibody encephalitis. 46% had psychotic symptoms as an initial symptom.	Autoimmune	Limbic encephalitis
Bechter K, Deisenhammer F. 2017 [180]	Psychiatric syndromes other than dementia.	Overview of differential diagnostic issues in new-onset cases of severe mental illness, focusing on CSF abnormalities.	Autoimmune	Limbic encephalitis

Author/Date	Article Title	Article Characteristics	Type of Pathology	Disease
Hermans T, Santens P, Matton C, Oostra K, Heylens G, Herremans S, Lemmens. 2018 [182]	Anti-NMDA receptor encephalitis: still unknown and underdiagnosed by physicians and especially by psychiatrists?	Description of anti-NMDAR typical presentation and diagnostic criteria with a focus on the risk of delay in diagnosis illustrated by the case of a 25-year-old woman presenting with FEP.	Autoimmune	Limbic encephalitis
Lee J, Yu HJ, Lee J. 2018 [43]	Hashimoto encephalopathy in pediatric patients: Homogeneity in clinical presentation and heterogeneity in antibody titers.	Review of clinical manifestations, antibody titers, and treatment responses in 6 children (ages 10–17) diagnosed with Hashimoto encephalopathy. 66.7% presented with psychotic symptoms.	Autoimmune	Hashimoto encephalopathy
Voice J, Ponterio JM, Lakhi N. 2017 [183]	Psychosis secondary to an incidental teratoma: a "heads-up" for psychiatrists and gynecologists.	Pathophysiology overview of anti-NMDA encephalitis secondary to ovarian teratoma. Case report of a 17-year-old woman presenting with FEP.	Autoimmune	Limbic encephalitis
Chandra SR, Viswanathan LG, Sindhu DM, Pai AR. 2017 [29]	Subacute Noninfective Inflammatory Encephalopathy: Our Experience and Diagnostic Problems.	Clinical manifestation analysis for patients with immune-mediated encephalopathy. 70% of 42 patients (ages 11–75) presented with FEP. Delay to diagnosis could reach 2 years.	Autoimmune	Limbic encephalitis
Gable M, Glaser C. 2017 [17]	Anti-N-Methyl-d-Aspartate Receptor Encephalitis Appearing as a New-Onset Psychosis: Disease Course in Children and Adolescents Within the California Encephalitis Project.	Clinical characterization and laboratory findings in 24 patients aged <18 with anti-NMDAR encephalitis. 66% presented with FEP. Psychotic symptoms can persist for more than 3 weeks before other symptoms appear.	Autoimmune	Limbic encephalitis
Mantere O, Saarela M, Kieseppä T, Raij T, Mäntylä T, Lindgren M, Rikandi E, Stoecker W, Teegen B, Suvisaari J. 2018 [184]	Anti-neuronal anti-bodies in patients with early psychosis.	Investigation of antineuronal antibodies in blood samples from 70 patients with a FEP, 6 patients with a clinical high risk for psychosis, and 34 controls. One CHR patient had anti-NMDAR antibodies. All other tests were negative.	Autoimmune	Limbic encephalitis
Xiao X, Gui S, Bai P, Bai Y, Shan D, Hu Y, Bui-Nguyen TM, Zhou R. 2017 [185]	Anti-NMDA-receptor encephalitis during pregnancy: A case report and literature review.	Review of anti-NMDAR clinical manifestations during pregnancy. Case report of a 24-year-old pregnant woman presenting with FEP.	Autoimmune	Limbic encephalitis
Hao Q, Wang D, Guo L, Zhang B. 2017 [18]	Clinical characterization of autoimmune encephalitis and psychosis.	Retrospective study of clinical presentation and laboratory examinations for 70 patients (ages 12–75) diagnosed with autoimmune encephalitis. 44% had psychiatric symptoms as initial presentation.	Autoimmune	Limbic encephalitis
Singh RK, Bhoi SK, Kalita J, Misra UK. 2017 [186]	Cerebral Venous Sinus Thrombosis Presenting Feature of Systemic Lupus Erythematosus.	Overview of SLE complications.	Autoimmune	Systemic lupus erythematosus
Bost C, Pascual O, Honnorat J. 2016 [15]	Autoimmune encephalitis in psychiatric institutions: current perspectives.	Review of psychiatric and behavioral manifestations of autoimmune encephalitis. Psychotic symptoms are the first symptoms in >40% of patients.	Autoimmune	Limbic encephalitis
Schou M, Sæther SG, Borowski K, Teegen B, Kondziella D, Stoecker W, Vaaler A, Reitan SK. 2016 [187]	Prevalence of serum anti-neuronal autoantibodies in patients admitted to acute psychiatric care.	Investigation of serum prevalence of 6 anti-neuronal antibodies in 925 patients admitted to acute psychiatric inpatient care.	Autoimmune	Limbic encephalitis
Brenton JN, Goodkin HP. 2016 [30]	Antibody-Mediated Autoimmune Encephalitis in Childhood.	Review of autoimmune encephalitis occurring in childhood: clinical presentation and laboratory findings.	Autoimmune	Limbic encephalitis
Shimizu Y, Yasuda S, Kako Y, Nakagawa S, Kanda M, Hisada R, Ohmura K, Shimamura S, Shida H, Fujieda Y, Kato M, Oku K, Bohgaki T, Horita T, Kusumi I, Atsumi T. 2016 [40]	Post-steroid neuropsychiatric manifestations are significantly more frequent in SLE compared with other systemic autoimmune diseases and predict better prognosis compared with de novo neuropsychiatric SLE.	Analysis of psychiatric manifestations in 146 patients with SLE, 43 with NPSLE, and 162 with other systemic autoimmune diseases and comparison after administration of corticosteroids.	Autoimmune	Systemic lupus erythematosus
Lancaster, E. J Clin Neurol. 2016 [16]	The Diagnosis and Treatment of Autoimmune Encephalitis.	Overview of autoimmune encephalitis clinical presentation and ancillary test findings.	Autoimmune	Limbic encephalitis

Author/Date	Article Title	Article Characteristics	Type of Pathology	Disease
Lazar-Molnar E, Tebo AE. 2015 [23]	Autoimmune NMDA receptor encephalitis.	Review of NMDAR antibody encephalitis clinical manifestations and diagnostic strategies.	Autoimmune	Limbic encephalitis
Kruse JL, Jeffrey JK, Davis MC, Dearlove J, IsHak WW, Brooks JO 3rd. 2014 [19]	Anti-N-methyl-D-aspartate receptor encephalitis: a targeted review of clinical presentation, diagnosis, and approaches to psychopharmacologic management.	2 case reports (16 and 19 year-old women with initial psychotic symptoms) and overview of the evaluation and treatment of anti-NMDAR encephalitis.	Autoimmune	Limbic encephalitis
Pollak TA, McCormack R, Peakman M, Nicholson TR, David AS. 2014 [188]	Prevalence of anti-N-methyl-D-aspartate (NMDA) receptor [corrected] antibodies in patients with schizophrenia and related psychoses: a systematic review and meta-analysis.	Systematic literature search, including 7 studies (1441 patients) to evaluate the prevalence of anti-NMDAR antibodies in patients with a primary psychiatric diagnosis without clinical signs of encephalitis.	Autoimmune	Limbic encephalitis
Armangue T, Petit-Pedrol M, Dalmau J. 2012 [24]	Autoimmune encephalitis in children.	Overview of autoimmune encephalitis clinical presentations in patients under 18 years old.	Autoimmune	Limbic encephalitis
de Holanda NC, de Lima DD, Cavalcanti TB, Lucena CS, Bandeira F. 2011 [44]	Hashimoto's encephalopathy: systematic review of the literature and an additional case.	Case report of a 23-year-old woman and systematic review of clinical features of Hashimoto encephalopathy (52 articles/130 patients).	Autoimmune	Hashimoto encephalopathy
Bertsias GK, Ioannidis JP, Aringer M, Bollen E, Bombardieri S, Bruce IN, Cervera R, Dalakas M, Doria A, Hanly JG, Huizinga TW, Isenberg D, Kallenberg C, Piette JC, Schneider M, Scolding N, Smolen J, Stara A, Tassiulas I, Tektonidou M, Tincani A, van Buchem MA, van Vollenhoven R, Ward M, Gordon C, Boumpas DT. 2010 [189]	EULAR recommendations for the management of systemic lupus erythematosus with neuropsychiatric manifestations: report of a task force of the EULAR standing committee for clinical affairs.	Recommendations.	Autoimmune	Systemic lupus erythematosus
Greenberg BM. 2009 [169]	The neurologic manifestations of systemic lupus erythematosus.	Overview of the neurologic and psychiatric clinical manifestations of SLE.	Autoimmune	Systemic lupus erythematosus
Hanly JG, Urowitz MB, Siannis F, Farewell V, Gordon C, Bae SC, Isenberg D, Dooley MA, Clarke A, Bernatsky S, Gladman D, Fortin PR, Manzi S, Steinsson K, Bruce IN, Ginzler E, Aranow C, Wallace DJ, Ramsey-Goldman R, van Vollenhoven R, Sturfelt G, Nived O, Sanchez-Guerrero J, Alarcón GS, Petri M, Khamashta M, Zoma A, Font J, Kalunian K, Douglas J, Qi Q, Thompson K, Merrill JT. 2008 [39]	Systemic Lupus International Collaborating Clinics. Autoantibodies and neuropsychiatric events at the time of systemic lupus erythematosus diagnosis: results from an international inception cohort study.	Analysis of the associations between autoantibodies and neuropsychiatric events in a cohort of 412 patients with a new diagnosis of SLE.	Autoimmune	Systemic lupus erythematosus
Steinlin MI, Blaser SI, Gilday DL, Eddy AA, Logan WJ, Laxer RM, Silverman ED. 1995 [190]	Neurologic manifestations of pediatric systemic lupus erythematosus.	Retrospective analysis of neurologic and psychiatric manifestations in 91 children (<18 years old) with a diagnosis of SLE.	Autoimmune	Systemic lupus erythematosus
Yancey CL, Doughty RA, Athreya BH. 1981 [191]	Central nervous system involvement in childhood systemic lupus erythematosus.	Retrospective analysis of neurologic and psychiatric manifestations in 37 children with a diagnosis of SLE.	Autoimmune	Systemic lupus erythematosus
Zandi MS. 2016 [20]	Autoimmune Encephalitis.	Overview of the most common clinical syndromes of autoimmune encephalitis.	Autoimmune	Limbic encephalitis
Nasr JT, Andriola MR, Coyle PK. 2000 [46]	ADEM: literature review and case report of acute psychosis presentation.	Case report of a 14-year-old girl and systematic review of ADEM presenting with acute psychosis.	Autoimmune	Acute disseminated encephalomyelitis
Weiss DB, Dyrud J, House RM, Beresford TP. 2005 [192]	Psychiatric manifestations of autoimmune disorders.	Review of psychiatric symptoms in autoimmune disorders.	Autoimmune	
O'Rourke L, Murphy KC. 2019 [150]	Recent developments in understanding the relationship between 22q11.2 deletion syndrome and psychosis.	Review including 70 papers focusing on the relationship between 22q11 deletion syndrome and psychosis.	Genetic	22q11 deletion syndrome

