

Review

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Review

A Comprehensive Review of Secondary Movement Disorders

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Abstract: Secondary movement disorders (SMDs) represent a heterogeneous group of neurological conditions characterized by abnormal, involuntary movements resulting from various underlying causes. This comprehensive review aims to thoroughly understand SMDs, including their epidemiology, pathophysiology, clinical manifestations, diagnostic approach, and management strategies. SMDs encompass a spectrum of movement abnormalities, including dystonia, chorea, athetosis, myoclonus, tics, and tremors. They can arise from a multitude of etiologies, such as drug-induced effects, post-infectious processes, metabolic disturbances, and structural brain lesions. The epidemiology of SMDs varies depending on the underlying cause, with some conditions being more prevalent in specific populations or age groups. Understanding the pathophysiology of SMDs is crucial for accurate diagnosis and targeted management. Diagnosing SMDs requires a comprehensive approach, including a detailed history, physical examination, laboratory investigations, neuroimaging studies, and sometimes electrophysiological studies. Management of SMDs is multifaceted and may involve pharmacological, non-pharmacological, and surgical interventions. Individualized treatment plans are essential, considering the underlying cause, severity of symptoms, and patient preferences. Emerging concepts in the field of SMDs include advancements in genetic research, identification of biomarkers, and development of novel therapeutic approaches. There is a growing focus on patient-centered care and improving the quality of life for individuals with SMDs. In conclusion, SMDs pose diagnostic and therapeutic challenges, requiring a multidisciplinary approach for optimal management. Continued research and collaboration are essential for advancing our understanding and treatment of these complex disorders.

Keywords: secondary movement disorders; dystonia; chorea; athetosis; myoclonus; tics; tremor; drug-induced movement disorders; post-infectious movement disorders; metabolic disorders

1. Introduction

1.1. Definition and Classification of Secondary Movement Disorders

Secondary movement disorders (SMDs) are a group of neurological conditions characterized by abnormal, involuntary movements that result from various underlying etiologies [1]. These disorders are categorized based on their clinical features, underlying causes, and affected anatomical regions. Common types of SMDs include dystonia, chorea, athetosis, myoclonus, tics, and tremor. Dystonia involves sustained or intermittent muscle contractions that lead to abnormal movements or postures, which can be focal, segmental, or generalized. Chorea is characterized by irregular, rapid, jerky movements that flow randomly from one body part to another [2]. Athetosis is marked by slow, writhing movements, often affecting the hands, feet, and face. Myoclonus manifests as sudden, brief muscle contractions or jerks, while tics are sudden, repetitive movements or vocalizations [3].

The classification of SMDs is based on the underlying cause, which can include drug-induced effects, post-infectious processes, metabolic disturbances, and structural brain lesions. Drug-induced movement disorders can result from medications such as antipsychotics, antidepressants, and

antiepileptic drugs. Post-infectious movement disorders may occur following streptococcal infections, leading to conditions like Sydenham's chorea. Metabolic disorders such as Wilson's disease and hyperthyroidism [4] can also cause movement abnormalities. Structural brain lesions, including tumors [5], strokes [6], and other abnormalities, can disrupt standard movement control pathways [7], leading to movement disorders [8].

Understanding the classification and underlying causes of SMDs is crucial for accurate diagnosis and management [9]. A comprehensive evaluation, including a detailed history, physical examination, laboratory tests, and neuroimaging studies, is essential to determine the underlying cause of the movement disorder. Treatment strategies for SMDs vary depending on the underlying cause and may include medications, physical therapy, and surgery. A multidisciplinary approach involving neurologists, movement disorder specialists, and other healthcare professionals is often necessary to provide optimal care for individuals with SMDs.

1.2. Epidemiology of Secondary Movement Disorders

The epidemiology of secondary movement disorders (SMDs) varies depending on the underlying cause and the specific type of movement disorder [10]. Overall, SMDs are considered rare compared to primary movement disorders. However, certain SMDs may be more prevalent in particular populations or age groups.

One of the most common causes of SMDs is drug-induced movement disorders, which can occur in individuals of any age but are more commonly seen in older adults due to the higher prevalence of conditions requiring medication [11]. Antipsychotic medications, which are widely used in the elderly population, are a significant cause of drug-induced movement disorders.

Post-infectious movement disorders, such as Sydenham's chorea, are more commonly seen in children and adolescents following streptococcal infections. These movement disorders are thought to occur due to an autoimmune response triggered by the infection.

Metabolic disorders, such as Wilson's disease, can lead to movement abnormalities and are more commonly seen in young adults. Wilson disease is a genetic disorder that affects copper metabolism and can result in movement disorders, liver disease, and psychiatric symptoms.

Structural brain lesions, such as tumors or strokes, can occur at any age but are more common in older adults. These lesions can disrupt normal movement control pathways, leading to various movement disorders depending on the location and extent of the lesion [12].

Overall, the epidemiology of SMDs is complex and varies depending on the underlying cause and population studied. Further research is needed to understand better the prevalence and risk factors associated with SMDs to improve diagnosis and management strategies [13].

1.3. Pathophysiology of Secondary Movement Disorders

The pathophysiology of secondary movement disorders (SMDs) is complex and varies depending on the underlying cause and specific type of movement disorder. However, common underlying mechanisms contribute to the development of SMDs across different etiologies. One of the critical pathophysiological mechanisms is dysfunction in the basal ganglia-thalamocortical circuit, which plays a crucial role in motor control. Disruption of this circuitry can lead to abnormal movements seen in SMDs.

In drug-induced movement disorders, such as tardive dyskinesia, antipsychotic medications block dopamine receptors in the basal ganglia, leading to an imbalance in dopamine signaling. This imbalance can result in hyperactivity of indirect pathway neurons, which are involved in inhibiting movement, leading to abnormal involuntary movements. Similarly, in metabolic disorders like Wilson disease, copper accumulation in the basal ganglia can disrupt normal neurotransmission and lead to movement abnormalities [14]. And the glycemic level abnormalities can cause dyskinesias [15].

In post-infectious movement disorders like Sydenham's chorea [16], an autoimmune response triggered by streptococcal infections can result in inflammation and damage to the basal ganglia [17]. This damage disrupts the normal function of the basal ganglia-thalamocortical circuit, leading to

choreiform movements [18]. Structural brain lesions, such as tumors or strokes, can directly damage the basal ganglia or its connections, leading to abnormal movements. Additionally, these lesions can disrupt the motor cortex's average inhibitory and excitatory balance, further contributing to developing movement disorders [19].

Overall, the pathophysiology of SMDs involves complex interactions between various brain regions involved in motor control. Dysfunction in the basal ganglia-thalamocortical circuit is a common feature across different etiologies of SMDs. Understanding these underlying mechanisms is crucial for developing targeted treatment strategies for SMDs.

2. Clinical Manifestations of Movement Disorders

Clinical manifestations of movement disorders vary widely depending on the specific type of disorder and the underlying cause. However, some standard features can help distinguish different movement disorders and guide diagnostic evaluation.

One of the critical features of movement disorders is the type of abnormal movement involved. For example, in dystonia, patients may exhibit sustained or intermittent muscle contractions that result in abnormal postures or repetitive movements. These movements are often patterned and may be triggered or worsened by specific actions or positions. In contrast, chorea is characterized by brief, irregular, and unpredictable movements that flow from one body part to another, giving a "dance-like" appearance. Athetosis is characterized by slow, writhing movements, particularly affecting the hands, feet, and face. Myoclonus involves sudden, brief muscle contractions or jerks that can occur spontaneously or in response to stimuli. Tics are sudden, repetitive movements or vocalizations often preceded by an urge or sensation.

The distribution of abnormal movements can also provide important clues to the underlying diagnosis. Dystonia can be focal, affecting a single body region, such as the neck (cervical dystonia or torticollis), hand (writer's cramp), or face (blepharospasm). It can also be segmental, involving adjacent body regions, or generalized, affecting multiple body regions. Chorea and athetosis typically affect various body regions and can be generalized or more pronounced on one side of the body [20]. Myoclonus can be focal, segmental, or generalized, and the distribution of movements can vary depending on the underlying cause. Tics are often multifocal, involving different body parts at other times.

