SECTION 1 Diagnostics

Entries A–Z

A

Absence Seizures (Petit Mal or Generalized Nonconvulsive Seizures)

Epidemiology and Demographics: Typical absence seizures account for 10% of epileptic seizures in children. Annual incidence is estimated at 0.7–4.6/100,000 in the general population, and up to 6–8/100,000 in children aged 0–15 years. Associated with a variety of generalized epilepsy syndromes. Age of onset varies depending on the coinciding syndrome, generally in the first two decades. Early onset more consistent with childhood absence epilepsy or myoclonic astatic epilepsy, whereas presentation near puberty supports juvenile absence epilepsy or juvenile myoclonic epilepsy. May occur among other seizure types in more severe epilepsy syndromes, such as Lennox–Gastaut syndrome.

Disorder Description: Nonconvulsive generalized seizure type characterized by brief episodes of abrupt loss of consciousness, lasting 10–20 seconds, which may occur on a frequent, recurrent basis. Associated with subtle freezing or oral or manual automatisms. Of shorter duration than complex partial seizures, and a postictal period is not present. Events can be mistaken for daydreaming, which may contribute to delay in diagnosis. Typical absence seizures have a 3-Hz generalized spike and wave pattern on the EEG, often provoked by hyperventilation. Ethosuximide is used when absence seizures occur in isolation, valproate when there is co-occurrence with generalized tonic–clonic seizures (GTCS).

Atypical absence seizures have less abrupt onset and offset and may be prolonged. Often presenting with a loss of muscle tone (a gradual slump) and subtle myoclonic jerks. Loss of awareness may be incomplete, with physical activity continuing more slowly or with frequent errors. Atypical absence seizures are associated with a generalized slow spike and wave pattern of <2.5 Hz, and typically occur in the setting of more severe epilepsy syndromes with intellectual impairment.

Symptoms

Localization site	Comment
Cerebral hemispheres	Sudden interruption of consciousness. Automatisms, in the form of lip-smacking, chewing, and fumbling movements of the fingers. Mild tonic stiffening
Mental status and psychiatric aspects/complications	Deficits in global cognitive function, attention and focus, visual-spatial functions, and visual memory most pronounced during active disease. Cognitive status depends on underlying syndrome

Secondary Complications: Attentional issues may result in decreased school performance. Increased risk of accidental injury associated with driving or operating heavy machinery.

Treatment Complications: Some antiepileptic drugs (AEDs), such as vigabatrin, gabapentin, phenytoin, phenobarbital, or carbamazepine and their derivatives, can worsen seizures and should be avoided for certain epilepsy types. Valproate side effects include hyperactivity, weight gain, transaminitis, thrombocytopenia, pancreatitis, and known teratogenic effects. Ethosuximide most commonly causes dizziness and gastrointestinal complaints. Rare side effects include psychosis.

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Acquired Hepatocerebral Degeneration

Epidemiology and Demographics: Acquired hepatocerebral degeneration (AHD) is a rare condition that occurs in about 1% of patients with cirrhosis. This condition can occur in patients with severe and decompensated chronic liver failure and can happen at any age.

Disorder Description: AHD is associated with portosystemic shunting, either iatrogenic or spontaneous. Clinical presentation of AHD closely resembles Wilson's disease and therefore it is sometimes called non-Wilsonian hepatocerebral degeneration. Patients often have hepatic encephalopathy, followed by extrapyramidal symptoms such as rigidity, bradykinesia, tremor, and choreoathetosis. MRI of the brain often shows hyperintense signal changes on T1-weighted images and hypointense or normal signal on T2-weighted images in the basal ganglia and cerebellum.

Symptoms

Localization site	Comment
Cerebral hemispheres	Hepatic encephalopathy Parkinsonism
Cerebellum	Ataxia

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Actinomycosis

Epidemiology and Demographics: Actinomycosis affects males more so than females, between 20 and 60 years old.

Disorder Description: Actinomycosis is a slowly progressive bacterial infection, most commonly caused by the gram-positive, anaerobic, or microaerophilic rod *Actinomyces israelii*. It normally resides in the nasopharyngeal, gastrointestinal, and female genital tracts. Infection may spread by cervicofacial infections (most common) or hematogenous spread. Risk factors for infection include poor oral hygiene or dental procedures, trauma, use of contraceptive intrauterine devices (IUDs), or immunocompromised state.[1]

Symptoms

Localization site	Comment
Cerebral hemispheres	Brain abscesses (most common) Meningitis Encephalitis Subdural empyema Dural sinus thrombosis[2]
Base of skull	Basilar meningitis
Spinal cord	Epidural abscess causing cord compression Myelopathy[3]
Syndromes with combined spinal cord and peripheral nerve lesions	Myeloradiculopathy[3]

Secondary Complications: Chronic meningitis may develop from brain abscesses, which may lead to hydrocephalus.