Author/Date	Article Title	Article Characteristics	Type of Pathology	Disease
Ducharme S, Bajestan S, Dickerson BC, Voon V. 2017 [162]	Psychiatric Presentations of C9orf72 Mutation: What Are the Diagnostic Implications for Clinicians?	Systematic review of the literature, including 43 articles related to psychiatric manifestations of the C9orf72 mutation.	Genetic	C9orf72 mutation
Tang SX, Yi JJ, Calkins ME, Whinna DA, Kohler CG, Souders MC, McDonald-McGinn DM, Zackai EH, Emanuel BS, Gur RC, Gur RE. 2014 [149]	Psychiatric disorders in 22q11.2 deletion syndrome are prevalent but undertreated.	Cross-sectional study including 112 patients (ages 8–45) with a diagnosis of 22q11DS to characterize psychiatric manifestations.	Genetic	22q11 deletion syndrome
Al-Owain M, Colak D, Albakheet A, Al-Younes B, Al-Humaidi Z, Al-Sayed M, Al-Hindi H, Al-Sugair A, Al-Muhaideb A, Rahbeeni Z, Al-Sehli A, Al-Fadhli F, Ozand PT, Taylor RW, Kaya N. 2013 [145]	Clinical and biochemical features associated with BCS1L mutation.	Clinical presentation and laboratory results for 9 patients with a BCS1L mutation.	Genetic	BSC1L mutation
Anglin RE, Garside SL, Tarnopolsky MA, Mazurek MF, Rosebush PI. 2012 [148]	The psychiatric manifestations of mitochondrial disorders: a case and review of theliterature.	Systematic review of the literature, including 50 case reports of patients with mitochondrial disorders who initially presented with psychiatric symptoms.	Genetic	Mitochondrial disorders
Bourgeois JA, Coffey SM, Rivera SM, Hessl D, Gane LW, Tassone F, Greco C, Finucane B, Nelson L, Berry-Kravis E, Grigsby J, Hagerman PJ, Hagerman RJ. 2009 [143]	A review of fragile X premutation disorders: expanding the psychiatric perspective.	Review of clinical, genetic, molecular, and neuroimaging manifestations of fragile X premutation disorders.	Genetic	Fragile X premutation disorder
Soni S, Whittington J, Holland AJ, Webb T, Maina EN, Boer H, Clarke D. 2008 [158]	The phenomenology and diagnosis of psychiatric illness in people with Prader–Willisyndrome.	Historical cohort study including 119 patients with PWS to investigate clinical psychiatric features.	Genetic	Prader–Willi syndrome
Mulle JG, Gambello MJ, Cook EH, Rutkowski TP, Glassford M. 1993 [146]	3q29 Recurrent Deletion.	Disease overview (clinical characteristics, diagnosis, management).	Genetic	3q29 deletion
Finucane BM, Lusk L, Arkilo D, Chamberlain S, Devinsky O, Dindot S, Jeste SS, LaSalle JM, Reiter LT, Schanen NC, Spence SJ, Thibert RL, Calvert G, Luchsinger K, Cook EH. 1993 [147]	15q Duplication Syndrome and Related Disorders.	Disease overview (clinical characteristics, diagnosis, management).	Genetic	15q duplication syndrome
Brodziński S, Nasierowski T. 2019 [139]	Psychosis in Borrelia burgdorferi infection -part I: epidemiology, pathogenesis, diagnosis and treatment of neuroborreliosis.	Clinical overview.	Infectious	Neuroborreliosis
Yolken RH, Torrey EF. 2008 [130]	Are some cases of psychosis caused by microbial agents? A review of the evidence.	Review of infectious agents causing psychotic symptoms.	Infectious	Toxoplasmosis Cytomegalovirus
Pradhan S, Yadav R, Mishra VN. 2007 [129]	Toxoplasma meningoencephalitis in HIV-seronegative patients: clinical patterns, imaging features and treatment outcome.	Group study with 15 patients presenting with toxoplasma meningoencephalitis.	Infectious	HIV
Buhrich N, Cooper DA. 1987 [193]	Requests for psychiatric consultation concerning 22 patients with AIDS and ARC.	Retrospective analysis of psychiatric symptoms in 22 patients with AIDS.	Infection	HIV
Faoucher M, Demily C. 2019 [50]	The psychopharmacology of Wilson disease and other metabolic disorders.	Disease overview. 30% may initially present with psychotic symptoms.	Metabolic	Wilson's disease
Sitarska D, Ługowska A. 2019 [58]	Laboratory diagnosis of the Niemann-Pick type C disease: an inherited neurodegenerative disorder of cholesterol metabolism.	Review of clinical manifestations and laboratory results.	Metabolic	Niemann–Pick disease
Rego T, Farrand S, Goh AMY, Eratne D, Kelso W, Mangelsdorf S, Velakoulis D, Walterfang M. 2019 [54]	Psychiatric and Cognitive Symptoms Associated with Niemann-Pick Type C Disease: Neurobiology and Management.	Review of clinical manifestations of NPC. Initial psychotic symptoms range from 40 to 55%.	Metabolic	Niemann–Pick disease
Sekijima Y. 2018. [90]	Hereditary Transthyretin Amyloidosis.	Disease overview (clinical characteristics, diagnosis, management).	Metabolic	Hereditary transthyretin amyloidosis

Author/Date	Article Title	Article Characteristics	Type of Pathology	Disease
Gomez-Ospina N. 2017 [84]	Arylsulfatase A Deficiency.	Disease overview (clinical characteristics, diagnosis, management).	Metabolic	Arylsulfatase A deficiency
Biswas S, Paul N, Das SK. 2017 [70]	Nonmotor Manifestations of Wilson's Disease.	Disease overview (clinical characteristics, treatment, imaging).	Metabolic	Wilson's disease
Hendriksz CJ, Anheim M, Bauer P, Bonnot O, Chakrapani A, Corvol JC, de Koning TJ, Degtyareva A, Dionisi-Vici C, Doss S, Duning T, Giunti P, Iodice R, Johnston T, Kelly D, Klünemann HH, Lorenzl S, Padovani A, Pocovi M, Synofzik M, Terblanche A, Then Bergh F, Topçu M, Tranchant C, Walterfang M, Velten C, Kolb SA. 2017 [52]	The hidden Niemann-Pick type C patient: clinical niches for a rare inherited metabolic disease.	Review of the literature. Psychotic symptoms at initial presentation are reported in 55% of cases.	Metabolic	Niemann–Pick disease
Shulman LM, David NJ, Weiner WJ. 1995 [194]	Psychosis as the initial manifestation of adult-onset Niemann-Pick disease type C.	Review of adult-onset NPC clinical presentations and case report of a 38-year-old man.	Metabolic	Niemann–Pick disease
Bonnot O, Herrera PM, Tordjman S, Walterfang M. 2015 [57]	Secondary psychosis induced by metabolic disorders.	Systematic review of psychiatric symptoms and cognitive impairments in neurometabolic diseases, including 63 publications.	Metabolic	Disorders of homocysteine metabolism Urea cycle disorders Porphyria Wilson's disease Niemann-Pick disease Cerebrotendinous xanthomatosis
Demily C, Sedel F. 2014 [59]	Psychiatric manifestations of treatable hereditary metabolic disorders in adults.	Review of psychiatric symptoms in hereditary metabolic disorders.	Metabolic	Hereditary metabolic disorders
Chawla J, Kvarnberg D. 2014 [109]	Hydrosoluble vitamins.	Clinical presentation overview of hydrosuble vitamin deficiencies.	Metabolic	Wernicke encephalopathy and Korsakoff disease
Zimbrean PC, Schilsky ML. 2014 [71]	Psychiatric aspects of Wilson disease: a review.	Systematic review of psychiatric manifestations in Wilson's disease, including 90 articles.	Metabolic	Wilson's disease
Huang CC, Chu NS. 1992 [195]	Wilson's disease: clinical analysis of 71 cases and comparison with previous Chinese series.	Comparative study of clinical presentations in 3 series of patients with Wilson's disease.	Metabolic	Wilson's disease
Walterfang M, Bonnot O, Mocellin R, Velakoulis D. 2013 [49]	The neuropsychiatry of inborn errors of metabolism.	Review of major metabolic disorders associated with secondary psychiatric illness.	Metabolic	Inborn errors of metabolism
Staretz-Chacham O, Choi JH, Wakabayashi K, Lopez G, Sidransky E. 2010 [56]	Psychiatric and behavioral manifestations of lysosomal storage disorders.	Review of psychiatric and behavioral manifestations in lysosomal storage disorders.	Metabolic	Lysosomal storage disorders
Roze E, Gervais D, Demeret S, Ogier de Baulny H, Zittoun J, Benoist JF, Said G, Pierrot-Deseilligny C, Bolgert F. 2003 [74]	Neuropsychiatric disturbances in presumed late-onset cobalamin C disease.	Case reports of a 16-year-old girl and her 24-year-old sister. Review of neurologic and psychiatric symptoms in late-onset vitamin B12 disturbances.	Metabolic	Disorders of homocysteine metabolism
Thomson AD, Marshall EJ. 2006 [107]	The natural history and pathophysiology of Wernicke's Encephalopathy and Korsakoff's Psychosis.	Review of symptoms related to Wernicke's encephalopathy and Korsakoff's psychosis.	Metabolic	Wernicke encephalopathy and Korsakoff disease
Bonnot. 2019 [55]	Systematic review of psychiatric signs in Niemann-Pick type C disease.	Systematic review of psychiatric symptoms in NPC disease, including 40 publications and data from 58 patients.	Metabolic	Niemann–Pick disease
Walterfang M, Wood SJ, Velakoulis D, Copolov D, Pantelis C. 2005 [196]	Diseases of white matter and schizophrenia-like psychosis.	Review of diseases affecting white matter tracks associated with psychotic symptoms.	Metabolic	Niemann–Pick disease Arylsulfatase A deficiency Cerebrotendinous xanthomatosis
Rosebush PI, MacQueen GM, Clarke JT, Callahan JW, Strasberg PM, Mazurek MF. 1995 [96]	Late-onset Tay-Sachs disease presenting as catatonic schizophrenia: diagnostic and treatment issues.	Case report of a 17-year-old boy and review of psychiatric manifestations and treatment of psychotic symptoms in late-onset Tay-Sachs disease.	Metabolic	Tay-Sachs disease