The temporal profile of abnormal movements is another important consideration. Some movement disorders, such as Parkinson's, exhibit a characteristic pattern of progression over time [21]. Parkinson's disease is characterized by gradual symptoms, starting with a unilateral resting tremor, bradykinesia (slowness of movement), rigidity, and postural instability. These symptoms typically progress slowly over the years, with asymmetric involvement early in the disease course. In contrast, some drug-induced movement disorders, such as acute dystonia or akathisia, can have a sudden onset and may occur shortly after starting or changing medications [22].

Other neurological or non-neurological features can also help differentiate between different movement disorders [23]. For example, certain movement disorders, such as Huntington's disease, are associated with cognitive impairment and psychiatric symptoms. Wilson disease, a metabolic disorder that can cause movement disorders, is also associated with liver dysfunction and psychiatric symptoms. Structural brain lesions, such as tumors or strokes, may present with focal neurological deficits in addition to abnormal movements.

Overall, the clinical manifestations of movement disorders are diverse and can vary widely depending on the specific type of disorder and underlying cause. A careful history, physical examination, and appropriate diagnostic testing are essential for accurate diagnosis and management.

3. Etiologies of Secondary Movement Disorders

3.1. Drug-Induced Movement Disorders

3.1.1. Antipsychotic Medications

Antipsychotic medications are commonly used in the treatment of psychiatric disorders such as schizophrenia, bipolar disorder, and severe depression with psychotic features. However, one of the significant side effects associated with these medications is the development of movement disorders, known as antipsychotic-induced movement disorders (AIMDs). These AIMDs can range from acute and reversible to chronic and irreversible conditions, posing significant challenges in the management of patients receiving antipsychotic treatment [24].

One of the most well-known AIMDs is tardive dyskinesia (TD), characterized by involuntary, repetitive movements, most commonly involving the face and mouth, such as lip smacking, tongue protrusion, and grimacing. TD is more familiar with typical antipsychotics, such as haloperidol and chlorpromazine, although it can also occur with atypical antipsychotics, such as risperidone and olanzapine. The pathophysiology of TD is not fully understood but is thought to involve dopamine receptor hypersensitivity and changes in dopamine receptor density in the basal ganglia. Therefore, the benefits and malicious effects of antipsychotics should be discussed with the patients and caregivers [25].

Acute dystonia is another AIMD characterized by sustained muscle contractions, most commonly affecting the neck, face, and tongue muscles, leading to abnormal postures or movements. Acute dystonia typically occurs within the first few days of starting or increasing the dose of antipsychotic medication and is more familiar with high-potency typical antipsychotics, such as haloperidol. Acute dystonia is believed to result from an imbalance between dopamine and acetylcholine in the basal ganglia [26].

Parkinsonism, characterized by symptoms such as bradykinesia (slowness of movement), rigidity, tremor, and postural instability, is another common AIMD associated with antipsychotic use. Parkinsonism is more familiar with typical antipsychotics and is thought to result from the blockade of dopamine D2 receptors in the nigrostriatal pathway, which is involved in motor control. Atypical antipsychotics have a lower risk of causing Parkinsonism due to their lower affinity for D2 receptors.

Akathisia is a subjective feeling of restlessness and an urge to move, often accompanied by objective findings such as rocking back and forth or pacing. Akathisia is more familiar with typical antipsychotics and can be mistaken for agitation or worsening of underlying psychiatric symptoms. The pathophysiology of akathisia is not well understood but is believed to involve dysregulation of dopaminergic and serotonergic pathways in the brain.

3.1.2. Antidepressant Medications

Antidepressant medications are widely used in the treatment of depression, anxiety disorders, and other psychiatric conditions [27]. While generally safe and effective, some antidepressants are associated with the development of movement disorders, known as antidepressant-induced movement disorders (AIMDs). These AIMDs can range from mild and reversible to severe and persistent, highlighting the importance of monitoring and early recognition of these side effects [28].

One of the most well-known AIMDs associated with antidepressants is serotonin syndrome, which can occur with the use of selective serotonin reuptake inhibitors (SSRIs), serotonin-norepinephrine reuptake inhibitors (SNRIs), and other serotonergic medications. Serotonin syndrome is characterized by a constellation of symptoms, including agitation, confusion, hyperthermia, tremor, and muscle rigidity, and can be life-threatening if not recognized and treated promptly. The exact mechanism of serotonin syndrome is not fully understood but is believed to involve excessive serotonergic activity in the central nervous system [29].

Another AIMD associated with antidepressants is drug-induced parkinsonism, which can occur with the use of certain antidepressants, particularly older tricyclic antidepressants (TCAs) [30], monoamine oxidase inhibitors (MAOIs) [31], and buspirone [32]. Drug-induced parkinsonism is characterized by symptoms similar to Parkinson's disease, including bradykinesia, rigidity, tremor, and postural instability. The pathophysiology of drug-induced parkinsonism is thought to involve

the blockade of dopamine receptors in the nigrostriatal pathway, leading to a relative deficiency of dopamine [33].

Other less common AIMDs associated with antidepressants include akathisia, dystonia, and tardive dyskinesia [34]. Akathisia is characterized by subjective restlessness and an urge to move, often accompanied by objective signs such as pacing or fidgeting [35]. Dystonia involves sustained muscle contractions that result in abnormal postures or movements, while tardive dyskinesia is characterized by involuntary, repetitive movements, most commonly involving the face and mouth [36]. Early recognition and management of these AIMDs are essential to prevent long-term complications and improve patient outcomes.

3.1.3. Antiepileptic Medications

Antiseizure medications, also known as antiepileptic drugs (AEDs), are commonly used to treat epilepsy and other neurological disorders [37]. While these medications are effective in controlling seizures, some are associated with the development of movement disorders, known as antiepileptic drug-induced movement disorders (AEDIMDs) [38]. These movement disorders can vary in severity and presentation, highlighting the importance of monitoring and early recognition of these side effects [39].

One of the most well-known AEDIMDs is drug-induced parkinsonism, which can occur with the use of specific AEDs [40], particularly older medications such as valproate [41] and carbamazepine [42]. Drug-induced parkinsonism is characterized by symptoms similar to Parkinson's disease, including bradykinesia, rigidity, tremor, and postural instability. The exact mechanism of drug-induced Parkinsonism is not fully understood [43]. Still, it is believed to involve the blockade of dopamine receptors in the nigrostriatal pathway, leading to a relative dopamine deficiency. Other authors believe valproate and abnormal movements are associated with the hyperammonemic state [44].

Another AEDIMD associated with antiseizure medications is tardive dyskinesia [45], which can occur with the use of drugs such as phenytoin [46] and carbamazepine. Tardive dyskinesia is characterized by involuntary, repetitive movements, most commonly involving the face and mouth, such as lip smacking, tongue protrusion, and grimacing [47]. The pathophysiology of tardive dyskinesia is not fully understood but is thought to involve dopamine receptor hypersensitivity and changes in dopamine receptor density in the basal ganglia.

Other less common AEDIMDs associated with antiseizure medications include dystonia, chorea, and myoclonus. Dystonia involves sustained muscle contractions that result in abnormal postures or movements, while brief, irregular, and unpredictable movements characterize chorea. Myoclonus refers to sudden, brief muscle contractions or jerks that can occur spontaneously or in response to stimuli [48]. Early recognition and management of these AEDIMDs are essential to prevent long-term complications and improve patient outcomes. Interestingly, new antiseizure medications besides Field were not associated with movement disorders [49].

3.1.4. Antiemetic Medications

Antiemetic medications prevent or treat nausea and vomiting, which can occur due to various causes such as motion sickness, chemotherapy, surgery, or gastrointestinal disorders [50]. While generally safe and effective, some antiemetics are associated with the development of movement disorders known as antiemetic-induced movement disorders (AIMDs). These AIMDs can range from mild and reversible to severe and persistent, highlighting the importance of monitoring and early recognition of these side effects.