Treatment Complications: Response to medication may be slow and take months to take effect.

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Acute Brachial Neuritis (Parsonage–Turner Syndrome)

Epidemiology and Demographics: More common among middle-aged men (70%); average age 41.4 years. Mostly unilateral onset, involving the dominant limb.

Disorder Description: Acute brachial neuritis, also known as Parsonage–Turner syndrome, brachial plexus neuritis, or neuralgic amyotrophy, is an immune-mediated, multifocal peripheral nervous system disorder. Most lesions are axonal but some may be demyelinating. Targets predominantly proximal motor axons. Most commonly affected nerves include the long thoracic, suprascapular, axillary, and musculocutaneous. The diagnosis is difficult, with single nerve involvement; local compression may be suspected first. The symptoms include sudden onset of severe pain in the shoulder or scapular area that radiates down the arm or up the neck. The pain often awakens patient from sleep. The pain usually resolves within a few weeks. Weakness becomes evident as the pain resolves and the limb is being used more frequently. Can lead to muscle atrophy. Paresthesia is less intense than the pain. The treatment includes analgesics, including narcotics and neuropathic pain medications. Oral corticosteroid course may also be beneficial in reducing the pain. After pain has been controlled, physical and occupational therapies are recommended. Recovery rates of 36% at 1 year, 75% at 2 years, and 89% at 3 years have been reported.

Symptoms

Localization site	Comment
Plexus	Brachial plexus, trunk, cord, nerve branches
Mononeuropathy or mononeuropathy multiplex	Can present as single or multiple nerve involvement
Muscle	Atrophy and weakness

Treatment Complications: Narcotic analgesics used for painful crises can lead to sedation or depressed mentation.

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Acute Disseminated Encephalomyelitis (ADEM)

Epidemiology and Demographics: Acute disseminated encephalomyelitis (ADEM) has an incidence of 0.2–0.4/100,000 per year in North America. There are no clear data suggesting higher prevalence in one race. However, it is found to be slightly more common in males than females, given a gender ratio of 1.3:1. The median age of onset is 5–8 years, and in North America its peak incidence is in the winter and spring months.

Disorder Description: An autoimmune demyelinating disease involving the central nervous system. It is usually preceded by viral infection or immunizations. It usually presents immediately after an infectious illness, or several days later. The onset is usually acute to subacute. Symptoms present over hours or 1 to 2 days. There are encephalitic and myelitic types. Encephalitic symptoms include confusion, seizures, fever, somnolence, headache, and neck stiffness. Rarely, choreoathetosis occurs. Patients can experience decerebrate rigidity and coma. Patients with myelitic type experience loss of sensation, bowel/bladder paralysis, and varying degrees of extremity weakness, paraplegia, or quadriplegia.

Symptoms

Localization site	Comment
Cerebral hemispheres	Seizures, confusion, coma, meningeal irritation
Mental status and psychiatric aspects/complications	Confusion, lethargy
Brainstem	Decerebrate rigidity, coma
Cranial nerves	Optic neuritis
Spinal cord	Quadriplegia, paraplegia, sensory disturbance, bowel/bladder dysfunction

Treatment Complications: Steroid therapy can lead to psychosis, confusion, weight gain, hyperglycemia, myopathy, glaucoma, cataracts, osteoporosis, or vertebral fractures.

Plasma exchange complications include anaphylaxis, hypocalcemia, and depletion of immunoglobulins and coagulation factors.

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Acute Inflammatory Demyelinating Polyneuropathy (AIDP) and Miller Fisher Variant of Guillain–Barré Syndrome

Epidemiology and Demographics: Acute inflammatory demyelinating polyneuropathy (AIDP) has a worldwide incidence of 0.6–4.0/100,000 with a mean age of onset of 40 years. Overall incidence is lower in children, 0.34–1.34/100,000, and higher in people over age 50, 1.7–3.3/100,000. It is more common in males, with a male-to-female ratio of 1.7:1. It is found in all races. It has no predilection for a specific time of year; however, summer epidemics of the axonal variant have been reported in northern China. Two-thirds of cases are preceded by upper respiratory infection or diarrhea due to *Campylobacter jejuni*. Has been associated with swine flu H1N1 vaccine.