Author/Date	Article Title	Article Characteristics	Type of Pathology	Disease
MacQueen GM, Rosebush PI, Mazurek MF. 1998 [95]	Neuropsychiatric aspects of the adult variant of Tay-Sachs disease.	Review of neurologic and psychiatric manifestations in late-onset Tay-Sachs disease, including 64 cases.	Metabolic	Tay-Sachs disease
Toro C, Shirvan L, Tifft C. 1999 [97]	HEXA Disorders.	Disease overview (clinical characteristics, diagnosis, management).	Metabolic	Tay-Sachs disease
Albers JW, Fink JK. 2004 [197]	Porphyric neuropathy.	Review of neurologic and psychiatric symptoms in porphyria.	Metabolic	Porphyria
Qadiri MR, Church SE, McColl KE, Moore MR, Youngs GR. 1986 [198]	Chester porphyria: a clinical study of a new form of acute porphyria.	Retrospective analysis of porphyria clinical characteristics in 41 patients from the same family.	Metabolic	Porphyria
Chandrakumar A, Bhardwaj A, 't Jong GW. 2018 [112]	Review of thiamine deficiency disorders: Wernicke encephalopathy and Korsakoff psychosis.	Disease overview (epidemiology, etiopathogeneseis, neuropathology, genetics, clinical presentation, and diagnosis).	Neurologic	Wernicke encephalopathy and Korsakoff disease
Ramos ARS, Garrett C. 2017 [99]	Huntington's Disease: Premotor Phase.	Review of the evidence regarding the symptoms in the premotor state of Huntington's disease.	Neurologic	Huntington's disease
Sum-Ping O, Guilleminault C. 2016 [116]	Kleine-Levin Syndrome.	Disease overview (epidemiology, clinical presentation, etiology, evaluation, and management).	Neurologic	Kleine–Levin syndrome
Cooper JJ, Ovsiew F. 2013 [199]	The relationship between schizophrenia and frontotemporal dementia.	Review of clinical presentations of schizophrenia and frontotemporal dementia.	Neurologic	Frontotemporal dementia
Velakoulis D, Walterfang M, Mocellin R, Pantelis C, McLean C. 2009 [103]	Frontotemporaldementia presenting as schizophrenia-like psychosis in young people: clinicopathological series and review of cases.	Case series including 17 patients with FTD and a systematic review including 751 cases from 199 publications.	Neurologic	Frontotemporal dementia
Kishi Y, Konishi S, Koizumi S, Kudo Y, Kurosawa H, Kathol RG. 2004 [119]	Schizophrenia and narcolepsy: a review with a case report.	Review of psychotic manifestations in narcolepsy and case report of a 25-year-old woman.	Neurologic	Narcolepsy
Douglass AB. 2003 [120]	Narcolepsy: differential diagnosis or etiology in some cases of bipolar disorder and schizophrenia?	Review of psychiatric symptoms in narcolepsy.	Neurologic	Narcolepsy
Freudenreich O, Schulz SC, Goff DC. 2009 [127]	Initial medical work-up of first-episode psychosis: a conceptual review.	Conceptual review focusing on laboratory tests and imaging performances to exclude medical causes of psychosis.	Overview	
Giannitelli M, Consoli A, Raffin M, Jardri R, Levinson DF, Cohen D, Laurent-Levinson C. 2018 [13]	An overview of medical risk factors for childhood psychosis: Implications for research and treatment.	Systematic review of somatic disorders that can present with psychotic symptoms in children and early adolescents, including 66 publications and clinical data from a cohort of 160 patients who received schizophrenia spectrum diagnoses.	Overview	
Griswold KS, Del Regno PA, Berger RC. 2015 [200]	Recognition and Differential Diagnosis of Psychosis in Primary Care.	Overview of medical conditions that can present with acute psychosis.	Overview	
Keshavan MS, Kaneko Y. 2013 [14]	Secondary psychoses: an update.	Overview of causes of secondary psychoses and investigations to rule out these causes.	Overview	
Falkai P. 1996 [5]	Differential diagnosis in acute psychotic episode.	Review of somatic conditions presenting with acute psychosis.	Overview	
Orygen, Early Psychosis Guidelines Writing Group and EPPIC National Support Program. 2016 [1]	Australian Clinical Guidelines for Early Psychosis, 2nd edition update.	Guidelines.	Academic society recommendation (Australia)	
Haute Autorité de Santé. 2017 [201]	Guide-Affection de Longue Durée, Schizophrénies.	Guidelines.	Academic society recommendation (France)	
McGorry P, Killackey E, Elkins K, Lambert M, Lambert T. 2003 [168]	Summary Australian and New Zealand clinical practice guideline for the treatment of schizophrenia	Guidelines.	Academic society recommendation (Australia and New Zealand)	

Author/Date	Article Title	Article Characteristics	Type of Pathology	Disease
Canadian Psychiatric Association. 2005 [202]	Clinical practice guidelines. Treatment of schizophrenia.	Guidelines.	Academic society recommendation (Canada)	
Lehman AF, Lieberman JA, Dixon LB et al. 2004 [203]	Practice guideline for the treatment of patients with schizophrenia, second edition.	Guidelines.	Academic society recommendation (USA)	

Appendix B

	Pathology	Clinical Signs	Psychiatric Signs	Paraclinical Exams		
				Laboratory Tests	Imaging	Others
Autoimmune	Limbic encephalitis(NMDA)	The prodromal phase of viral appearance Memory disorders Behavioral disorders Temporal or generalized epileptic seizures Orofacial dyskinesias. No localizing signs on neurologic examination	Sudden, noisy onset Personality change Pseudo-catatonic episodes Delusional ideas of persecution Hallucinations (visual and/or auditory) Intolerance to neuroleptics Risk of neuroleptic malignant syndrome	Blood: anti-NMDA-r antibodies CSF: oligoclonal bands, specific antibodies, inflammation	MRI: T2 or FLAIR signal hyperintensity in the temporal region PET: Association of hypo- and hypermetabolisms Anteroposterior gradient with frontal and temporal hypermetabolism and occipital hypometabolism	EEG: Focal or diffuse slow or disorganized activity, epileptic activity, or extreme delta brush
	Systemic Lupus Erythematosus	Asthenia Discoid skin rash Photosensitivity Mouth ulcers Arthritis Inflammation of the serous membranes (pleural or pericardial) Renal disorders Neurologic disorders (seizures, psychotic symptoms) Blood disorder	Hallucinations Delusions	Blood: Antinuclear antibodies, anti-DNA antibodies, anti-smith antibodies	MRI: T2 small focal hyperintense lesions in the subcortical and periventricular white matter	
	Hashimoto encephalopathy	Acute or insidious symptoms Convulsions Cognitive decline Myoclonus Tremors Fluctuations in the level of consciousness	Psychotic symptoms Depression	Blood: Anti-thyroid antibodies CSF: Anti-thyroid antibodies	MRI: White matter T2 hyperintensities	EEG: Nonspecific abnormalities
	Acute disseminated encephalomyelitis	Optic neuritis Ataxia Impairment of consciousness Rapid onset fever Weakness	Irritability Psychosis	CSF: Lymphocytic pleocytosis Elevated level of myelin basic protein	MRI: Multiple, widespread asymmetric T2 hyperintense lesions	EEG: Disturbed sleep pattern Focal or general slowing
Metabolic	Niemann–Pick	Dystonia, Dysmetria, Dysarthria, Dysphagia Supranuclear vertical paralysis Convulsions Gelastic cataplexy Liver disorders Developmental retardation Learning difficulties	Paranoid episodes Auditory hallucinations Disorganized thoughts Resistance and intolerance to neuroleptics			Cutaneous biopsy: Identification of cholesterol deposits Genetics: Search for specific mutations in the NPC1 and NPC2 genes
	Wilson's	Complex cognitive disorders involving executive functions Memory disorders Neurovisual disorders Kayser-Fleisher rings	Mood disordersPersonality change Hallucinations Intolerance to neuroleptics	Blood: Copper levels Ceruloplasmin Urines: Copper levels	MRI: Hypersignals in the thalamic and lenticular nuclei Hypodensity of the basal ganglia	EEG: Aspecific defects
	Disorders of homocysteine metabolism	Episodes of thrombosis Cognitive decline Dementia Movement disorders Behavioral disorders	Hallucinations		MRI: Demyelination	Amino acids chromatography

	Pathology	Clinical Signs	Psychiatric Signs	Paraclinical Exams		
				Laboratory Tests	Imaging	Others
Metabolic	Acute intermittent porphyria	Intermittent, cyclical pain	Anxiety Impatience Suicide attempts Isolated psychotic symptoms Catatonia	Urine: Increased rates of porphobilinogen		
	Cerebrotendinous xanthomasis	Developmental disorders Chronic diarrhea Seizure Progressive spastic paraparesis Cerebellar ataxia Polyneuropathy Cognitive disorders Tendinous xanthomas	Depression Psychosis Attention deficit hyperactivity disorder	Blood: Cholestanol dosage	MRI: Dendritic hypersignal in regions of the cerebellar nuclei	
	Arysulfatase A deficiency	Educational difficulties Progressive spasticity Incontinence Seizure Dementia	Personality change Emotional lability		MRI: Leukodystrophy	Genetics: Biallelic ARSA pathogens
	Hereditary Transthyretin Amyloidosis	Progressive sensorimotor autonomic neuropathy Cardiomyopathy Renal disease Vitreous opacities Amylosis	Psychotic symptoms			
	Urea Cycle Disorders	Altered level of consciousness Seizures Learning disabilities Developmental delay Hepatomegaly, elevated liver enzymes	Hyperactivity Mood alteration Behavioral changes Psychotic symptoms	Blood: Hyperammonemia		
	Tay Sachs	Speech disorder Muscle weakness Gait disturbance	Mood disorders Psychotic symptoms	Blood: Hexosaminidase levels		
Neurologic	Huntington	Movement disorders Cognitive decline Executive disorder Involuntary choreic movements	Depression Apathy Irritability Psychotic symptoms Sleep disorders			Genetic testing
	Frontotemporal dementia	Disinhibition Apathy Hyperorality Neurologic disorders	Psychotic symptoms			Search for C9ORF72 mutation
	Wernicke and Korsakoff	Memory disorders	Hallucinations	Blood: Thiamine concentration Functional enzymatic assay of transketolase activity	MRI: Hyperintensity in the thalamic area, the mammillary bodies, and the periaqueductal area	
	Kleine–Levin	Hypersomnia Hyperphagia Hypersexuality	Feelings of derealization Depression Anxiety disorder Suicidal behaviors			
	Narcolepsy	Excessive daytime sleepiness Cataplexy Sleep attacks	Hallucinations			EEG: Nocturnal polysomnography Daytime measurement of sleep latency
Infectious	Human Immuno deficiency virus		Delusions of persecution Ideas of grandeur Hallucinations	Blood: HIV serology		
	Toxoplasmosis	Adenopathy Mononucleosis-like infectious episode Meningoencephalitis	Psychotic symptoms	Blood: IgG and IgM antibodies CSF: Low-key inflammation	MRI: Extended inflammatory lesions abscess	
	Cytomegalovirus	HIV infection Encephalitis Enteritis Pneumonia Fever Adenopathy	Auditory hallucinations Delusions Tangential thoughts Blunting of emotions	Blood: IgG and IgM antibodies		
	Syphilis	HIV co-infection	Psychotic symptoms	Direct examination by darkfield microscope immunofluorescence PCR (polymerase chain reaction) test		

	Pathology	Clinical Signs	Psychiatric Signs	Paraclinical Exams		
				Laboratory Tests	Imaging	Others
Infectious	Lyme Borreliosis	Radicular pain Paresis Confusion Cerebellar ataxia	Mood disorders Psychotic symptoms	CSF: Elevated cell count Elevated protein Oligoclonal IgG bands Blood: ELISA antibody test		
Genetic	22q11 deletion syndrome	Congenital heart defects Abnormalities of the palate Hypoparathyroidism Immunological disorders Numerous other developmental anomalies	Psychotic symptoms Mood disorders Anxiety disorders Attention deficit hyperactivity disorder			Genetics: Search for 22q11 mutation
	Prader–Willi syndrome	Hypotonia Hypogonadism Hyperphagia Learning disabilitiesSmall size Tolerance to cutaneous pain	Fluctuating symptoms Anxiety Agitation Delusions of persecution Sleep disorders Emotional involvement			Genetics: Search for 15q11-q13 mutation
	C9orf72 mutations	Cognitive and behavioral troubles	Delusions Hallucinations Concerns about health		MRI: Cortical atrophy	