One of the most well-known AIMDs associated with antiemetic medications is tardive dyskinesia (TD), which can occur with the use of dopamine receptor-blocking agents such as metoclopramide and prochlorperazine. TD is characterized by involuntary, repetitive movements, most commonly involving the face and mouth, such as lip smacking, tongue protrusion, and grimacing. The pathophysiology of TD is not fully understood but is thought to involve dopamine receptor hypersensitivity and changes in dopamine receptor density in the basal ganglia [51].

Another AIMD associated with antiemetic medications is acute dystonia, which can occur shortly after starting or increasing the dose of dopamine receptor-blocking agents. Acute dystonia is characterized by sustained muscle contractions, most commonly affecting the neck, face, and tongue muscles, leading to abnormal postures or movements. Acute dystonia is believed to result from an imbalance between dopamine and acetylcholine in the basal ganglia.

Other less common AIMDs associated with antiemetic medications include akathisia, which is characterized by subjective feelings of restlessness and an urge to move, and Parkinsonism, which involves symptoms such as bradykinesia (slowness of movement), rigidity, tremor, and postural instability. Early recognition and management of these AIMDs are essential to prevent long-term complications and improve patient outcomes. Also, using scales can help evaluate the individual for further assessment in the future [52].

3.1.5. Other Medications

Other medications, beyond those already mentioned, can also be associated with the development of movement disorders. These movement disorders, known as drug-induced movement disorders (DIMDs), can manifest as a variety of abnormal movements and can range in severity from mild to debilitating.

One example of a DIMD is drug-induced tremor, which can be caused by a variety of medications, including certain antidepressants, antipsychotics [53], and stimulants [54]. This tremor can vary in intensity and may affect different body parts, such as the hands, arms, head, or voice [55]. The exact mechanism of drug-induced tremor is not fully understood but is thought to involve disruption of the average balance of neurotransmitters in the brain.

Another example of a DIMD is drug-induced myoclonus, which is characterized by sudden, involuntary muscle jerks or twitches. Myoclonus can be caused by various medications, including certain antibiotics, antipsychotics, and anticonvulsants [56]. The mechanism of drug-induced myoclonus is thought to involve hyperexcitability of neurons in the brain and spinal cord [57]. Interestingly, among the antibiotics, fluoroquinolones were commonly associated with myoclonus [58], especially ciprofloxacin [59].

Certain medications can also cause drug-induced dystonia, which is characterized by sustained or intermittent muscle contractions that result in abnormal postures or movements [60]. Dystonia can be caused by drugs such as antipsychotics, antiemetics, and certain antidepressants [61]. The mechanism of drug-induced dystonia is believed to involve alterations in dopamine signaling in the basal ganglia.

Overall, while medications can be effective in treating various conditions, they can also have unintended side effects, including the development of movement disorders. It is essential for healthcare providers to be aware of the potential for DIMDs and to monitor patients closely for any signs of these side effects. Early recognition and management of DIMDs can minimize their impact on patient's quality of life. Other medications that should be remembered that are associated with movement disorders Field [62] include mood stabilizers such as lithium [63].

3.2. *Post-Infectious Movement Disorders*

3.2.1. Post-Streptococcal Movement Disorders

Post-streptococcal movement disorders (PSMDs) are a group of movement disorders that occur following a streptococcal infection, typically caused by *Streptococcus pyogenes*. These disorders are thought to result from an autoimmune response triggered by the infection, leading to inflammation and dysfunction in the basal ganglia, a brain region involved in movement control [64].

One of the most well-known PSMDs is Sydenham's chorea, also known as St. Vitus dance. Sydenham's chorea is characterized by rapid, uncoordinated, involuntary movements that primarily affect the face, hands, and feet [65]. These movements may be accompanied by muscle weakness, emotional lability, and cognitive changes. Sydenham's chorea typically occurs in children and adolescents, often several weeks after a streptococcal infection, such as strep throat or scarlet fever.

Another PSMD is a pediatric autoimmune neuropsychiatric disorder associated with streptococcal infections (PANDAS), which is characterized by the sudden onset of obsessive-compulsive disorder (OCD) or tic disorders following a streptococcal infection. Children with PANDAS may also experience other neuropsychiatric symptoms, such as anxiety, mood swings, and behavioral regression. The exact mechanism of PANDAS is not fully understood but is believed to involve an immune response that targets the basal ganglia, leading to the development of neuropsychiatric symptoms.

Treatment of PSMDs typically involves addressing the underlying streptococcal infection with antibiotics. In some cases, immunomodulatory therapies, such as corticosteroids or intravenous immunoglobulin (IVIG), may suppress the immune response and reduce inflammation in the brain. Symptomatic treatment with medications to help control movement symptoms, such as anticonvulsants or neuroleptics, may also be necessary.

Overall, PSMDs are rare but critical neurological manifestations of streptococcal infections. Early recognition and treatment are crucial to preventing long-term complications and improving affected individuals' outcomes, while well-established methods can be briefly described and appropriately cited.

3.2.2. Other Post-Infectious Movement Disorders

In addition to post-streptococcal movement disorders, other post-infectious movement disorders can occur following various infections. These movement disorders are believed to result from an autoimmune response triggered by the infection, leading to inflammation and dysfunction in the brain regions involved in movement control [66].

One example of a post-infectious movement disorder is acute disseminated encephalomyelitis (ADEM), characterized by widespread brain and spinal cord inflammation following a viral [67] or bacterial infection [68]. ADEM can lead to various neurological symptoms [69], including movement abnormalities such as ataxia (loss of coordination) and tremor [70]. Also, the association of transverse myelitis and Guillain-Barré syndrome [71] was already associated with abnormal movements [72].

Another post-infectious movement disorder is opsoclonus-myoclonus syndrome (OMS), which is characterized by rapid, involuntary eye movements (opsoclonus) and sudden, brief muscle contractions (myoclonus) [73]. OMS is often associated with viral infections, particularly neuroblastoma-associated opsoclonus-myoclonus syndrome (NOMS), which occurs in children with neuroblastoma [74].

Post-infectious cerebellitis is another condition that can lead to movement abnormalities following an infection [75], characterized by inflammation in the cerebellum, a brain region involved in coordination and balance. Post-infectious cerebellitis can lead to symptoms such as ataxia, tremor, and dysarthria [76], like in cases of herpes zoster infection [77] or even in cases of neurosyphilis. Neurosyphilis was already reported presenting with status epilepticus [78] and parkinsonism [79].

The treatment of post-infectious movement disorders depends on the underlying cause and the specific symptoms [80]. Addressing the underlying infection with appropriate antiviral or antibacterial medications is crucial in many cases. Immunomodulatory therapies, such as corticosteroids or intravenous immunoglobulin (IVIG), may also suppress the immune response and reduce inflammation in the brain [81].

3.3. Metabolic Disorders

3.3.1. Wilson Disease

Wilson disease is a rare genetic disorder characterized by the abnormal accumulation of copper in various tissues, particularly the liver and brain. This excess copper can lead to a wide range of symptoms, including hepatic dysfunction, neurological symptoms, and psychiatric manifestations. One of the critical features of Wilson's disease is the development of movement disorders, which can be one of the earliest signs of the disease.

The movement disorders associated with Wilson's disease are primarily dystonia and tremor. Dystonia in Wilson disease typically presents as a focal or segmental dystonia affecting specific body regions such as the hands, feet, or face. This dystonia can lead to abnormal postures or repetitive movements. A tremor in Wilson disease is usually a resting tremor, which occurs when the affected body part is at rest and improves with movement. This tremor is similar to the tremor seen in Parkinson's disease.

The movement disorders in Wilson disease are believed to result from the accumulation of copper in the basal ganglia, a brain region involved in movement control. Copper is usually excreted from the body through bile, but in individuals with Wilson disease, a genetic mutation impairs this process, leading to copper accumulation. The exact mechanism by which copper accumulation leads to movement disorders is not fully understood but is thought to involve disruption of normal neurotransmission in the basal ganglia.