Disorder Description: AIDP is an autoimmune attack of peripheral nerve myelin with secondary axonal loss. It typically presents as a symmetric ascending weakness, mild sensory loss, and hypo- or areflexia following a viral illness and, in some cases, more specifically with infection with *C. jejuni*. Its onset occurs over several days to 2 weeks. Proximal muscles can be affected before distal. The most common initial symptom is numbness of the toes. Weakness usually starts in the legs, ascends to the arms, and at times can lead to facial diplopia and respiratory failure. It can also lead to autonomic dysfunction including tachycardia, bradycardia, facial flushing, and fluctuating blood pressure. Urinary retention occurs in 15% of patients. Recovery occurs over weeks to months. Most patients recover with mild residual motor and sensory deficits. However, 10% of patients are left with disability.

Severe respiratory muscle weakness requiring ventilator support develops in 10–30% of patients, paresthesia (and sometimes pain) in the hands and feet occurs in up to 80% of patients. Dysautonomia occurs in 70% of patients, presenting as tachycardia (most common), urinary retention, and alternating hypertension/hypotension, and can be associated with sudden death.

Miller Fisher syndrome is a variant of Guillain–Barré syndrome (GBS)/AIDP. It presents as a descending paralysis. The most common manifestations are ophthalmoplegia, ataxia, and areflexia. Eighty-five percent to 90% of patients with Miller Fisher syndrome have antibodies against GQ1b, a ganglioside nerve component.

Diagnosis is clinical, confirmed with cerebrospinal fluid (CSF) studies and electromyography/nerve conduction velocity (EMG/NCV) tests. Time course is about 4 weeks and self-limiting. Other conditions to consider in the differential diagnosis include vitamin B1 deficiency, acute arsenic poisoning, n-hexane exposure, vasculitis, Lyme disease, tick paralysis, porphyria, sarcoidosis, leptomeningeal disease, paraneoplastic disease, critical illness myopathy, and chronic inflammatory demyelinating polyneuropathy (CIDP).

Symptoms

Localization site	Comment
Cerebral hemispheres	Confusion, coma in variants of GBS, i.e., Bickerstaff encephalitis
Mental status and psychiatric aspects/complications	Confusion, coma in variants of GBS, i.e., Bickerstaff encephalitis
Brainstem	Confusion, coma, ophthalmoplegia, ataxia in variants of GBS, i.e., Bickerstaff encephalitis

Localization site	Comment
Cranial nerves	Cranial nerves III–VII and IX–XII may be involved, diplopia, ophthalmoplegia, dysphagia, dysarthria, facial droop
Peripheral nerve/muscle	Sensory disturbance, symmetric limb weakness, areflexia, respiratory failure, bulbar muscle weakness, myalgias
Unclear localization	Dysautonomia due to involvement of parasympathetic and sympathetic systems

Secondary Complications: Respiratory failure leading to mechanical ventilation and aspiration pneumonia. Immobilization leading to ileus, pulmonary embolus, or deep vein thrombosis. There can be residual weakness, sensory loss, or neuropathic pain. Death occurs in 3–5% of cases.

Treatment Complications: Initial treatment is supportive care – monitoring in the ICU setting, close respiratory monitoring (negative inspiratory force [NIF]/vital capacity [VC]), and ventilator support if necessary. For patients with dysautonomia, cardiac and blood pressure monitoring may be necessary. Main therapies include intravenous immunoglobulin (IVIG) or plasma exchange, which shortens the time to walking independently by 40–50%. Plasma exchange is recommended over IVIG for patients who are nonambulatory. There is a reported residual neuropathic pain in 40–50% of patients during the course of GBS.

Plasma exchange complications include anaphylaxis, hypocalcemia, and depletion of immunoglobulins and coagulation factors.

Adverse effects from IVIG include local injection site reaction. More serious side effects include anaphylaxis, from IgA deficiency, chest pain, myocardial infarction, tachycardia, hyponatremia, hemolysis, hemolytic anemia, thrombosis, hepatitis, anaphylaxis, backache, severe headache, aseptic meningitis, proteinuria, acute renal failure, hypokalemic nephropathy, pulmonary embolism, and transfusion-related lung injury. Plasmapheresis potential problems include mild allergic reaction, anaphylaxis, infection, or hypotension.

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Acute Stress Disorder

Epidemiology and Demographics: Prevalence rate is dependent on the nature and severity of the preceding traumatic event as well as the criteria used to define the disorder. Estimates range from 20 to 50% with interpersonal trauma (e.g., assault, rape, witnessing a mass shooting) and less than 20% with other forms of trauma (e.g., accidents, mild traumatic brain injury, burns). More common in women.