References

1. *Australian Clinical Guidelines for Early Psychosis-Second Edition Updated-Orygen, Revolution in Mind*; Orygen, The National Centre of Excellence in Youth Mental Health: Melbourne, Australia, 2016.
2. Kirkbride, J.B.; Hameed, Y.; Ankireddypalli, G.; Ioannidis, K.; Crane, C.M.; Nasir, M.; Kabacs, N.; Metastasio, A.; Jenkins, O.; Espandian, A.; et al. The Epidemiology of First-Episode Psychosis in Early Intervention in Psychosis Services: Findings from the Social Epidemiology of Psychoses in East Anglia [SEPEA] Study. *AJP* **2017**, *174*, 143–153. [CrossRef] [PubMed]
3. Albon, E.; Tsourapas, A.; Frew, E.; Davenport, C.; Oyebode, F.; Bayliss, S.; Arvanitis, T.; Meads, C. *Structural Neuroimaging in Psychosis: A Systematic Review and Economic Evaluation*; NIHR Journals Library: Winchester, UK, 2008.
4. Sleurs, D.; Dubertret, C.; Pignon, B.; Tebeka, S.; Le Strat, Y. Psychotic-like Experiences Are Associated with Physical Disorders in General Population: A Cross-Sectional Study from the NESARC II. *J. Psychosom. Res.* **2023**, *165*, 111128. [CrossRef] [PubMed]
5. Falkai, P. Differential Diagnosis in Acute Psychotic Episode. *Int. Clin. Psychopharmacol.* **1996**, *11* (Suppl. S2), 13–17. [CrossRef] [PubMed]
6. Johnstone, E.C.; MacMillan, J.F.; Crow, T.J. The Occurrence of Organic Disease of Possible or Probable Aetiological Significance in a Population of 268 Cases of First Episode Schizophrenia. *Psychol. Med.* **1987**, *17*, 371–379. [CrossRef] [PubMed]
7. Penttilä, M.; Jääskeläinen, E.; Hirvonen, N.; Isohanni, M.; Miettunen, J. Duration of Untreated Psychosis as Predictor of Long-Term Outcome in Schizophrenia: Systematic Review and Meta-Analysis. *Br. J. Psychiatry* **2014**, *205*, 88–94. [CrossRef]
8. Oliver, D.; Davies, C.; Crossland, G.; Lim, S.; Gifford, G.; McGuire, P.; Fusar-Poli, P. Can We Reduce the Duration of Untreated Psychosis? A Systematic Review and Meta-Analysis of Controlled Interventional Studies. *Schizophr. Bull.* **2018**, *44*, 1362–1372. [CrossRef]
9. Bost, C.; Pascual, O.; Honnorat, J. Autoimmune Encephalitis in Psychiatric Institutions: Current Perspectives. *Neuropsychiatr. Dis. Treat.* **2016**, *12*, 2775–2787. [CrossRef]
10. Dalmau, J.; Lancaster, E.; Martinez-Hernandez, E.; Rosenfeld, M.R.; Balice-Gordon, R. Clinical Experience and Laboratory Investigations in Patients with Anti-NMDAR Encephalitis. *Lancet Neurol.* **2011**, *10*, 63–74. [CrossRef]
11. Wang, H.-Y.; Li, T.; Li, X.-L.; Zhang, X.-X.; Yan, Z.-R.; Xu, Y. Anti-N-Methyl-D-Aspartate Receptor Encephalitis Mimics Neuroleptic Malignant Syndrome: Case Report and Literature Review. *Neuropsychiatr. Dis. Treat.* **2019**, *15*, 773–778. [CrossRef]
12. Hutton, B.; Salanti, G.; Caldwell, D.M.; Chaimani, A.; Schmid, C.H.; Cameron, C.; Ioannidis, J.P.A.; Straus, S.; Thorlund, K.; Jansen, J.P.; et al. The PRISMA Extension Statement for Reporting of Systematic Reviews Incorporating Network Meta-Analyses of Health Care Interventions: Checklist and Explanations. *Ann. Intern. Med.* **2015**, *162*, 777. [CrossRef]
13. Giannitelli, M.; Consoli, A.; Raffin, M.; Jardri, R.; Levinson, D.F.; Cohen, D.; Laurent-Levinson, C. An Overview of Medical Risk Factors for Childhood Psychosis: Implications for Research and Treatment. *Schizophr. Res.* **2018**, *192*, 39–49. [CrossRef] [PubMed]
14. Keshavan, M.S.; Kaneko, Y. Secondary Psychoses: An Update. *World Psychiatry* **2013**, *12*, 4–15. [CrossRef] [PubMed]
15. Honnorat, J.; Plazat, L.O. Autoimmune Encephalitis and Psychiatric Disorders. *Rev. Neurol.* **2018**, *174*, 228–236. [CrossRef] [PubMed]
16. Lancaster, E. The Diagnosis and Treatment of Autoimmune Encephalitis. *J. Clin. Neurol.* **2016**, *12*, 1–13. [CrossRef]
17. Gable, M.; Glaser, C. Anti-N-Methyl-d-Aspartate Receptor Encephalitis Appearing as a New-Onset Psychosis: Disease Course in Children and Adolescents Within the California Encephalitis Project. *Pediatr. Neurol.* **2017**, *72*, 25–30. [CrossRef]
18. Hao, Q.; Wang, D.; Guo, L.; Zhang, B. Clinical Characterization of Autoimmune Encephalitis and Psychosis. *Compr. Psychiatry* **2017**, *74*, 9–14. [CrossRef]

19. Kruse, J.L.; Jeffrey, J.K.; Davis, M.C.; Dearlove, J.; IsHak, W.W.; Iii, J.O.B. Anti-N-Methyl-d-Aspartate Receptor Encephalitis: A Targeted Review of Clinical Presentation, Diagnosis, and Approaches to Psychopharmacologic Management. *Ann. Clin. Psychiatry* **2014**, *26*, 10.
20. Zandi, M.S. Autoimmune Encephalitis. *FOC* **2016**, *14*, 432–436. [CrossRef]
21. Ford, B.; McDonald, A.; Srinivasan, S. Anti-NMDA Receptor Encephalitis: A Case Study and Illness Overview. *Drugs Context* **2019**, *8*, 212589. [CrossRef]
22. Kayser, M.S.; Dalmau, J. Anti-NMDA Receptor Encephalitis, Autoimmunity, and Psychosis. *Schizophr. Res.* **2016**, *176*, 36–40. [CrossRef]
23. Lazar-Molnar, E.; Tebo, A.E. Autoimmune NMDA Receptor Encephalitis. *Clin. Chim. Acta* **2015**, *438*, 90–97. [CrossRef] [PubMed]
24. Armangue, T.; Petit-Pedrol, M.; Dalmau, J. Autoimmune Encephalitis in Children. *J. Child Neurol.* **2012**, *27*, 1460–1469. [CrossRef] [PubMed]
25. Al-Diwani, A.; Handel, A.; Townsend, L.; Pollak, T.; Leite, M.I.; Harrison, P.J.; Lennox, B.R.; Okai, D.; Manohar, S.G.; Irani, S.R. The Psychopathology of NMDAR-Antibody Encephalitis in Adults: A Systematic Review and Phenotypic Analysis of Individual Patient Data. *Lancet Psychiatry* **2019**, *6*, 235–246. [CrossRef] [PubMed]
26. Warren, N.; Siskind, D.; O'Gorman, C. Refining the Psychiatric Syndrome of Anti-N-Methyl-d-Aspartate Receptor Encephalitis. *Acta Psychiatr. Scand.* **2018**, *138*, 401–408. [CrossRef]
27. Sarkis, R.A.; Coffey, M.J.; Cooper, J.J.; Hassan, I.; Lennox, B. Anti-N-Methyl-D-Aspartate Receptor Encephalitis: A Review of Psychiatric Phenotypes and Management Considerations: A Report of the American Neuropsychiatric Association Committee on Research. *JNP* **2019**, *31*, 137–142. [CrossRef]
28. Pollak, T.A.; Lennox, B.R.; Müller, S.; Benros, M.E.; Prüss, H.; Tebartz van Elst, L.; Klein, H.; Steiner, J.; Frodl, T.; Bogerts, B.; et al. Autoimmune Psychosis: An International Consensus on an Approach to the Diagnosis and Management of Psychosis of Suspected Autoimmune Origin. *Lancet Psychiatry* **2020**, *7*, 93–108. [CrossRef]
29. Chandra, S.R.; Viswanathan, L.G.; Sindhu, D.M.; Pai, A.R. Subacute Noninfective Inflammatory Encephalopathy: Our Experience and Diagnostic Problems. *Indian J. Psychol. Med.* **2017**, *39*, 183–187. [CrossRef]
30. Brenton, J.N.; Goodkin, H.P. Antibody-Mediated Autoimmune Encephalitis in Childhood. *Pediatr. Neurol.* **2016**, *60*, 13–23. [CrossRef]
31. Graus, F.; Titulaer, M.J.; Balu, R.; Benseler, S.; Bien, C.G.; Cellucci, T.; Cortese, I.; Dale, R.C.; Gelfand, J.M.; Geschwind, M.; et al. A Clinical Approach to Diagnosis of Autoimmune Encephalitis. *Lancet Neurol.* **2016**, *15*, 391–404. [CrossRef]
32. Guasp, M.; Servén, E.; Maudes, E.; Rosa-Justicia, M.; Martinez-Hernandez, E.; Boix-Quintana, E.; Bioque, M.; Casado, V.; Módena-Ouarzi, Y.; Guanyabens, N.; et al. Clinical, Neuroimmunologic, and CSF Investigations in First Episode Psychosis. *Neurology* **2021**, *97*, e61–e75. [CrossRef]
33. Endres, D.; Leypoldt, F.; Bechter, K.; Hasan, A.; Steiner, J.; Domschke, K.; Wandinger, K.-P.; Falkai, P.; Arolt, V.; Stich, O.; et al. Autoimmune Encephalitis as a Differential Diagnosis of Schizophreniform Psychosis: Clinical Symptomatology, Pathophysiology, Diagnostic Approach, and Therapeutic Considerations. *Eur. Arch. Psychiatry Clin. Neurosci.* **2020**, *270*, 803–818. [CrossRef] [PubMed]
34. Steiner, J.; Prüss, H.; Köhler, S.; Frodl, T.; Hasan, A.; Falkai, P. Autoimmune Encephalitis with Psychosis: Warning Signs, Step-by-Step Diagnostics and Treatment. *World J. Biol. Psychiatry* **2018**, *21*, 241–254. [CrossRef] [PubMed]
35. Yazdany, J.; Dall'Era, M. Definition and Classification of Lupus and Lupus-Related Disorders. In *Dubois' Lupus Erythematosus and Related Syndromes*; Wallace, D.J., Hahn, B.H., Eds.; W.B. Saunders: Philadelphia, PA, USA, 2013; pp. 1–7. ISBN 978-1-4377-1893-5.
36. Lisnevskaia, L.; Murphy, G.; Isenberg, D. Systemic Lupus Erythematosus. *Lancet* **2014**, *384*, 1878–1888. [CrossRef]
37. Khajezadeh, M.-A.; Zamani, G.; Moazzami, B.; Nagahi, Z.; Mousavi-Torshizi, M.; Ziaee, V. Neuropsychiatric Involvement in Juvenile-Onset Systemic Lupus Erythematosus. 2018. Available online: https://www.hindawi.com/journals/nri/2018/2548142/ (accessed on 6 April 2020).
38. Liang, M.H.; Corzillius, M.; Bae, S.C.; Lew, R.A.; Fortin, P.R.; Gordon, C.; Isenberg, D.; Alarcón, G.S.; Straaton, K.V.; Denburg, J.; et al. The American College of Rheumatology Nomenclature and Case Definitions for Neuropsychiatric Lupus Syndromes. *Arthritis Rheum.* **1999**, *42*, 599–608. [CrossRef]
39. Hanly, J.G.; Urowitz, M.B.; Siannis, F.; Farewell, V.; Gordon, C.; Bae, S.C.; Isenberg, D.; Dooley, M.A.; Clarke, A.; Bernatsky, S.; et al. Autoantibodies and Neuropsychiatric Events at the Time of Systemic Lupus Erythematosus Diagnosis: Results from an International Inception Cohort Study. *Arthritis Rheum.* **2008**, *58*, 843–853. [CrossRef] [PubMed]
40. Shimizu, Y.; Yasuda, S.; Kako, Y.; Nakagawa, S.; Kanda, M.; Hisada, R.; Ohmura, K.; Shimamura, S.; Shida, H.; Fujieda, Y.; et al. Post-Steroid Neuropsychiatric Manifestations Are Significantly More Frequent in SLE Compared with Other Systemic Autoimmune Diseases and Predict Better Prognosis Compared with de Novo Neuropsychiatric SLE. *Autoimmun. Rev.* **2016**, *15*, 786–794. [CrossRef]
41. Garcia, L.P.W.; Gladman, D.D.; Urowitz, M.; Touma, Z.; Su, J.; Johnson, S.R. New EULAR/ACR 2019 SLE Classification Criteria: Defining Ominosity in SLE. *Ann. Rheum. Dis.* **2021**, *80*, 767. [CrossRef]
42. Chong, J.Y.; Rowland, L.P.; Utiger, R.D. Hashimoto Encephalopathy: Syndrome or Myth? *Arch. Neurol.* **2003**, *60*, 164–171. [CrossRef]
43. Lee, J.; Yu, H.J.; Lee, J. Hashimoto Encephalopathy in Pediatric Patients: Homogeneity in Clinical Presentation and Heterogeneity in Antibody Titers. *Brain Dev.* **2018**, *40*, 42–48. [CrossRef]