Treatment of Wilson disease involves reducing copper accumulation in the body through chelation therapy, which consists of medications that bind to copper and facilitate its excretion. Zinc supplements may also be used to block copper absorption in the intestines. In cases where there is severe liver damage, liver transplantation may be necessary. Early diagnosis and treatment are crucial to prevent irreversible damage and improve outcomes for individuals with Wilson disease.

3.3.2. Other Metabolic Disorders

In addition to Wilson's disease, several other metabolic disorders can lead to movement disorders [82]. These disorders are often characterized by abnormalities in the metabolism of specific substances, leading to the accumulation of toxic byproducts or the deficiency of essential molecules, which can affect the function of the nervous system and lead to movement abnormalities [83]. Another common metabolic abnormality is hypokalemia, leading to areflexia and weakness, which was rarely reported in cases of primary sjögren syndrome [84].

One example is Huntington's disease, a genetic disorder caused by a mutation in the HTT gene, which accumulates an abnormal form of the huntingtin protein in the brain. Huntington's disease is characterized by a triad of symptoms, including progressive motor dysfunction (chorea, dystonia, and rigidity), cognitive impairment, and psychiatric disturbances [85]. The movement disorders in Huntington's disease are believed to result from dysfunction in the basal ganglia and related brain regions.

Another example is maple syrup urine disease (MSUD), a rare genetic disorder characterized by the body's inability to break down certain amino acids. This leads to the accumulation of toxic byproducts, including branched-chain amino acids, which can affect the nervous system and lead to movement abnormalities, among other symptoms. The movement disorders in MSUD can include tremors, dystonia, and ataxia [86].

Wilson disease, as mentioned earlier, is another example of a metabolic disorder that can lead to movement disorders. Wilson disease is characterized by the abnormal accumulation of copper in various tissues, including the brain, leading to movement abnormalities such as dystonia and tremor. Also, the deficiency of copper can lead to subacute demyelination of the spinal cord and blood dyscrasia [87].

Treatment of metabolic disorders that lead to movement disorders often involves dietary modifications, such as restricting the intake of specific amino acids in MSUD or chelation therapy to remove excess copper in Wilson disease. In some cases, symptomatic treatment with medications to help control movement symptoms may also be necessary. Early diagnosis and treatment are crucial to prevent long-term complications and improve outcomes for individuals with these disorders [88].

3.4. Structural Brain Lesions

Structural brain lesions, such as tumors, strokes, and traumatic brain injuries, can lead to movement disorders through various mechanisms, including electrolyte abnormalities such as hyponatremia [89]. These lesions can disrupt normal brain function and affect areas involved in movement control, leading to a wide range of movement abnormalities.

One common movement disorder associated with structural brain lesions is hemiballismus, characterized by sudden, violent, and involuntary flinging movements of one arm and leg on the same side of the body [90]. Hemiballismus is often caused by damage to the subthalamic nucleus, a deep brain structure involved in movement control, typically due to a stroke or other structural lesion. Interestingly, more studies should be done with the stroke of cortical hand knob area due to the anatomical singularity of this region [91].

Another movement disorder associated with structural brain lesions is hemidystonia, characterized by sustained or intermittent muscle contractions that cause abnormal postures or repetitive movements on one side of the body [92]. Hemidystonia can occur due to damage to the basal ganglia or its connections, often as a result of a stroke or brain injury [93].

Structural brain lesions can also lead to secondary Parkinsonism, which is characterized by symptoms similar to Parkinson's disease, including bradykinesia (slowness of movement), rigidity, tremor, and postural instability. Secondary Parkinsonism can occur due to damage to the nigrostriatal pathway, which is involved in dopamine signaling, as a result of a brain lesion.

Treatment of movement disorders associated with structural brain lesions often involves addressing the underlying cause of the lesion, such as surgery to remove a tumor or medication to manage symptoms of a stroke. In some cases, symptomatic treatment with drugs to help control movement symptoms may also be necessary. Physical therapy and rehabilitation may also be beneficial in improving mobility and function. Early diagnosis and treatment are crucial to prevent long-term complications and improve outcomes for individuals with movement disorders associated with structural brain lesions.

3.4.1. Vascular Lesions

Vascular lesions in the brain, such as strokes and arteriovenous malformations (AVMs), can lead to movement disorders through various mechanisms. These lesions can disrupt normal blood flow in the brain, leading to ischemia (lack of oxygen) or hemorrhage (bleeding), which can damage brain tissue and affect areas involved in movement control.

One of the most common movement disorders associated with vascular lesions is hemiparesis, weakness, or paralysis on one side of the body. Hemiparesis can occur due to damage to the motor cortex or the corticospinal tracts, which control voluntary movement resulting from a stroke or other vascular lesion.

Another movement disorder associated with vascular lesions is ataxia, characterized by a lack of coordination and unsteady gait. Ataxia can occur due to damage to the cerebellum or its connections, often due to a stroke or bleeding in the posterior circulation of the brain [94]. However, phenytoin can also lead to cerebellar atrophy [95]. A joint presentation that can be confounded with ataxia is astasia [96], which was rarely reported with structural brain lesions [97].

Vascular lesions can also lead to secondary Parkinsonism [98], as mentioned earlier, which is characterized by symptoms similar to Parkinson's disease [99]. Secondary Parkinsonism can occur due to damage to the nigrostriatal pathway, which is involved in dopamine signaling, as a result of a vascular lesion.

Treatment of movement disorders associated with vascular lesions often involves addressing the underlying vascular lesion, such as surgery to remove an AVM or medication to manage symptoms of a stroke [100]. Rehabilitation and physical therapy may also be beneficial in improving mobility and function. Early diagnosis and treatment are crucial to prevent long-term complications and improve outcomes for individuals with movement disorders associated with vascular lesions.

3.4.2. Tumors

Brain tumors can lead to movement disorders through various mechanisms, depending on their location and size. Tumors can directly compress or invade brain regions involved in movement control, disrupt normal brain function, or cause increased pressure within the skull, leading to neurological symptoms, including movement abnormalities.

One common movement disorder associated with brain tumors is hemiparesis, which is weakness or paralysis on one side of the body. Hemiparesis can occur due to compression or invasion of the motor cortex or the corticospinal tracts by the tumor, leading to impaired voluntary movement on the affected side.

Another movement disorder associated with brain tumors is ataxia, which is characterized by a lack of coordination and unsteady gait. Ataxia can occur due to compression or invasion of the cerebellum or its connections by the tumor, disrupting normal cerebellar function and coordination of movement.

Brain tumors can also lead to focal dystonia, which is characterized by sustained or intermittent muscle contractions that cause abnormal postures or repetitive movements. Focal dystonia can occur due to compression or invasion of the basal ganglia or its connections by the tumor, leading to abnormal signaling in the motor pathways [101].

Treatment of movement disorders associated with brain tumors often involves surgical resection of the tumor, if possible, to relieve compression and restore normal brain function. Radiation therapy and chemotherapy may also be used to shrink the cancer and reduce symptoms. Rehabilitation and physical therapy may be beneficial in improving mobility and function. Early diagnosis and treatment are crucial to prevent long-term complications and improve outcomes for individuals with movement disorders associated with brain tumors.

3.4.3. Traumatic Brain Injury

Traumatic brain injury (TBI) can lead to movement disorders through various mechanisms, depending on the severity and location of the injury. TBI can result from a blow or jolt to the head or a penetrating head injury that disrupts normal brain function. Movement disorders associated with TBI can range from mild to severe and can be temporary or permanent.

One common movement disorder associated with TBI is spasticity, characterized by increased muscle tone and stiffness, leading to abnormal posture and movement. Spasticity can occur due to damage to the motor pathways in the brain or spinal cord, leading to abnormal signaling to the muscles.

Another movement disorder associated with TBI is chorea, characterized by rapid, involuntary, jerky movements that flow randomly from one body part to another. Chorea can occur due to disruption of the basal ganglia or its connections, leading to abnormal signaling in the motor pathways.

TBI can also lead to ataxia, characterized by a lack of coordination and unsteady gait. Ataxia can occur due to damage to the cerebellum or its connections, leading to impaired balance and coordination of movement.