Disorder Description: Acute stress disorder is characterized by development of significant distress or functional impairment post exposure to a traumatic event. Per DSM-5, a traumatic event is defined as "exposure to actual or threatened death, serious injury or sexual violence."[1] To meet criteria, symptoms must be present within 3 days to 1 month after the traumatic exposure. Symptoms include intrusive memories and/or dreams, dissociative reactions (e.g., flashbacks), negative mood, dissociative symptoms (e.g., amnesia for aspects of the event), avoidance symptoms, and hyperarousal, including sleep disturbance, irritability, unprovoked anger or aggression, and/or exaggerated startle response. Symptoms are not better accounted for by effects of a substance, medical condition, or brief psychotic disorder. Risk factors include pre-existing psychiatric disorder, high severity of exposure, presence of prior traumatic exposure, and female gender.

Symptoms

Localization site	Comment
Mental status and psychiatric aspects/complications	Intrusive thoughts, flashbacks, dissociative symptoms (e.g., amnesia), nightmares, deficits in attention, concentration and declarative memory, hyperarousal symptoms (e.g., sleep disturbances, irritability, unprovoked anger/aggression, exaggerated startle response), anhedonia

Secondary Complications: Posttraumatic stress disorder, alcohol/substance abuse, depressive disorders, anxiety disorders, sleep disorders.

Treatment Complications: Treatment with benzodiazepines may improve hyperarousal symptoms. However, there is risk for sedation and development of dependence and tolerance if used chronically.

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1. American Psychiatric Association. *Diagnostic and statistical manual of mental disorders: DSM-5*. 5th ed. Washington, DC: American Psychiatric Association; 2013.

Suggested Reading

Kessler RC, Sonnega A, Bromet E, et al. Posttraumatic stress disorder in the national comorbidity survey. *Arch Gen Psychiatry*. 1995;52(12):1048–60.

Adenovirus

Epidemiology and Demographics: Worldwide distribution and infections occur in every season. Causes 5–10% of all febrile illnesses in infants and young children.[1]

Central nervous system (CNS) dysfunction was identified in 3.3% of children with adenovirus infection, mostly occurring in those younger than 5 years.[2]

Disorder Description: Adenoviruses are nonenveloped viruses with double-stranded DNA that most commonly cause upper respiratory tract illness, but may cause other systemic illness. Risk factors for infection include immunosuppression, people who live in close quarters (those in military barracks or college dormitories). Adenoviruses tend to be more prevalent in day-care centers and in households with young children. Transmission may be via aerosol droplets, fecal–oral route, transplants, vertical transmission during vaginal birth, and by contact with contaminated fomites.[3]

Symptoms

Localization site	Comment
Cerebral hemispheres	Meningitis[2] Encephalitis Seizures Hydrocephalus
Mental status and psychiatric aspects/complications	Altered mental status Visual hallucinations
Spinal cord	Transverse myelitis
Anterior horn cells	Flaccid paralysis

Secondary Complications: Seizure disorder may be a complication.

Treatment Complications: Ribavirin may cause hyperammonemia, which may suppress mental status.

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Adenylosuccinate Lyase Deficiency

Epidemiology and Demographics: 1/70,000 live births; rare, autosomal recessive. Second-most common urea cycle disorder. Druze community in Israel has carrier frequency 1/41, with 20 cases also being diagnosed in Finland. Associated with high morbidity and mortality rates. A low-protein diet initiated early may prevent neurologic sequelae. ASL gene maps to 7q11.21

Disorder Description: Argininosuccinate (ASA) lyase deficiency results in defective cleavage of ASA and argininosuccinic aciduria. It leads to deficiency of both ureagenesis and nitric oxide (NO) production. Like all urea cycle disorders, it has a propensity to cause hyperammonemia in affected individuals.

Adenylosuccinate lyase (ASL) deficiency presents in two main forms – a severe neonatal form and late-onset form.

The severe neonatal form is indistinguishable from other urea cycle disorders, characterized by the triad of hyperammonemia, encephalopathy, and respiratory alkalosis. Vomiting, lethargy, and hypothermia present a few days after birth, and if left untreated or unrecognized, can result in death.