44. de Holanda, N.C.P.; de Lima, D.D.; Cavalcanti, T.B.; Lucena, C.S.; Bandeira, F. Hashimoto's Encephalopathy: Systematic Review of the Literature and an Additional Case. *JNP* **2011**, *23*, 384–390. [CrossRef]
45. Krupp, L.B.; Banwell, B.; Tenembaum, S. Consensus Definitions Proposed for Pediatric Multiple Sclerosis and Related Disorders. *Neurology* **2007**, *68*, S7. [CrossRef] [PubMed]
46. Nasr, J.T.; Andriola, M.R.; Coyle, P.K. Adem: Literature Review and Case Report of Acute Psychosis Presentation. *Pediatr. Neurol.* **2000**, *22*, 8–18. [CrossRef] [PubMed]
47. Tahan, A.A.; Arora, S.; Alzeer, A.; Tahan, F.A.; Malabarey, T.; Daif, A. Acute Disseminated Encephalomyelitis: The Importance of Early Magnetic Resonance Imaging. *Eur. J. Neurol.* **1997**, *4*, 52–58. [CrossRef] [PubMed]
48. Tardieu, M.; Banwell, B.; Wolinsky, J.S.; Pohl, D.; Krupp, L.B. Consensus Definitions for Pediatric MS and Other Demyelinating Disorders in Childhood. *Neurology* **2016**, *87*, S8–S11. [CrossRef]
49. Walterfang, M.; Bonnot, O.; Mocellin, R.; Velakoulis, D. The Neuropsychiatry of Inborn Errors of Metabolism. *J. Inherit. Metab. Dis.* **2013**, *36*, 687–702. [CrossRef]
50. Faoucher, M.; Demily, C. Chapter 11—The Psychopharmacology of Wilson Disease and Other Metabolic Disorders. In *Handbook of Clinical Neurology*; Reus, V.I., Lindqvist, D., Eds.; Psychopharmacology of Neurologic Disease; Elsevier: Amsterdam, The Netherlands, 2019; Volume 165, pp. 191–205.
51. Vanier, M.T. Niemann-Pick Disease Type C. *Orphanet J. Rare Dis.* **2010**, *5*, 16. [CrossRef]
52. Hendriksz, C.J.; Anheim, M.; Bauer, P.; Bonnot, O.; Chakrapani, A.; Corvol, J.-C.; de Koning, T.J.; Degtyareva, A.; Dionisi-Vici, C.; Doss, S.; et al. The Hidden Niemann-Pick Type C Patient: Clinical Niches for a Rare Inherited Metabolic Disease. *Curr. Med. Res. Opin.* **2017**, *33*, 877–890. [CrossRef]
53. Patterson, M.C.; Hendriksz, C.J.; Walterfang, M.; Sedel, F.; Vanier, M.T.; Wijburg, F. Recommendations for the Diagnosis and Management of Niemann–Pick Disease Type C: An Update. *Mol. Genet. Metab.* **2012**, *106*, 330–344. [CrossRef]
54. Rego, T.; Farrand, S.; Goh, A.M.Y.; Eratne, D.; Kelso, W.; Mangelsdorf, S.; Velakoulis, D.; Walterfang, M. Psychiatric and Cognitive Symptoms Associated with Niemann-Pick Type C Disease: Neurobiology and Management. *CNS Drugs* **2019**, *33*, 125–142. [CrossRef]
55. Bonnot, O.; Klünemann, H.-H.; Velten, C.; Martin, J.V.T.; Walterfang, M. Systematic Review of Psychiatric Signs in Niemann-Pick Disease Type C. *World J. Biol. Psychiatry* **2019**, *20*, 320–332. [CrossRef]
56. Staretz-Chacham, O.; Choi, J.H.; Wakabayashi, K.; Lopez, G.; Sidransky, E. Psychiatric and Behavioral Manifestations of Lysosomal Storage Disorders. *Am. J. Med. Genet. Part B Neuropsychiatr. Genet.* **2010**, *153B*, 1253–1265. [CrossRef] [PubMed]
57. Bonnot, O.; Herrera, P.M.; Tordjman, S.; Walterfang, M. Secondary Psychosis Induced by Metabolic Disorders. *Front. Neurosci.* **2015**, *9*, 177. [CrossRef] [PubMed]
58. Sitarska, D.; Ługowska, A. Laboratory Diagnosis of the Niemann-Pick Type C Disease: An Inherited Neurodegenerative Disorder of Cholesterol Metabolism. *Metab. Brain Dis.* **2019**, *34*, 1253–1260. [CrossRef]
59. Demily, C.; Sedel, F. Psychiatric Manifestations of Treatable Hereditary Metabolic Disorders in Adults. *Ann. Gen. Psychiatry* **2014**, *13*, 27. [CrossRef] [PubMed]
60. Taly, A.B.; Meenakshi-Sundaram, S.; Sinha, S.; Swamy, H.S.; Arunodaya, G.R. Wilson Disease: Description of 282 Patients Evaluated Over 3 Decades. *Medicine* **2007**, *86*, 112. [CrossRef]
61. Dening, T.R.; Berrios, G.E. Wilson's Disease: Psychiatric Symptoms in 195 Cases. *Arch. Gen. Psychiatry* **1989**, *46*, 1126–1134. [CrossRef]
62. Medalia, A.; Scheinberg, I.H. Psychopathology in Patients with Wilson's Disease. *Am. J. Psychiatry* **1989**, *146*, 662–664. [CrossRef]
63. Akil, M.; Schwartz, J.A.; Dutchak, D.; Yuzbasiyan-Gurkan, V.; Brewer, G.J. The Psychiatric Presentations of Wilson's Disease. *J. Neuropsychiatry Clin. Neurosci.* **1991**, *3*, 377–382. [CrossRef]
64. Dening, T.R. *The Neuropsychiatry of Wilson's Disease*; University of Newcastle upon Tyne: Newcastle upon Tyne, UK, 1989.
65. Akil, M.; Brewer, G.J. Psychiatric and Behavioral Abnormalities in Wilson's Disease. *Adv. Neurol.* **1995**, *65*, 171–178.
66. Srinivas, K.; Sinha, S.; Taly, A.B.; Prashanth, L.K.; Arunodaya, G.R.; Janardhana Reddy, Y.C.; Khanna, S. Dominant Psychiatric Manifestations in Wilson's Disease: A Diagnostic and Therapeutic Challenge! *J. Neurol. Sci.* **2008**, *266*, 104–108. [CrossRef]
67. Sagawa, M.; Takao, M.; Nogawa, S.; Mizuno, M.; Murata, M.; Amano, T.; Koto, A. Wilson's disease associated with olfactory paranoid syndrome and idiopathic thrombocytopenic purpura. *No Shinkei* **2003**, *55*, 899–902.
68. Wichowicz, H.M.; Cubała, W.J.; Sławek, J. Wilson's Disease Associated with Delusional Disorder. *Psychiatry Clin. Neurosci.* **2006**, *60*, 758–760. [CrossRef] [PubMed]
69. Spyridi, S.; Diakogiannis, I.; Michaelides, M.; Sokolaki, S.; Iacovides, A.; Kaprinis, G. Delusional Disorder and Alcohol Abuse in a Patient with Wilson's Disease. *Gen. Hosp. Psychiatry* **2008**, *30*, 585–586. [CrossRef] [PubMed]
70. Biswas, S.; Paul, N.; Das, S.K. Nonmotor Manifestations of Wilson's Disease. In *International Review of Neurobiology*; Chaudhuri, K.R., Titova, N., Eds.; Nonmotor Parkinson's: The Hidden Face; Academic Press: Cambridge, MA, USA, 2017; Volume 134, pp. 1443–1459.
71. Zimbrean, P.C.; Schilsky, M.L. Psychiatric Aspects of Wilson Disease: A Review. *Gen. Hosp. Psychiatry* **2014**, *36*, 53–62. [CrossRef]
72. Gilbody, S.; Lewis, S.; Lightfoot, T. Methylenetetrahydrofolate Reductase (MTHFR) Genetic Polymorphisms and Psychiatric Disorders: A HuGE Review. *Am. J. Epidemiol.* **2007**, *165*, 1–13. [CrossRef]
73. Li, S.C.H.; Stewart, P.M. Homocystinuria and Psychiatric Disorder: A Case Report. *Pathology* **1999**, *31*, 221–224. [CrossRef]