Treatment of movement disorders associated with TBI often involves rehabilitation and physical therapy to improve mobility and function. Medications may also be used to manage symptoms such as spasticity or chorea. In severe cases, surgery may be necessary to address structural damage or reduce pressure on the brain. Early diagnosis and treatment are crucial to prevent long-term complications and improve outcomes for individuals with movement disorders associated with TBI.

3.4.4. Other Structural Lesions

In addition to tumors, strokes, and traumatic brain injuries, other structural brain lesions can lead to movement disorders. These lesions can include vascular malformations, such as arteriovenous malformations (AVMs) or cavernous malformations, as well as cysts, abscesses, and other abnormal growths in the brain. Especially in cases of cerebral venous sinus thrombosis [102].

Vascular malformations, such as AVMs, are abnormal tangles of blood vessels that can disrupt normal blood flow in the brain and lead to ischemia (lack of oxygen) or hemorrhage (bleeding), which can damage brain tissue and affect areas involved in movement control [103]. AVMs can cause various movement disorders, including hemiparesis, ataxia, and chorea, depending on their location and size.

Cysts and abscesses in the brain can also lead to movement disorders, depending on their location and size [104]. These lesions can cause compression of surrounding brain tissue, leading to symptoms such as weakness, tremors, and dystonia [105]. Noteworthy, these lesions are commonly associated with drug-resistant epilepsy [106].

Other structural lesions, such as brainstem lesions or lesions in the thalamus or basal ganglia, can also lead to movement disorders. These lesions can disrupt normal brain function and affect areas involved in movement control, leading to a wide range of movement abnormalities [107].

Treatment of movement disorders associated with structural brain lesions often involves addressing the underlying cause of the lesion, such as surgery to remove a vascular malformation or cyst. Rehabilitation and physical therapy may also be beneficial in improving mobility and function. Early diagnosis and treatment are crucial to prevent long-term complications and improve outcomes for individuals with movement disorders associated with structural brain lesions.

3.5. Other Causes

3.5.1. Paraneoplastic Syndromes

Paraneoplastic syndromes are a group of rare disorders that occur in some people with cancer. These syndromes are caused by the body's immune system mistakenly attacking normal cells in the nervous system in response to a cancerous tumor elsewhere in the body. Paraneoplastic syndromes can affect various parts of the nervous system, including the brain, spinal cord, peripheral nerves, and muscles, leading to many symptoms, including movement disorders.

One example of a movement disorder associated with paraneoplastic syndromes is opsoclonus-myoclonus syndrome (OMS), which is characterized by rapid, involuntary eye movements (opsoclonus) and sudden, brief muscle contractions (myoclonus). OMS is often associated with neuroblastoma, a type of cancer that occurs in children, although it can also happen with other types of tumors. Another uncommon association is with infectious diseases [108].

Another example is paraneoplastic cerebellar degeneration, characterized by the progressive loss of function in the cerebellum, a brain region involved in coordination and balance. Paraneoplastic cerebellar degeneration can lead to symptoms such as ataxia (lack of coordination), tremor, and dysarthria (difficulty speaking). This syndrome is often associated with ovarian, breast, or lung cancer.

Paraneoplastic syndromes can also lead to other movement disorders, such as chorea (rapid, involuntary movements), dystonia (sustained or intermittent muscle contractions that cause abnormal postures or repetitive movements), and parkinsonism (symptoms similar to Parkinson's disease, such as tremor, bradykinesia, rigidity, and postural instability) [109].

Treatment of paraneoplastic syndromes often involves treating the underlying cancer with surgery, chemotherapy, or radiation therapy. Immunotherapy may also suppress the immune response and reduce inflammation in the nervous system. Symptomatic treatment with medications to help control movement symptoms may also be necessary. Early diagnosis and treatment are crucial to prevent long-term complications and improve outcomes for individuals with paraneoplastic syndromes.

3.5.2. Neurodegenerative Disorders

Neurodegenerative disorders are a group of progressive diseases that primarily affect the neurons in the brain. These disorders are characterized by neurons' gradual degeneration and death, leading to a decline in cognitive function, movement abnormalities, and other neurological symptoms. Several neurodegenerative disorders are associated with movement disorders, including Parkinson's disease, Huntington's disease, and amyotrophic lateral sclerosis (ALS).

Parkinson's disease is a neurodegenerative disorder characterized by the loss of dopamine-producing neurons in the substantia nigra, a region of the brain involved in movement control [110]. This loss of dopamine leads to the motor symptoms of Parkinson's disease, including bradykinesia (slowness of movement), rigidity, tremor, and postural instability. Parkinson's disease can also lead

to non-motor symptoms, such as cognitive impairment, mood disorders, and autonomic dysfunction. In Parkinson's disease, the worsened cognitive function is associated with the impairment of the motor function [111].

Huntington's disease is another neurodegenerative disorder characterized by the progressive degeneration of neurons in the brain, particularly in the basal ganglia. Huntington's disease is caused by a genetic mutation that produces an abnormal form of the huntingtin protein. The movement disorders associated with Huntington's disease, including chorea (rapid, involuntary movements), dystonia (sustained or intermittent muscle contractions that cause abnormal postures or repetitive movements), and bradykinesia, are often preceded by cognitive and psychiatric symptoms.

Amyotrophic lateral sclerosis (ALS), also known as Lou Gehrig's disease, is a neurodegenerative disorder that affects the motor neurons in the brain and spinal cord [112]. ALS leads to progressive muscle weakness and atrophy, eventually affecting the ability to move, speak, swallow, and breathe. While ALS primarily affects the motor neurons, some individuals with ALS may also develop movement abnormalities such as spasticity, tremor, or dystonia. However, this should be a differential diagnosis excluding secondary causes such as radiculopathies [113].

Treatment of movement disorders associated with neurodegenerative disorders often involves symptomatic management to improve quality of life. Medications may be used to help control movement symptoms, such as levodopa for Parkinson's disease or tetrabenazine for Huntington's disease. Physical therapy, occupational therapy, and speech therapy may also be beneficial in improving mobility and function. While there is currently no cure for neurodegenerative disorders, ongoing research aims to develop new treatments to slow or stop disease progression. Early diagnosis and management are crucial to optimize outcomes for individuals with these disorders [114].

3.5.3. Inherited Metabolic Disorders

Inherited metabolic disorders are genetic conditions that result in abnormalities in the body's metabolism. These disorders are caused by defects in enzymes or other proteins involved in metabolism, leading to the accumulation of toxic substances or the deficiency of essential molecules. Inherited metabolic disorders can affect various organs and systems in the body, including the brain, and can lead to a wide range of symptoms, including movement disorders.

One example of an inherited metabolic disorder that can lead to movement disorders is Wilson's disease, which was mentioned earlier. Wilson disease is characterized by the abnormal accumulation of copper in various tissues, including the brain, leading to movement abnormalities such as dystonia and tremor.

Another example is maple syrup urine disease (MSUD), also mentioned earlier. MSUD is characterized by the body's inability to break down certain amino acids, accumulating toxic byproducts that can affect the nervous system and lead to movement abnormalities, among other symptoms. Also, glutaric aciduria type 1 can lead to movement disorders [115].

Other inherited metabolic disorders that can lead to movement disorders include organic acidemias, such as propionic acidemia and methylmalonic acidemia, characterized by the accumulation of organic acids in the body [116]. These organic acids can affect the nervous system and lead to movement abnormalities, among other symptoms. Also, non-inherited metabolic disorders due to electrolyte abnormalities can cause movement disorders [117].

Treatment of inherited metabolic disorders often involves dietary modifications, such as restricting the intake of specific amino acids or organic acids, to prevent the accumulation of toxic substances [118]. Supplementation with vitamins or other nutrients may sometimes be necessary to correct deficiencies. Early diagnosis and treatment are crucial to avoid long-term complications and improve outcomes for individuals with inherited metabolic disorders.

3.5.4. Toxic Exposures

Toxic exposures can lead to movement disorders through various mechanisms, depending on the type of toxin involved and the extent of exposure. Toxins can directly damage neurons in the

brain or peripheral nervous system, disrupt neurotransmitter function, or alter the balance of ions and other molecules involved in neuronal signaling, leading to movement abnormalities.