Late-onset forms are characterized by episodic hyperammonemia, triggered by acute infection, stress, or dietary noncompliance. Patients may also display chronic symptoms such as episodic vomiting, mental status changes, lethargy, and behavioral abnormalities associated with hyperammonemia. Some patients may present with cognitive impairment, behavioral abnormalities, or learning disabilities in the absence of any documented episodes of hyperammonemia. A pathognomonic phenotype includes a combination of neurocognitive deficiency, hepatitis with cirrhosis, trichorrhexis nodosa (friable hair), and systemic hypertension (due to NO deficiency).

ASA is an intermediate in the pathway of urea synthesis from ammonia and is split into arginine and fumarate in a reaction catalyzed by argininosuccinate lyase. As a result of this enzyme defect, patients have increased ammonia, both ASA and citrulline accumulate in the blood, and ASA is excreted in the urine. There is considerable phenotypic variability. Symptoms related to the central nervous system (CNS) are due to the toxic effects of hyperammonemia, although some have suggested a possible direct toxic effect of argininosuccinic acid on the brain.

Symptoms

Localization site	Comment
Cerebral hemispheres	In acute setting of hyperammonemia – delirium, somnolence, obtundation, coma, cerebral edema, seizures Long term – delayed development, intellectual disabilities, behavioral difficulties, learning difficulties, attention deficit hyperactivity disorder
Basal ganglia/cerebellum	Hypotonia, ataxia, tremor, choreoathetotic movement disorder

Secondary Complications: Hepatic failure may lead to a hepatic encephalopathy. Secondary coagulopathy may lead to intracranial hemorrhage.

Treatment Complications: Treatment of metabolic crises consists of withdrawal of protein from diet; promotion of anabolism (supplementing oral intake with intravenous lipids, glucose, and intravenous insulin); and sodium benzoate (intravenous nitrogen scavenging therapy).

Long-term interventions include lifelong dietary management (protein restriction), arginine base supplementation, and oral nitrogen scavenging therapy.

Complications of treatments include electrolyte disturbances or dehydration.

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Adjustment Disorder

Epidemiology and Demographics: Adjustment disorder (AD) is diagnosed in an estimated 5–20% of individuals in outpatient mental health settings and up to 50% of inpatient psychiatric consultations.

Disorder Description: AD is defined in DSM-5 as the manifestation of marked, disproportionate distress and/or impaired function referable to an identifiable stressor. Symptoms must be clinically significant, with onset within 3 months of the stressor and continue no more than 6 months post-resolution of the stressor or associated consequences. Symptoms must not be better accounted for by bereavement, another mental disorder, or exacerbation of a pre-existing condition.

Can be associated with anxiety, depressed mood, conduct disturbance, or a combination of these. Classified as acute (duration less than 6 months) versus chronic (duration greater than 6 months). Commonly occurs in setting of coexisting psychiatric and/or medical illness.

Symptoms

Localization site	Comment
Mental status and psychiatric aspects/complications	Depressed mood, anxiety, rage, antisocial behavior

Secondary Complications: AD with depressed mood carries an increased risk for attempted and completed suicide. When disturbance of conduct is associated, there can be reckless and/or aggressive behavior toward property or others.

Treatment Complications: Both brief supportive psychotherapy and short-term use of benzodiazepines and antidepressant medication may be considered. Side effects of benzodiazepines include risk for sedation and development of dependence and tolerance if used chronically. Selective serotonin reuptake inhibitor (SSRI) medications have rare potential to increase risk of suicide; therefore, they should be monitored with caution.

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Adrenal Insufficiency

Epidemiology and Demographics: Secondary adrenal insufficiency is more common than primary adrenal insufficiency (Addison's disease), with an estimated prevalence of 150–280 per million, affecting more women than men, usually in the sixth decade of life.[1]

Disorder Description: Life-threatening disorder caused by primary or secondary adrenal insufficiency due to a dysfunction of the hypothalamic–pituitary axis. Most cases of primary adrenal insufficiency (failure of the adrenal glands to produce sufficient cortisol and sometimes aldosterone) are caused by autoimmune adrenalitis. Secondary adrenal insufficiency is usually caused by any process that interferes with corticotropin secretion that involves the pituitary gland.[2]

Symptoms

Localization site	Comment
Cerebral hemispheres	Seizures precipitated by hyponatremia/hypoglycemia
Mental status and psychiatric aspects/complications	Memory impairment, depression, psychosis[3] more common in primary adrenal insufficiency Confusion or disorientation seen in the acute setting of bilateral adrenal injury
Cranial nerves	External ophthalmoplegia as seen in Kearns–Sayre syndrome
Pituitary gland	Severe headache and visual symptoms with organic injury to the pituitary gland
Muscle	Myalgias and joint pain[4]; weakness; flexion contractures