74. Roze, E.; Gervais, D.; Demeret, S.; de Baulny, H.O.; Zittoun, J.; Benoist, J.-F.; Said, G.; Pierrot-Deseilligny, C.; Bolgert, F. Neuropsychiatric Disturbances in Presumed Late-Onset Cobalamin C Disease. *Arch. Neurol.* **2003**, *60*, 1457–1462. [CrossRef]
75. Kühnel, A.; Gross, U.; Doss, M.O. Hereditary Coproporphyria in Germany: Clinical-Biochemical Studies in 53 Patients. *Clin. Biochem.* **2000**, *33*, 465–473. [CrossRef]
76. Crimlisk, H.L. The Little Imitator–Porphyria: A Neuropsychiatric Disorder. *J. Neurol. Neurosurg. Psychiatry* **1997**, *62*, 319–328. [CrossRef]
77. Bonkowsky, H.L.; Schady, W. Neurologic Manifestations of Acute Porphyria. *Semin. Liver Dis.* **1982**, *2*, 108–124. [CrossRef]
78. Kumar, B. Acute Intermittent Porphyria Presenting Solely with Psychosis: A Case Report and Discussion. *Psychosomatics* **2012**, *53*, 494–498. [CrossRef] [PubMed]
79. Jain, G.; Bennett, J.I.; Resch, D.S.; Godwin, J.E. Schizoaffective Disorder with Missed Diagnosis of Acute Porphyria: A Case Report and Overview. *Prim. Care Companion CNS Disord.* **2011**, *13*, 26826. [CrossRef] [PubMed]
80. Salen, G.; Steiner, R.D. Epidemiology, Diagnosis, and Treatment of Cerebrotendinous Xanthomatosis (CTX). *J. Inherit. Metab. Dis.* **2017**, *40*, 771–781. [CrossRef] [PubMed]
81. Nie, S.; Chen, G.; Cao, X.; Zhang, Y. Cerebrotendinous Xanthomatosis: A Comprehensive Review of Pathogenesis, Clinical Manifestations, Diagnosis, and Management. *Orphanet J. Rare Dis.* **2014**, *9*, 179. [CrossRef] [PubMed]
82. Sedel, F.; Baumann, N.; Turpin, J.-C.; Lyon-Caen, O.; Saudubray, J.-M.; Cohen, D. Psychiatric Manifestations Revealing Inborn Errors of Metabolism in Adolescents and Adults. *J. Inherit. Metab. Dis.* **2007**, *30*, 631–641. [CrossRef]
83. Hyde, T.M.; Ziegler, J.C.; Weinberger, D.R. Psychiatric Disturbances in Metachromatic Leukodystrophy: Insights into the Neurobiology of Psychosis. *Arch. Neurol.* **1992**, *49*, 401–406. [CrossRef]
84. Gomez-Ospina, N. Arylsulfatase A Deficiency. In *GeneReviews®*; Adam, M.P., Ardinger, H.H., Pagon, R.A., Wallace, S.E., Bean, L.J., Stephens, K., Amemiya, A., Eds.; University of Washington: Seattle, WA, USA, 1993.
85. Aicardi, J. The Inherited Leukodystrophies: A Clinical Overview. *J. Inherit. Metab. Dis.* **1993**, *16*, 733–743. [CrossRef]
86. Mahmood, A.; Berry, J.; Wenger, D.A.; Escolar, M.; Sobeih, M.; Raymond, G.; Eichler, F.S. Metachromatic Leukodystrophy: A Case of Triplets with the Late Infantile Variant and a Systematic Review of the Literature. *J. Child Neurol.* **2010**, *25*, 572–580. [CrossRef]
87. Baumann, N.; Masson, M.; Carreau, V.; Lefevre, M.; Herschkowitz, N.; Turpin, J.C. Adult Forms of Metachromatic Leukodystrophy: Clinical and Biochemical Approach. *DNE* **1991**, *13*, 211–215. [CrossRef]
88. Benson, M.D.; Uemichi, T. Transthyretin Amyloidosis. *Amyloid* **1996**, *3*, 44–56. [CrossRef]
89. Ando, Y.; Nakamura, M.; Araki, S. Transthyretin-Related Familial Amyloidotic Polyneuropathy. *Arch. Neurol.* **2005**, *62*, 1057–1062. [CrossRef] [PubMed]
90. Sekijima, Y. Hereditary Transthyretin Amyloidosis. In *GeneReviews®*; Adam, M.P., Ardinger, H.H., Pagon, R.A., Wallace, S.E., Bean, L.J., Stephens, K., Amemiya, A., Eds.; University of Washington: Seattle, WA, USA, 1993.
91. Brett, M.; Persey, M.R.; Reilly, M.M.; Revesz, T.; Booth, D.R.; Booth, S.E.; Hawkins, P.N.; Pepys, M.B.; Morgan-Hughes, J.A. Transthyretin Leu12Pro Is Associated with Systemic, Neuropathic and Leptomeningeal Amyloidosis. *Brain* **1999**, *122*, 183–190. [CrossRef] [PubMed]
92. Häberle, J.; Boddaert, N.; Burlina, A.; Chakrapani, A.; Dixon, M.; Huemer, M.; Karall, D.; Martinelli, D.; Crespo, P.S.; Santer, R.; et al. Suggested Guidelines for the Diagnosis and Management of Urea Cycle Disorders. *Orphanet J. Rare Dis.* **2012**, *7*, 32. [CrossRef]
93. Lichter-Konecki, U.; Caldovic, L.; Morizono, H.; Simpson, K. *Ornithine Transcarbamylase Deficiency*; University of Washington: Seattle, WA, USA, 2016.
94. Shapiro, B.E.; Pastores, G.M.; Gianutsos, J.; Luzy, C.; Kolodny, E.H. Miglustat in Late-Onset Tay-Sachs Disease: A 12-Month, Randomized, Controlled Clinical Study with 24 Months of Extended Treatment. *Genet. Med.* **2009**, *11*, 425–433. [CrossRef] [PubMed]
95. MacQueen, G.M.; Rosebush, P.I.; Mazurek, M.F. Neuropsychiatric Aspects of the Adult Variant of Tay-Sachs Disease. *JNP* **1998**, *10*, 10–19. [CrossRef] [PubMed]
96. Rosebush, P.I.; MacQueen, G.M.; Clarke, J.T.R.; Callahan, J.W.; Strasberg, P.M.; Mazurek, M.F. Late-Onset Tay-Sachs Disease Presenting as Catatonic Schizophrenia: Diagnostic and Treatment Issues. *J. Clin. Psychiatry* **1995**, *56*, 347–353. [PubMed]
97. Toro, C.; Shirvan, L.; Tifft, C. HEXA Disorders. In *GeneReviews®*; Adam, M.P., Ardinger, H.H., Pagon, R.A., Wallace, S.E., Bean, L.J., Stephens, K., Amemiya, A., Eds.; University of Washington: Seattle, WA, USA, 1993.
98. Ross, C.A.; Tabrizi, S.J. Huntington's Disease: From Molecular Pathogenesis to Clinical Treatment. *Lancet Neurol.* **2011**, *10*, 83–98. [CrossRef]
99. Ramos, A.R.S.; Garrett, C. Huntington's Disease: Premotor Phase. *NDD* **2017**, *17*, 313–322. [CrossRef]
100. Martinez-Horta, S.; Perez-Perez, J.; van Duijn, E.; Fernandez-Bobadilla, R.; Carceller, M.; Pagonabarraga, J.; Pascual-Sedano, B.; Campolongo, A.; Ruiz-Idiago, J.; Sampedro, F.; et al. Neuropsychiatric Symptoms Are Very Common in Premanifest and Early Stage Huntington's Disease. *Park. Relat. Disord.* **2016**, *25*, 58–64. [CrossRef]
101. Hogarth, P.; Kayson, E.; Kieburtz, K.; Marder, K.; Oakes, D.; Rosas, D.; Shoulson, I.; Wexler, N.S.; Young, A.B.; Zhao, H. Interrater Agreement in the Assessment of Motor Manifestations of Huntington's Disease. *Mov. Disord.* **2005**, *20*, 293–297. [CrossRef]
102. Neary, D.; Snowden, J.; Mann, D. Frontotemporal Dementia. *Lancet Neurol.* **2005**, *4*, 771–780. [CrossRef] [PubMed]

103. Velakoulis, D.; Walterfang, M.; Mocellin, R.; Pantelis, C.; McLean, C. Frontotemporal Dementia Presenting as Schizophrenia-like Psychosis in Young People: Clinicopathological Series and Review of Cases. *Br. J. Psychiatry* **2009**, *194*, 298–305. [CrossRef] [PubMed]
104. Neary, D.; Snowden, J.S.; Gustafson, L.; Passant, U.; Stuss, D.; Black, S.; Freedman, M.; Kertesz, A.; Robert, P.H.; Albert, M.; et al. Frontotemporal Lobar Degeneration. *Neurology* **1998**, *51*, 1546. [CrossRef] [PubMed]
105. McKhann, G.M.; Albert, M.S.; Grossman, M.; Miller, B.; Dickson, D.; Trojanowski, J.Q. Clinical and Pathological Diagnosis of Frontotemporal Dementia: Report of the Work Group on Frontotemporal Dementia and Pick's Disease. *Arch. Neurol.* **2001**, *58*, 1803–1809. [CrossRef]
106. Rascovsky, K.; Hodges, J.R.; Knopman, D.; Mendez, M.F.; Kramer, J.H.; Neuhaus, J.; van Swieten, J.C.; Seelaar, H.; Dopper, E.G.P.; Onyike, C.U.; et al. Sensitivity of Revised Diagnostic Criteria for the Behavioural Variant of Frontotemporal Dementia. *Brain* **2011**, *134*, 2456–2477. [CrossRef] [PubMed]
107. Thomson, A.D.; Marshall, E.J. The Natural History and Pathophysiology of Wernicke's Encephalopathy and Korsakoff's Psychosis. *Alcohol Alcohol.* **2006**, *41*, 151–158. [CrossRef]
108. Thomson, A.D. Mechanisms of Vitamin Deficiency in Chronic Alcohol Misusers and the Development of the Wernicke-Korsakoff Syndrome. *Alcohol Alcohol.* **2000**, *35*, 1–2. [CrossRef]
109. Chawla, J.; Kvarnberg, D. Chapter 59—Hydrosoluble Vitamins. In *Handbook of Clinical Neurology*; Biller, J., Ferro, J.M., Eds.; Neurologic Aspects of Systemic Disease Part II; Elsevier: Amsterdam, The Netherlands, 2014; Volume 120, pp. 891–914.
110. Dhir, S.; Tarasenko, M.; Napoli, E.; Giulivi, C. Neurological, Psychiatric, and Biochemical Aspects of Thiamine Deficiency in Children and Adults. *Front. Psychiatry* **2019**, *10*, 207. [CrossRef]
111. Kopelman, M.D.; Thomson, A.D.; Guerrini, I.; Marshall, E.J. The Korsakoff Syndrome: Clinical Aspects, Psychology and Treatment. *Alcohol Alcohol.* **2009**, *44*, 148–154. [CrossRef]
112. Chandrakumar, A.; Bhardwaj, A.; Jong, G.W. Review of Thiamine Deficiency Disorders: Wernicke Encephalopathy and Korsakoff Psychosis. *J. Basic Clin. Physiol. Pharmacol.* **2018**, *30*, 153–162. [CrossRef]
113. Lavault, S.; Golmard, J.-L.; Groos, E.; Brion, A.; Dauvilliers, Y.; Lecendreux, M.; Franco, P.; Arnulf, I. Kleine–Levin Syndrome in 120 Patients: Differential Diagnosis and Long Episodes. *Ann. Neurol.* **2015**, *77*, 529–540. [CrossRef] [PubMed]
114. AlShareef, S.M.; Smith, R.M.; BaHammam, A.S. Kleine-Levin Syndrome: Clues to Aetiology. *Sleep Breath* **2018**, *22*, 613–623. [CrossRef] [PubMed]
115. Arnulf, I.; Rico, T.J.; Mignot, E. Diagnosis, Disease Course, and Management of Patients with Kleine-Levin Syndrome. *Lancet Neurol.* **2012**, *11*, 918–928. [CrossRef] [PubMed]
116. Sum-Ping, O.; Guilleminault, C. Kleine-Levin Syndrome. *Curr. Treat. Options Neurol.* **2016**, *18*, 24. [CrossRef]
117. Arnulf, I. Kleine-Levin Syndrome. *Sleep Med. Clin.* **2015**, *10*, 151–161. [CrossRef]
118. Sateia, M.J. International Classification of Sleep Disorders-Third Edition. *Chest* **2014**, *146*, 1387–1394. [CrossRef]
119. Kishi, Y.; Konishi, S.; Koizumi, S.; Kudo, Y.; Kurosawa, H.; Kathol, R.G. Schizophrenia and Narcolepsy: A Review with a Case Report. *Psychiatry Clin. Neurosci.* **2004**, *58*, 117–124. [CrossRef]
120. Douglass, A.B. Narcolepsy: Differential Diagnosis or Etiology in Some Cases of Bipolar Disorder and Schizophrenia? *CNS Spectr.* **2003**, *8*, 120–126. [CrossRef]
121. Guelfi, J.D.; Rouillon, F. *Manuel de Psychiatrie*; Elsevier Masson: Issy Les Moulineaux, France, 2017; ISBN 978-2-294-74976-6.
122. Ey, H.; Bernard, P. *Manuel de Psychiatrie (6ème Edition)*; Elsevier Masson: Issy Les Moulineaux, France, 2010.
123. Alciati, A.; Fusi, A.; D'Arminio Monforte, A.; Coen, M.; Ferri, A.; Mellado, C. New-Onset Delusions and Hallucinations in Patients Infected with HIV. *J. Psychiatry Neurosci.* **2001**, *26*, 229–234.
124. Sewell, D.D. Schizophrenia and HIV. *Schizophr. Bull.* **1996**, *22*, 465–473. [CrossRef]
125. Dolder, C.R.; Patterson, T.L.; Jeste, D.V. HIV, Psychosis and Aging: Past, Present and Future. *AIDS* **2004**, *18*, 35. [CrossRef]
126. Shedlack, K.J.; Soldato-Couture, C.; Swanson, C.L. Rapidly Progressive Tardive Dyskinesia in AIDS. *Biol. Psychiatry* **1994**, *35*, 147–148. [CrossRef] [PubMed]
127. Freudenreich, O.; Schulz, S.C.; Goff, D.C. Initial Medical Work-up of First-Episode Psychosis: A Conceptual Review. *Early Interv. Psychiatry* **2009**, *3*, 10–18. [CrossRef]
128. Montoya, J.G.; Rosso, F. Diagnosis and Management of Toxoplasmosis. *Clin. Perinatol.* **2005**, *32*, 705–726. [CrossRef]
129. Pradhan, S.; Yadav, R.; Mishra, V.N. Toxoplasma Meningoencephalitis in HIV-Seronegative Patients: Clinical Patterns, Imaging Features and Treatment Outcome. *Trans. R. Soc. Trop. Med. Hyg.* **2007**, *101*, 25–33. [CrossRef] [PubMed]
130. Yolken, R.H.; Torrey, E.F. Are Some Cases of Psychosis Caused by Microbial Agents? *A Review of the Evidence. Mol. Psychiatry* **2008**, *13*, 470–479. [CrossRef]
131. Villard, O.; Cimon, B.; L'Ollivier, C.; Fricker-Hidalgo, H.; Godineau, N.; Houze, S.; Paris, L.; Pelloux, H.; Villena, I.; Candolfi, E. Serological Diagnosis of Toxoplasma Gondii Infection: Recommendations from the French National Reference Center for Toxoplasmosis. *Diagn. Microbiol. Infect. Dis.* **2016**, *84*, 22–33. [CrossRef]
132. Duchowny, M.; Caplan, L.; Siber, G. Cytomegalovirus Infection of the Adult Nervous System. *Ann. Neurol.* **1979**, *5*, 458–461. [CrossRef]
133. Torrey, E.F. Functional Psychoses and Viral Encephalitis. *Integr. Psychiatry* **1986**, *4*, 224–230.
134. LaFond, R.E.; Lukehart, S.A. Biological Basis for Syphilis. *Clin. Microbiol. Rev.* **2006**, *19*, 29–49. [CrossRef]