One example of a toxin that can lead to movement disorders is manganese, which can cause a condition known as manganism. Manganism is characterized by symptoms similar to Parkinson's disease, including tremor, rigidity, and bradykinesia, due to the accumulation of manganese in the basal ganglia, a brain region involved in movement control.

Another example is carbon monoxide poisoning, which can lead to a variety of neurological symptoms, including movement abnormalities. Carbon monoxide poisoning can disrupt oxygen delivery to the brain, leading to brain damage and movement disorders such as chorea, dystonia, or parkinsonism.

Exposure to certain pesticides, such as organophosphates or paraquat, can also lead to movement disorders. These pesticides can inhibit acetylcholinesterase, an enzyme that breaks down the neurotransmitter acetylcholine, leading to excessive acetylcholine signaling and neuronal dysfunction.

Other toxins that can lead to movement disorders include lead, mercury, and certain drugs of abuse, such as cocaine and amphetamines. These toxins can affect various neurotransmitter systems in the brain, leading to abnormal movement patterns.

Treatment of movement disorders associated with toxic exposures often involves removing the individual from the source of exposure and providing supportive care. In some cases, chelation therapy may remove toxic metals from the body. Physical and occupational therapy may also be beneficial in improving mobility and function. Early diagnosis and treatment are crucial to prevent long-term complications and improve outcomes for individuals with movement disorders associated with toxic exposures.

4. Diagnostic Approach to Secondary Movement Disorders

4.1. History and Physical Examination

The history and physical examination are crucial in evaluating patients with movement disorders. They provide valuable information that can help determine the underlying cause of the movement disorder and guide further diagnostic and management strategies.

During the history-taking process, the clinician should gather detailed information about the onset and progression of the movement disorder, including any associated symptoms or triggering factors. It is essential to inquire about the presence of any family history of movement disorders or neurological conditions, as well as any past medical history, including previous infections, trauma, or exposure to toxins.

A thorough physical examination should be performed, focusing on the neurological examination. This should include an assessment of mental status, cranial nerve function, motor function (including strength, tone, and coordination), sensory function, and reflexes [119]. Specific attention should be paid to abnormal movements, such as tremors, dystonia, chorea, or myoclonus, and any signs of parkinsonism, such as bradykinesia, rigidity, and postural instability [61].

Additional testing may be indicated based on the history and physical examination findings. This may include laboratory tests, such as blood or genetic testing, imaging studies, MRI or CT scans, and electrophysiological studies, such as electromyography (EMG) or nerve conduction studies.

A detailed history and thorough physical examination are essential in evaluating patients with movement disorders. They provide valuable information that can help guide further evaluation and management, ultimately leading to better patient outcomes.

4.2. Laboratory Investigations

Laboratory investigations are essential to the diagnostic workup for movement disorders, helping to identify underlying causes and guide treatment decisions. Blood tests are often the first step and can include a complete blood count (CBC), metabolic panel, and tests for inflammatory markers [120]. These tests can reveal abnormalities such as infections, metabolic imbalances, or signs

of autoimmune disorders, all of which can contribute to movement disorders. Interestingly, valproate is associated with dose-dependent pancytopenia [121], which can provide support for the diagnosis of valproate-induced movement disorders in these individuals.

Genetic testing is crucial for suspected hereditary movement disorders, providing insight into the genetic mutations responsible. For example, Huntington's disease, a progressive neurodegenerative disorder, is diagnosed through genetic testing for the HTT gene mutation. Other genetic tests can identify mutations associated with dystonias, ataxias, and other inherited movement disorders.

In cases of suspected toxic exposure, such as to heavy metals or certain medications, toxicology screening can confirm the presence of toxins in the body [122]. Additionally, cerebrospinal fluid (CSF) analysis, obtained through a lumbar puncture, can provide valuable information about infections, inflammation, or other neurological conditions affecting the brain and spinal cord [123]. These laboratory investigations, imaging studies, and clinical assessments provide a comprehensive diagnostic approach for patients with movement disorders.

4.3. Neuroimaging Studies

Neuroimaging studies are essential in evaluating patients with movement disorders, helping to identify structural and functional abnormalities in the brain that may contribute to the disorder [124]. Several neuroimaging modalities are commonly used in this context, including magnetic resonance imaging (MRI), computed tomography (CT), positron emission tomography (PET), and single-photon emission computed tomography (SPECT).

MRI is often the first-line imaging modality for patients with movement disorders, as it provides detailed images of the brain structures. MRI can help identify structural abnormalities such as tumors, strokes, or vascular malformations that may be causing or contributing to the movement disorder [125]. Additionally, advanced MRI techniques, such as diffusion-weighted imaging (DWI) and magnetic resonance spectroscopy (MRS), can provide information about tissue integrity and metabolic abnormalities in the brain. Another neuroimaging useful for supporting the diagnosis of Parkinson's disease is the cardiac 123I-metaiodobenzylguanidine (MIBG) scintigraphy [126].

CT scans may also be used to evaluate patients with movement disorders, particularly in emergencies or when MRI is not feasible. CT scans can help identify acute structural abnormalities, such as hemorrhage or large lesions, that may require immediate intervention.

Functional neuroimaging techniques like PET and SPECT can provide information about brain function in patients with movement disorders [127]. These techniques can help identify areas of abnormal brain activity or neurotransmitter function that may be associated with the movement disorder. For example, PET scans can assess dopamine activity in the brain, which is essential in conditions such as Parkinson's disease [128]. Neuroimaging studies are crucial in evaluating patients with movement disorders, helping identify underlying structural and functional abnormalities, and guiding treatment decisions [129].

4.4. Electrophysiological Studies

Electrophysiological studies are critical diagnostic tools used to evaluate patients with movement disorders. These studies assess the electrical activity of muscles and nerves, providing valuable information about the function of the neuromuscular system. Several electrophysiological studies may be used to evaluate movement disorders, including electromyography (EMG), nerve conduction studies (NCS), and evoked potentials.

EMG is a technique used to assess the electrical activity of muscles. It involves the insertion of a needle electrode into the muscle to record its electrical activity at rest and during contraction. EMG can help identify abnormal patterns of muscle activity, such as fibrillation potentials or fasciculations, which may indicate underlying nerve or muscle dysfunction.

NCS is used to assess the function of peripheral nerves by measuring the speed and strength of nerve signals. NCS involves the placement of surface electrodes on the skin overlying the nerve and delivering tiny electrical impulses to stimulate the nerve. By measuring the speed and strength of the

nerve's response, NCS can help identify abnormalities in nerve conduction, such as demyelination or axonal damage.

Evoked potentials are used to assess the function of the central nervous system by measuring the electrical activity generated in response to specific stimuli, such as visual or auditory stimuli [130]. Evoked potentials can help identify abnormalities in the transmission of sensory information along the central nervous system's pathways, which may indicate conditions such as multiple sclerosis or other demyelinating disorders, especially in cases of tumefactive multiple sclerosis that are recurrent [131].

Overall, electrophysiological studies are valuable diagnostic tools in evaluating movement disorders, providing important information about the function of the neuromuscular system, and helping to guide treatment decisions.

4.5. Other Diagnostic Modalities

In addition to laboratory investigations, neuroimaging studies, and electrophysiological studies, several other diagnostic modalities may be used to evaluate patients with movement disorders. These modalities can provide valuable information about the underlying cause of the movement disorder and help guide treatment decisions.

One such modality is cerebrospinal fluid (CSF) analysis, which involves collecting and analyzing CSF obtained through a lumbar puncture. CSF analysis can help identify signs of infection, inflammation, or other abnormalities in the central nervous system that may contribute to the movement disorder.

Genetic testing is another important diagnostic tool for suspected hereditary movement disorders. Genetic testing can identify specific genetic mutations associated with movement disorders, providing valuable information about disease progression, inheritance patterns, and potential treatment options.

Neuropsychological testing may also assess cognitive function in patients with movement disorders, particularly those that affect the basal ganglia or other brain regions involved in cognitive processing. Neuropsychological testing can help identify cognitive deficits associated with the movement disorder and guide treatment decisions.

Finally, video recording of movement disorders can be helpful in diagnostic evaluation, particularly for disorders with characteristic movement patterns, such as tremors or dystonia. Video recording can provide valuable information about the movement disorder's frequency, severity, and characteristics, helping to guide treatment decisions.

A comprehensive diagnostic approach incorporating multiple modalities is often necessary to accurately diagnose and effectively manage movement disorders. Each modality provides unique information that contributes to a thorough understanding of the underlying cause of the movement disorder and helps guide treatment decisions.

5. Management of Secondary Movement Disorders

5.1. General Principles

Managing secondary movement disorders involves a multifaceted approach to address the underlying cause, manage symptoms, and improve the patient's quality of life. Treatment strategies may vary depending on the specific etiology of the movement disorder. In cases where the movement disorder is caused by medication, adjusting the dosage or switching to an alternative medication may be sufficient to alleviate symptoms. However, if the movement disorder is due to a structural brain lesion, such as a tumor or stroke, more aggressive interventions may be necessary, such as surgery or radiation therapy.

In addition to treating the underlying cause, symptomatic management is often essential to the treatment plan. Medications may be prescribed to help control symptoms such as tremor, dystonia, or bradykinesia. Physical therapy and occupational therapy can also be beneficial in improving

mobility, coordination, and overall function. Assistive devices, such as braces or walking aids, may be recommended to help with mobility issues.

Furthermore, psychological support and counseling may be beneficial for patients with secondary movement disorders, particularly if the disorder has a significant impact on their quality of life. Support groups and counseling can provide emotional support, education, and coping strategies for dealing with the challenges of living with a movement disorder. Managing secondary movement disorders requires a comprehensive and individualized approach that addresses the underlying cause, manages symptoms, and supports the patient's overall well-being.

5.2. Pharmacological Management

Pharmacological management is a cornerstone in treating secondary movement disorders, aiming to alleviate symptoms, improve function, and enhance the quality of life for affected individuals. The choice of medication depends on the underlying cause of the movement disorder and the specific symptoms experienced by the patient. Dopaminergic agents, such as levodopa-carbidopa, are commonly used to manage movement disorders associated with dopamine deficiency, such as parkinsonism. These medications can help alleviate symptoms such as bradykinesia, rigidity, and tremor by increasing dopamine levels in the brain.

Anticholinergic agents, such as trihexyphenidyl and benztropine, may sometimes reduce tremors and dystonia. These medications work by blocking the action of acetylcholine, a neurotransmitter that can contribute to abnormal muscle contractions. Anticholinergic agents are often used in the treatment of Parkinson's disease and other movement disorders to help improve motor symptoms.

Benzodiazepines, such as clonazepam, may be prescribed to reduce anxiety and muscle spasms in some cases of movement disorders. These medications enhance the effects of gamma-aminobutyric acid (GABA), a neurotransmitter that inhibits the activity of excitatory neurons. Benzodiazepines can help reduce excessive muscle contractions and improve muscle relaxation in conditions such as myoclonus or tremor [132].

Other pharmacological options for the management of secondary movement disorders include antiepileptic drugs, such as gabapentin [133] and topiramate [134], which may be used to manage tremors or other movement abnormalities [135], particularly if they are associated with epilepsy or other neurological conditions [136]. Botulinum toxin injections are another treatment option for focal dystonias, such as cervical dystonia or blepharospasm. Botulinum toxin works by blocking the release of acetylcholine, thereby reducing muscle contractions and alleviating symptoms [137].

In conclusion, pharmacological management is crucial in treating secondary movement disorders, helping alleviate symptoms, and improving the quality of life for affected individuals. The choice of medication depends on the specific symptoms and underlying cause of the movement disorder, and treatment should be tailored to the individual patient. Close monitoring by a healthcare provider is essential to adjust medications as needed and minimize side effects, ensuring optimal outcomes for patients with secondary movement disorders.

5.3. Non-Pharmacological Management

Non-pharmacological management strategies are integral components of the comprehensive care plan for individuals with secondary movement disorders. These approaches aim to enhance quality of life, improve functional abilities, and alleviate symptoms through non-drug-based interventions. Physical therapy is a cornerstone of non-pharmacological management, focusing on improving mobility, strength, and coordination. Therapeutic exercises, stretching, and manual techniques can help reduce muscle stiffness, improve range of motion, and enhance overall physical function. Physical therapists tailor treatment plans to each individual's needs, addressing movement impairments and promoting optimal function.

Occupational therapy is another critical non-pharmacological intervention for secondary movement disorders, focusing on enhancing the individual's ability to perform activities of daily living (ADLs) and participate in meaningful activities. Occupational therapists work with individuals

to develop strategies and use adaptive devices that facilitate independence in daily tasks such as dressing, grooming, and eating. By addressing functional limitations and promoting independence, occupational therapy is crucial in improving individuals' overall quality of life with movement disorders.

Speech therapy is often recommended for individuals with movement disorders that affect speech and swallowing. Speech therapists assess and treat speech and swallowing difficulties, providing exercises and techniques to improve speech clarity, swallowing function, and overall communication skills. Speech therapy can help individuals overcome communication challenges and improve their ability to participate in social interactions, enhancing their quality of life.

5.4. Surgical Management

Surgical management plays a crucial role in treating specific secondary movement disorders [138], mainly when conservative treatments have been ineffective in controlling symptoms or when the underlying cause of the movement disorder can be directly addressed through surgery [139]. One of the most common surgical interventions for movement disorders is deep brain stimulation (DBS). DBS involves the implantation of electrodes into specific brain areas that control movement, such as the globus pallidus or subthalamic nucleus [140]. These electrodes deliver electrical impulses that help regulate abnormal brain activity associated with movement disorders, such as Parkinson's disease, essential tremor, and dystonia. DBS can significantly improve motor symptoms, reduce medication requirements, and enhance the quality of life for individuals with movement disorders.

Another surgical option for certain movement disorders is lesioning surgery, which involves creating a controlled lesion in specific brain areas to disrupt abnormal neural pathways [141]. Lesioning surgery is typically reserved for individuals with tremor-dominant Parkinson's disease or essential tremors who have not responded to medication or are not suitable candidates for DBS. Lesioning procedures, such as thalamotomy or pallidotomy, can reduce tremors and improve motor function in select patients [142].

In some cases, surgical management of secondary movement disorders may involve removing or treating an underlying structural lesion, such as a tumor or arteriovenous malformation (AVM), causing or contributing to the movement disorder. Surgical resection or embolization of the lesion can help alleviate symptoms and improve overall neurological function. Additionally, neurosurgical procedures may be used to treat certain types of dystonia or spasticity, such as selective peripheral denervation or intrathecal baclofen pump implantation, to improve motor function and quality of life. However, baclofen was also associated with the development of abnormal movements [143].

Overall, surgical management of secondary movement disorders requires careful consideration of the underlying cause, the specific symptoms, and the individual patient's goals and preferences. Surgical interventions must be performed by experienced neurosurgeons in specialized centers with expertise in managing movement disorders. While surgical management can offer significant benefits for select patients, it also carries risks, and the potential benefits and risks should be carefully weighed and discussed with the patient before proceeding with surgery.

6. Emerging Concepts and Future Directions

6.1. Genetics of Secondary Movement Disorders Subsection

Secondary movement disorders can have genetic components, but they are typically not considered primarily genetic disorders. Instead, genetic factors may interact with other environmental or acquired factors to contribute to developing secondary movement disorders. For example, specific genetic mutations may increase susceptibility to medication-induced movement disorders or predispose individuals to develop movement disorders in response to specific environmental triggers.

In some cases, secondary movement disorders may be caused by genetic conditions that affect multiple organ systems, including the nervous system. For example, Wilson disease, an inherited disorder of copper metabolism, can lead to movement disorders such as dystonia, tremor, and

parkinsonism. Similarly, certain inherited metabolic disorders, such as maple syrup urine disease or organic acidemias, can cause movement disorders due to metabolic dysfunction affecting the brain.