135. Singh, A.E.; Romanowski, B. Syphilis: Review with Emphasis on Clinical, Epidemiologic, and Some Biologic Features. *Clin. Microbiol. Rev.* **1999**, *12*, 187–209. [CrossRef] [PubMed]
136. Morshed, M.G. Current Trend on Syphilis Diagnosis: Issues and Challenges. In *Proceedings of the Infectious Diseases and Nanomedicine II*; Adhikari, R., Thapa, S., Eds.; Springer: New Delhi, India, 2014; pp. 51–64.
137. Rizzoli, A.; Hauffe, H.C.; Carpi, G.; Vourc'h, G.I.; Neteler, M.; Rosà, R. Lyme Borreliosis in Europe. *Eurosurveillance* **2011**, *16*, 19906. [CrossRef]
138. Fallon, B.A.; Nields, J.A. Lyme Disease: A Neuropsychiatric Illness. *Am. J. Psychiatry* **1994**, *151*, 13.
139. Brodziński, S.; Nasierowski, T. Psychosis in Borrelia Burgdorferi Infection—Part I: Epidemiology, Pathogenesis, Diagnosis and Treatment of Neuroborreliosis. *Psychiatr. Pol.* **2019**, *53*, 629–640. [CrossRef] [PubMed]
140. Fallon, B.A.; Levin, E.S.; Schweitzer, P.J.; Hardesty, D. Inflammation and Central Nervous System Lyme Disease. *Neurobiol. Dis.* **2010**, *37*, 534–541. [CrossRef] [PubMed]
141. Mygland, Å.; Ljøstad, U.; Fingerle, V.; Rupprecht, T.; Schmutzhard, E.; Steiner, I. EFNS Guidelines on the Diagnosis and Management of European Lyme Neuroborreliosis. *Eur. J. Neurol.* **2010**, *17*, 8-e4. [CrossRef]
142. Propping, P. Genetic Disorders Presenting as "Schizophrenia". Karl Bonhoeffer's Early View of the Psychoses in the Light of Medical Genetics. *Hum. Genet.* **1983**, *65*, 1–10. [CrossRef] [PubMed]
143. Bourgeois, J.; Coffey, S.; Rivera, S.M.; Hessl, D.; Gane, L.W.; Tassone, F.; Greco, C.; Finucane, B.; Nelson, L.; Berry-Kravis, E.; et al. Fragile X Premutation Disorders—Expanding the Psychiatric Perspective. *J. Clin. Psychiatry* **2009**, *70*, 852–862. [CrossRef]
144. Franke, P.; Leboyer, M.; Gänsicke, M.; Weiffenbach, O.; Biancalana, V.; Cornillet-Lefebre, P.; Françoise Croquette, M.; Froster, U.G.; Schwab, S.; Poustka, F.; et al. Genotype–Phenotype Relationship in Female Carriers of the Premutation and Full Mutation of FMR-1. *Psychiatry Res.* **1998**, *80*, 113–127. [CrossRef]
145. Al-Owain, M.; Colak, D.; Albakheet, A.; Al-Younes, B.; Al-Humaidi, Z.; Al-Sayed, M.; Al-Hindi, H.; Al-Sugair, A.; Al-Muhaideb, A.; Rahbeeni, Z.; et al. Clinical and Biochemical Features Associated with BCS1L Mutation. *J. Inherit Metab. Dis.* **2013**, *36*, 813–820. [CrossRef]
146. Mulle, J.G.; Gambello, M.J.; Cook, E.H.; Rutkowski, T.P.; Glassford, M. 3q29 Recurrent Deletion. In *GeneReviews®*; Adam, M.P., Ardinger, H.H., Pagon, R.A., Wallace, S.E., Bean, L.J., Stephens, K., Amemiya, A., Eds.; University of Washington: Seattle, WA, USA, 1993.
147. Finucane, B.M.; Lusk, L.; Arkilo, D.; Chamberlain, S.; Devinsky, O.; Dindot, S.; Jeste, S.S.; LaSalle, J.M.; Reiter, L.T.; Schanen, N.C.; et al. 15q Duplication Syndrome and Related Disorders. In *GeneReviews®*; Adam, M.P., Ardinger, H.H., Pagon, R.A., Wallace, S.E., Bean, L.J., Stephens, K., Amemiya, A., Eds.; University of Washington: Seattle, WA, USA, 1993.
148. Anglin, R.; Garside, S.; Tarnopolsky, M.; Mazurek, M.; Rosebush, P. The Psychiatric Manifestations of Mitochondrial Disorders: A Case and Review of the Literature. *J. Clin. Psychiatry* **2012**, *73*, 506–512. [CrossRef] [PubMed]
149. Tang, S.X.; Yi, J.J.; Calkins, M.E.; Whinna, D.A.; Kohler, C.G.; Souders, M.C.; McDonald-McGinn, D.M.; Zackai, E.H.; Emanuel, B.S.; Gur, R.C.; et al. Psychiatric Disorders in 22q11.2 Deletion Syndrome Are Prevalent but Undertreated. *Psychol. Med.* **2014**, *44*, 1267–1277. [CrossRef] [PubMed]
150. O'Rourke, L.; Murphy, K.C. Recent Developments in Understanding the Relationship between 22q11.2 Deletion Syndrome and Psychosis. *Curr. Opin. Psychiatry* **2019**, *32*, 67–72. [CrossRef] [PubMed]
151. Green, T.; Gothelf, D.; Glaser, B.; Debbane, M.; Frisch, A.; Kotler, M.; Weizman, A.; Eliez, S. Psychiatric Disorders and Intellectual Functioning Throughout Development in Velocardiofacial (22q11.2 Deletion) Syndrome. *J. Am. Acad. Child Adolesc. Psychiatry* **2009**, *48*, 1060–1068. [CrossRef] [PubMed]
152. Bassett, A.S.; Chow, E.W.C.; AbdelMalik, P.; Gheorghiu, M.; Husted, J.; Weksberg, R. The Schizophrenia Phenotype in 22q11 Deletion Syndrome. *AJP* **2003**, *160*, 1580–1586. [CrossRef] [PubMed]
153. Schreiner, M.J.; Lazaro, M.T.; Jalbrzikowski, M.; Bearden, C.E. Converging Levels of Analysis on a Genomic Hotspot for Psychosis: Insights from 22q11.2 Deletion Syndrome. *Neuropharmacology* **2013**, *68*, 157–173. [CrossRef]
154. Whittington, J.; Holland, T. *Prader-Willi Syndrome: Development and Manifestations*; Cambridge University Press: Cambridge, UK, 2004; ISBN 978-1-139-45245-8.
155. Clarke, D.J. Prader-Willi Syndrome and Psychoses. *Br. J. Psychiatry* **1993**, *163*, 680–684. [CrossRef]
156. Bouras, N.; Verhoeven, W.M.A.; Curfs, L.M.G.; Tuinier, S. Prader–Willi Syndrome and Cycloid Psychoses. *J. Intellect. Disabil. Res.* **1998**, *42*, 455–462. [CrossRef]
157. Bouras, N.; Beardsmore, A.; Dorman, T.; Cooper, S.-A.; Webb, T. Affective Psychosis and Prader–Willi Syndrome. *J. Intellect. Disabil. Res.* **1998**, *42*, 463–471. [CrossRef]
158. Soni, S.; Whittington, J.; Holland, A.J.; Webb, T.; Maina, E.N.; Boer, H.; Clarke, D. The Phenomenology and Diagnosis of Psychiatric Illness in People with Prader–Willi Syndrome. *Psychol. Med.* **2008**, *38*, 1505–1514. [CrossRef]
159. Rohrer, J.D.; Isaacs, A.M.; Mizielinska, S.; Mead, S.; Lashley, T.; Wray, S.; Sidle, K.; Fratta, P.; Orrell, R.W.; Hardy, J.; et al. C9orf72 Expansions in Frontotemporal Dementia and Amyotrophic Lateral Sclerosis. *Lancet Neurol.* **2015**, *14*, 291–301. [CrossRef] [PubMed]
160. Snowden, J.S.; Rollinson, S.; Thompson, J.C.; Harris, J.M.; Stopford, C.L.; Richardson, A.M.T.; Jones, M.; Gerhard, A.; Davidson, Y.S.; Robinson, A.; et al. Distinct Clinical and Pathological Characteristics of Frontotemporal Dementia Associated with C9ORF72 Mutations. *Brain* **2012**, *135*, 693–708. [CrossRef]