Genetic testing may be recommended in cases where there is suspicion of an underlying genetic cause of a secondary movement disorder. Genetic testing can help identify specific genetic mutations associated with movement disorders and may provide valuable information about disease progression, inheritance patterns, and potential treatment options. However, genetic testing is typically not the first step in evaluating secondary movement disorders. It is usually reserved for cases with strong clinical suspicion of an underlying genetic cause.

Overall, while genetic factors can play a role in the development of secondary movement disorders, they are often just one piece of the puzzle. A comprehensive evaluation considering various environmental, genetic, and acquired factors is essential for diagnosing and managing secondary movement disorders.

6.2. Biomarkers in Secondary Movement Disorders

Biomarkers are measurable indicators that can provide information about normal biological processes, pathogenic processes, or responses to therapeutic interventions. In the context of secondary movement disorders, biomarkers can play a crucial role in diagnosis, prognosis, and monitoring of disease progression. Several types of biomarkers have been studied for secondary movement disorders, including imaging, biochemical, and genetic biomarkers.

Imaging biomarkers, such as magnetic resonance imaging (MRI) or positron emission tomography (PET) scans, can provide valuable information about structural and functional changes in the brain that may be associated with movement disorders. For example, imaging biomarkers can help identify specific brain regions affected by neurodegenerative diseases or detect structural abnormalities such as tumors or vascular lesions that may be causing secondary movement disorders.

Biochemical biomarkers, such as specific proteins or metabolite levels in the blood or cerebrospinal fluid (CSF), can provide insights into the underlying pathogenic mechanisms of movement disorders. For example, biomarkers of neuroinflammation or oxidative stress may be elevated in individuals with movement disorders associated with neurodegenerative diseases.

Genetic biomarkers, such as specific genetic mutations associated with movement disorders, can help identify individuals at risk for developing certain movement disorders or predict disease progression. For example, genetic testing for mutations in the HTT gene can confirm a diagnosis of Huntington's disease, while genetic testing for mutations in the ATP7B gene can diagnose Wilson's disease.

Overall, biomarkers have the potential to revolutionize the diagnosis and management of secondary movement disorders by providing objective measures of disease progression and treatment response. However, further research is needed to validate and standardize biomarkers for clinical use in this context.

6.3. Novel Therapeutic Approaches

Novel therapeutic approaches for secondary movement disorders are continuously evolving, driven by advances in neuroscience and technology. One emerging strategy is focused ultrasound (FUS), which uses ultrasound waves to target specific brain regions and modulate neural activity. FUS has shown promise in treating essential tremor and Parkinson's disease by providing non-invasive, targeted treatment with fewer side effects compared to traditional surgical interventions. Ongoing research is exploring the potential of FUS in other movement disorders, such as dystonia and tremor associated with multiple sclerosis.

Another promising approach is using neuromodulation devices, such as responsive neurostimulation systems (RNS) and closed-loop deep brain stimulation (DBS). These devices monitor brain activity in real time and deliver targeted stimulation to modulate abnormal neural circuits. Closed-loop DBS, in particular, has shown promising results in improving motor symptoms and reducing medication requirements in Parkinson's disease. Future developments in

neuromodulation technology may enhance its effectiveness and expand its applications in treating movement disorders.

Advances in pharmacotherapy are also contributing to novel therapeutic approaches for secondary movement disorders [144]. For example, targeted therapies that modulate specific neurotransmitter systems, such as glutamate or adenosine, are being investigated for their potential in treating movement disorders. Additionally, repurposing existing medications, such as antiepileptic drugs or neuroprotective agents, for use in movement disorders is an active area of research, especially in individuals presenting with abnormal movements associated with epilepsy [145]. These approaches aim to improve symptom control and disease modification while minimizing the side effects of traditional pharmacological treatments.

The field of movement disorders is rapidly evolving, with novel therapeutic approaches offering new hope for individuals affected by these conditions. Collaborative efforts between researchers, clinicians, and industry partners are essential to advance these therapies from the laboratory to clinical practice, ultimately improving outcomes and quality of life for patients with secondary movement disorders.

6.4. Predict factors for DIP

Drug-induced parkinsonism (DIP) is a form of secondary parkinsonism that is caused by the use of certain medications, most commonly neuroleptic or antipsychotic drugs [146]. Several factors can influence the development of DIP, including the type and dose of the medication, the duration of treatment, and individual susceptibility factors [147]. Understanding these factors is essential for preventing and managing DIP effectively.

One of the primary factors influencing the development of DIP is the type of medication and its pharmacological properties. Neuroleptic or antipsychotic drugs, particularly first-generation agents such as haloperidol and chlorpromazine, are more strongly associated with DIP compared to second-generation agents like risperidone or olanzapine. The potency of the medication also plays a role, with higher-potency agents more likely to cause DIP. Additionally, the duration of treatment is essential, as more prolonged exposure to the offending medication increases the risk of developing DIP [148].

Individual susceptibility factors can also predispose specific individuals to develop DIP. Advanced age is a significant risk factor, as older adults may be more sensitive to the extrapyramidal side effects of medications. Other factors such as female gender, pre-existing movement disorders (e.g., essential tremor), and genetic predisposition (e.g., variations in the dopamine transporter gene) have also been identified as potential risk factors for DIP. Additionally, concomitant use of other medications that affect dopamine function, such as antiemetics or calcium channel blockers [149], can increase the risk of DIP.

Management of DIP involves discontinuing the offending medication if possible and switching to a less likely culprit, such as a second-generation antipsychotic. In some cases, reducing the dose of the offending drug or adding a dopamine receptor agonist may help alleviate symptoms. Physical and occupational therapy can also improve motor function and quality of life for individuals with DIP. Identifying and addressing the factors contributing to DIP is essential for optimizing treatment outcomes and minimizing the impact of this medication-induced movement disorder [150].

6.5. Patient-Centered Care and Quality of Life Issues

Patient-centered care is essential in managing secondary movement disorders, focusing on the patient's needs, preferences, and values [151]. This approach emphasizes shared decision-making between the healthcare provider and the patient, considering the patient's goals and concerns regarding their condition. In the context of movement disorders, patient-centered care involves addressing the physical symptoms and considering the disorder's impact on the patient's quality of life.

Quality of life issues are central to caring for patients with movement disorders, as these conditions can significantly impact daily functioning, independence, and emotional well-being.

Movement disorders can affect various aspects of life, including mobility, communication, social interactions, and mental health. Therefore, healthcare providers must assess and address these issues to improve patients' overall quality of life [152].

Patient-centered care for individuals with movement disorders involves a multidisciplinary approach, with healthcare providers from different specialties working together to address the patient's diverse needs. This may include neurologists, physical therapists, occupational therapists, speech therapists, psychologists, and social workers. By collaborating as a team, healthcare providers can develop comprehensive care plans addressing physical symptoms and the emotional, social, and practical aspects of living with a movement disorder [153].

In conclusion, patient-centered care and attention to quality-of-life issues are essential in managing secondary movement disorders. By focusing on the individual needs and preferences of each patient, healthcare providers can improve treatment outcomes, enhance patient satisfaction, and ultimately improve the overall quality of life for individuals living with these challenging conditions.

7. Conclusion

In conclusion, secondary movement disorders represent a diverse group of conditions with various underlying causes, including medication side effects, structural brain lesions, metabolic disorders, and neurodegenerative diseases. Managing these disorders requires a comprehensive approach considering the underlying cause, symptoms, and impact on the patient's quality of life. Pharmacological management plays a crucial role in symptom control, with medications such as dopaminergic agents, anticholinergics, and benzodiazepines commonly used to manage motor symptoms. Non-pharmacological approaches, including physical, occupational, and speech therapy, are also crucial in improving affected individuals' function and quality of life. Surgical interventions, such as deep brain stimulation and lesioning procedures, may be considered in select cases. Biomarkers and novel therapeutic approaches, such as gene therapy and neuromodulation techniques, promise future advancements. Patient-centered care, focusing on individual needs and preferences, is essential in optimizing treatment outcomes and improving the quality of life for patients with secondary movement disorders. Collaborative efforts between healthcare providers, researchers, and patients are crucial to advancing our understanding and managing these complex disorders.

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