161. Dobson-Stone, C.; Hallupp, M.; Bartley, L.; Shepherd, C.E.; Halliday, G.M.; Schofield, P.R.; Hodges, J.R.; Kwok, J.B.J. C9ORF72 Repeat Expansion in Clinical and Neuropathologic Frontotemporal Dementia Cohorts. *Neurology* **2012**, *79*, 995. [CrossRef] [PubMed]
162. Ducharme, S.; Bajestan, S.; Dickerson, B.C.; Voon, V. Psychiatric Presentations of C9orf72 Mutation: What Are the Diagnostic Implications for Clinicians? *JNP* **2017**, *29*, 195–205. [CrossRef] [PubMed]
163. Pruessner, M.; Cullen, A.E.; Aas, M.; Walker, E.F. The Neural Diathesis-Stress Model of Schizophrenia Revisited: An Update on Recent Findings Considering Illness Stage and Neurobiological and Methodological Complexities. *Neurosci. Biobehav. Rev.* **2017**, *73*, 191–218. [CrossRef] [PubMed]
164. Howes, O.D.; McCutcheon, R. Inflammation and the Neural Diathesis-Stress Hypothesis of Schizophrenia: A Reconceptualization. *Transl. Psychiatry* **2017**, *7*, e1024. [CrossRef]
165. Howick, J.; Glasziou, P.; Aronson, J.K. The Evolution of Evidence Hierarchies: What Can Bradford Hill's 'Guidelines for Causation' Contribute? *J. R. Soc. Med.* **2009**, *102*, 186–194. [CrossRef]
166. Manu, P.; Correll, C.U.; Wampers, M.; Mitchell, A.J.; Probst, M.; Vancampfort, D.; Hert, M.D. Markers of Inflammation in Schizophrenia: Association vs. *Causation. World Psychiatry* **2014**, *13*, 189–192. [CrossRef]
167. Congrès de Psychiatrie et de Neurologie de Langue Française; Krebs, M.-O. *Signes Précoces de Schizophrénie des Prodromes à la Notion de Prévention*; Dunod: Paris, France, 2015; ISBN 978-2-10-073964-6.
168. McGorry, P.; Killackey, E.; Elkins, K.; Lambert, M.; Lambert, T. RANZCP Clinical Practice Guideline Team for the Treatment of Schizophrenia. Summary Australian and New Zealand Clinical Practice Guideline for the Treatment of Schizophrenia 2003. *Australas. Psychiatry* **2003**, *11*, 136–147. [CrossRef]
169. Greenberg, B.M. The Neurologic Manifestations of Systemic Lupus Erythematosus. *Neurologist* **2009**, *15*, 115. [CrossRef]
170. Debbabi, W.; Essid, A.; Chiboub, M.; Kharrat, I.; Samet, S. Encéphalopathie corticosensible associée à une thyroïdite auto-immune. *Ann. D'endocrinologie* **2017**, *78*, 346. [CrossRef]
171. Branson, B.M.; Handsfield, H.H.; Lampe, M.A.; Janssen, R.S.; Taylor, A.W.; Lyss, S.B.; Clark, J.E. Revised Recommendations for HIV Testing of Adults, Adolescents, and Pregnant Women in Health-Care Settings. *Morb. Mortal. Wkly. Rep. Recomm. Rep.* **2006**, *55*, 1–17.
172. Horowitz, A.; Shifman, S.; Rivlin, N.; Pisanté, A.; Darvasi, A. A Survey of the 22q11 Microdeletion in a Large Cohort of Schizophrenia Patients. *Schizophr. Res.* **2005**, *73*, 263–267. [CrossRef]
173. Bertsias, G.; Ioannidis, J.P.A.; Boletis, J.; Bombardieri, S.; Cervera, R.; Dostal, C.; Font, J.; Gilboe, I.M.; Houssiau, F.; Huizinga, T.; et al. EULAR Recommendations for the Management of Systemic Lupus Erythematosus. Report of a Task Force of the EULAR Standing Committee for International Clinical Studies Including Therapeutics. *Ann. Rheum. Dis.* **2008**, *67*, 195–205. [CrossRef]
174. González-Valcárcel, J.; Rosenfeld, M.R.; Dalmau, J. Differential Diagnosis of Encephalitis Due to Anti-NMDA Receptor Antibodies. *Neurología* **2010**, *25*, 409–413. [CrossRef] [PubMed]
175. León-Caballero, J.; Pacchiarotti, I.; Murru, A.; Valentí, M.; Colom, F.; Benach, B.; Pérez, V.; Dalmau, J.; Vieta, E. Bipolar Disorder and Antibodies against the N-Methyl-d-Aspartate Receptor: A Gate to the Involvement of Autoimmunity in the Pathophysiology of Bipolar Illness. *Neurosci. Biobehav. Rev.* **2015**, *55*, 403–412. [CrossRef]
176. Falkenberg, I.; Benetti, S.; Raffin, M.; Wuyts, P.; Pettersson-Yeo, W.; Dazzan, P.; Morgan, K.D.; Murray, R.M.; Marques, T.R.; David, A.S.; et al. Clinical Utility of Magnetic Resonance Imaging in First-Episode Psychosis. *Br. J. Psychiatry* **2017**, *211*, 231–237. [CrossRef]
177. Lancaster, E. Continuum: The Paraneoplastic Disorders. *Contin. Minneap. Minn.* **2015**, *21*, 452–475. [CrossRef]
178. Fénelon, G. Les hallucinations en neurologie. *Prat. Neurol. FMC* **2014**, *5*, 277–286. [CrossRef]
179. Gresa-Arribas, N.; Titulaer, M.J.; Torrents, A.; Aguilar, E.; McCracken, L.; Leypoldt, F.; Gleichman, A.J.; Balice-Gordon, R.; Rosenfeld, M.R.; Lynch, D.; et al. Antibody Titres at Diagnosis and during Follow-up of Anti-NMDA Receptor Encephalitis: A Retrospective Study. *Lancet Neurol.* **2014**, *13*, 167–177. [CrossRef]
180. Bechter, K.; Deisenhammer, F. Chapter 17—Psychiatric Syndromes Other than Dementia. In *Handbook of Clinical Neurology*; Deisenhammer, F., Teunissen, C.E., Tumani, H., Eds.; Cerebrospinal Fluid in Neurologic Disorders; Elsevier: Amsterdam, The Netherlands, 2017; Volume 146, pp. 285–296.
181. Wang, D.; Hao, Q.; He, L.; He, L.; Wang, Q. LGI1 Antibody Encephalitis—Detailed Clinical, Laboratory and Radiological Description of 13 Cases in China. *Compr. Psychiatry* **2018**, *81*, 18–21. [CrossRef] [PubMed]
182. Hermans, T.; Santens, P.; Matton, C.; Oostra, K.; Heylens, G.; Herremans, S.; Lemmens, G.M.D. Anti-NMDA Receptor Encephalitis: Still Unknown and Underdiagnosed by Physicians and Especially by Psychiatrists? *Acta Clin. Belg.* **2018**, *73*, 364–367. [CrossRef] [PubMed]
183. Voice, J.; Ponterio, J.M.; Lakhi, N. Psychosis Secondary to an Incidental Teratoma: A "Heads-up" for Psychiatrists and Gynecologists. *Arch. Womens Ment. Health* **2017**, *20*, 703–707. [CrossRef] [PubMed]
184. Mantere, O.; Saarela, M.; Kieseppä, T.; Raij, T.; Mäntylä, T.; Lindgren, M.; Rikandi, E.; Stoecker, W.; Teegen, B.; Suvisaari, J. Anti-Neuronal Anti-Bodies in Patients with Early Psychosis. *Schizophr. Res.* **2018**, *192*, 404–407. [CrossRef] [PubMed]
185. Xiao, X.; Gui, S.; Bai, P.; Bai, Y.; Shan, D.; Hu, Y.; Bui-Nguyen, T.M.; Zhou, R. Anti-NMDA-Receptor Encephalitis during Pregnancy: A Case Report and Literature Review. *J. Obstet. Gynaecol. Res.* **2017**, *43*, 768–774. [CrossRef] [PubMed]
186. Singh, R.K.; Bhoi, S.K.; Kalita, J.; Misra, U.K. Cerebral Venous Sinus Thrombosis Presenting Feature of Systemic Lupus Erythematosus. *J. Stroke Cerebrovasc. Dis.* **2017**, *26*, 518–522. [CrossRef] [PubMed]

187. Schou, M.; Sæther, S.G.; Borowski, K.; Teegen, B.; Kondziella, D.; Stoecker, W.; Vaaler, A.; Reitan, S.K. Prevalence of Serum Anti-Neuronal Autoantibodies in Patients Admitted to Acute Psychiatric Care. *Psychol. Med.* **2016**, *46*, 3303–3313. [CrossRef]
188. Pollak, T.A.; McCormack, R.; Peakman, M.; Nicholson, T.R.; David, A.S. Prevalence of Anti-N-Methyl-D-Aspartate (NMDA) Receptor [Corrected] Antibodies in Patients with Schizophrenia and Related Psychoses: A Systematic Review and Meta-Analysis. *Psychol. Med.* **2014**, *44*, 2475–2487. [CrossRef]
189. Bertsias, G.K.; Ioannidis, J.P.A.; Aringer, M.; Bollen, E.; Bombardieri, S.; Bruce, I.N.; Cervera, R.; Dalakas, M.; Doria, A.; Hanly, J.G.; et al. EULAR Recommendations for the Management of Systemic Lupus Erythematosus with Neuropsychiatric Manifestations: Report of a Task Force of the EULAR Standing Committee for Clinical Affairs. *Ann. Rheum. Dis.* **2010**, *69*, 2074–2082. [CrossRef]
190. Steinlin, M.I.; Blaser, S.I.; Gilday, D.L.; Eddy, A.A.; Logan, W.J.; Laxer, R.M.; Silverman, E.D. Neurologic Manifestations of Pediatric Systemic Lupus Erythematosus. *Pediatr. Neurol.* **1995**, *13*, 191–197. [CrossRef]
191. Yancey, C.L.; Doughty, R.A.; Athreya, B.H. Central Nervous System Involvement in Childhood Systemic Lupus Erythematosus. *Arthritis Rheum* **1981**, *24*, 1389–1395. [CrossRef] [PubMed]
192. Weiss, D.B.; Dyrud, J.; House, R.M.; Beresford, T.P. Psychiatric Manifestations of Autoimmune Disorders. *Curr. Treat. Options Neurol.* **2005**, *7*, 413–417. [CrossRef] [PubMed]
193. Buhrich, N.; Cooper, D.A. Requests for Psychiatric Consultation Concerning 22 Patients with AIDS and ARC. *Aust. N. Z. J. Psychiatry* **1987**, *21*, 346–353. [CrossRef]
194. Shulman, L.M.; David, N.J.; Weiner, W.J. Psychosis as the Initial Manifestation of Adult-Onset Niemann-Pick Disease Type C. *Neurology* **1995**, *45*, 1739–1743. [CrossRef]
195. Huang, C.C.; Chu, N.S. Wilson's Disease: Clinical Analysis of 71 Cases and Comparison with Previous Chinese Series. *J. Formos. Med. Assoc.* **1992**, *91*, 502–507.
196. Walterfang, M.; Wood, S.J.; Velakoulis, D.; Copolov, D.; Pantelis, C. Diseases of White Matter and Schizophrenia-like Psychosis. *Aust. N. Z. J. Psychiatry* **2005**, *39*, 746–756. [CrossRef]
197. Albers, J.W.; Fink, J.K. Porphyric Neuropathy. *Muscle Nerve* **2004**, *30*, 410–422. [CrossRef]
198. Qadiri, M.R.; Church, S.E.; McColl, K.E.; Moore, M.R.; Youngs, G.R. Chester Porphyria: A Clinical Study of a New Form of Acute Porphyria. *Br. Med. J. Clin. Res. Ed.* **1986**, *292*, 455–459. [CrossRef]
199. Cooper, J.J.; Ovsiew, F. The Relationship between Schizophrenia and Frontotemporal Dementia. *J. Geriatr. Psychiatry Neurol.* **2013**, *26*, 131–137. [CrossRef]
200. Griswold, K.S.; Del Regno, P.A.; Berger, R.C. Recognition and Differential Diagnosis of Psychosis in Primary Care. *Am. Fam. Physician* **2015**, *91*, 856–863.
201. ALD n° 23—Schizophrénies. Available online: https://www.has-sante.fr/jcms/c_565630/fr/ald-n-23-schizophrenies (accessed on 17 August 2023).
202. Canadian Psychiatric Association Clinical Practice Guidelines. Treatment of Schizophrenia. *Can. J. Psychiatry* **2005**, *50*, 7S–57S.
203. Lehman, A.F.; Lieberman, J.A.; Dixon, L.B.; McGlashan, T.H.; Miller, A.L.; Perkins, D.O.; Kreyenbuhl, J.; American Psychiatric Association; Steering Committee on Practice Guidelines. Practice Guideline for the Treatment of Patients with Schizophrenia, Second Edition. *Am. J. Psychiatry* **2004**, *161*, 1–56. [PubMed]

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