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NEUROLOGY OF SYSTEMIC DISEASES (J BILLER, SECTION EDITOR)

Movement Disorders in Metabolic Disorders

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Abstract

Purpose of Review We provide a review of the movement disorders that complicate selected metabolic disorders, including the abnormal movements that may appear during or after their treatment.

Recent Findings Movement disorders may be underrecognized when arising in the context of a broad range of metabolic disorders.

Summary Abnormal movements may occur as the initial manifestation of a systemic disease, at any time during its course, or as a result of the medical interventions required for its management. Ascertaining movement phenomenology in acute and subacute presentations may assist in the determination of the specific underlying metabolic disorder. The management of movement disorders associated with metabolic disorders depends on the underlying pathophysiology.

Keywords Movement disorders · Abnormal movements · Metabolic disorders · Electrolytes · Internal medicine

Introduction

Movement disorders such as parkinsonism, tremor, dystonia, chorea, and myoclonus most often arise in several neurodegenerative or structural diseases of the basal ganglia [1]. They may also be part of the clinical manifestations of systemic metabolic disorders, as the initial feature, complicating its course, or as a result of the corrective treatment [2•]. These metabolic disorders may also be associated with neurological manifestations that help the clinician suspect the nature of the problem, such as disorders of consciousness, headache, and seizures [3]. The metabolic origin can also be suspected when movement abnormalities appear in emergency settings or in intensive care units [4].

Some common metabolic disorders, such as organ failure (particularly liver or renal insufficiency), endocrinological diseases (e.g., hyperglycemia), and electrolyte disturbances, are frequently present with neurological dysfunction [5–7]. The pathophysiology of these movement disorders is complex and insufficiently understood [6]. Movement disorders may be the key abnormality, such as hemichorea in non-ketotic hyperglycemia, or important component of a major syndrome, such as the myoclonus of renal insufficiency or the asterixis of hepatic encephalopathy [5–7]. Emphasizing the role of the neurologist, the clinical recognition of certain movements may serve to guide the proper diagnostic metabolic workup. Treatment of the abnormal movements in these cases usually relies on the correction of the underlying metabolic disorders.

In this article, we comprehensively reviewed the most common metabolic disorders whose clinical spectrum includes movement disorders and those arising in the context of the correction of the metabolic derangements. Abnormal movements associated with selected metabolic disorders are highlighted in Table 1.

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Table 1 Movement disorders and selected examination signs in metabolic disorders

	Metabolic disorders		
Movement disorders	Common	Uncommon, but typical	Uncommon
Parkinsonism		Hypoparathyroidism (with basal ganglia calcification)	Hypothyroidism Hepatic failure Hyperparathyroidism Renal failure
Chorea		Polycythemia	Anoxic encephalopathy Hyperthyroidism Hypoglycemia Hypoparathyroidism Hepatic failure Renal failure Hypernatremia Hypomagnesemia
Hemichorea/hemiballism		Hyperglycemia	Polycythemia
Myoclonus	Hepatic failure Renal failure Hypernatremia Anoxic encephalopathy		Hypoparathyroidism Hypoglycemia Hypercalcemia Alkalosis Hypomagnesemia
Tremor	Hyperthyroidism Hepatic failure Hypernatremia		Anoxic encephalopathy Alkalosis Hypomagnesemia
Dystonia			Hyperthyroidism Hepatic failure Hypoparathyroidism Anoxic encephalopathy
Ballism			Hyperthyroidism
Asterixis	Hepatic failure Renal failure		Acidosis
Ataxia			Renal failure Hepatic failure
Paroxysmal dyskinesias			Hypoglycemia
Orofacial dyskinesias		Hepatic failure	Polycythemia (vera and idiopathic)
Restless legs syndrome	Chronic renal failure Iron deficiency		
Tics			Anoxic encephalopathy
Tetany	Hypocalcemia		Hypomagnesemia Alkalosis
Trousseau's sign[a]	Hypocalcemia Hypomagnesemia		

[a] See video—segment 2

Endocrinological Diseases

Thyroid Diseases

Hypothyroidism

Hypothyroidism and parkinsonism have many overlapping symptoms such as fatigue, depression, constipation, slowed movements, and gait impairment. Thus, hypothyroidism is in the differential diagnosis for subtle parkinsonian features, particularly when associated with other signs such as brittle hair and temperature intolerance. Serum levels of thyroid hormones should always be requested in these cases. Of note, the use of levodopa may decrease TSH levels without affecting thyroid function [8]. In addition, Hashimoto's thyroiditis, as well as Hashimoto's encephalopathy, may present with chorea and myoclonus [9]. An encephalopathic state may not be necessary for chorea or myoclonus to arise in the

context of Hashimoto's thyroiditis, and corticosteroid response may be absent. This has rendered inaccurate the term, "steroid-responsive encephalopathy associated with autoimmune thyroiditis" [10].

Hyperthyroidism

Hyperthyroidism may be associated with a range of movement disorders including chorea, ballism, athethosis, and dystonia, probably because thyroid hormones may affect the dopaminergic pathways [11]. Tremor is the most common movement disorder observed in hyperthyroidism, and it may occur in isolation or associated with systemic features, such as tachycardia and sweating [12]. This is a postural and action tremor, affecting the upper limbs. Rare presentations include cervical dystonia, ballism, myoclonus, ataxia, and stiff-person syndrome [10, 11]. In the latter, the endocrinopathy is autoimmune in nature, due to antibodies against glutamic acid decarboxylase (anti-GAD) [13]. The presence of hyperthyroidism in Parkinson's disease and other parkinsonisms precludes tremor control until the thyroid disease is identified and treated [12, 14]. Dystonia (writer's cramp) and chorea have been associated with Graves' disease [11, 15]. Chorea induced by thyroid hormones replacement has also been occasionally described [16]. Symptoms tend to respond to beta-blockers and attenuate after correction of elevated thyroid levels.

Parathyroid Disease

Hypoparathyroidism

Hypocalcemia related to idiopathic hypoparathyroidism may cause parkinsonism, dystonia, myoclonus, and chorea (video—segment 1) [17]. Rare cases of paroxysmal chorea may be kinesigenic [18]. An important clue to the presence of hypoparathyroidism is the presence of calcifications in the basal ganglia, whose pattern is similar to genetic causes of Fahr's syndrome [19] (Fig. 1a–c).

Parkinsonism associated with hypoparathyroidism-related calcification of the basal ganglia usually does not respond to levodopa therapy, although some reports have suggested some therapeutic effect [20]. A pathophysiological mechanism may be direct dopaminergic dysfunction by hypoparathyroidism [17]. Parkinsonism may also occur as late complication after thyroidectomy [21].

In pseudohypoparathyroidism, PTH levels are normal, but a biological insensitivity to PTH occurs, which leads to hypocalcemia. In general, it may also be associated with parkinsonism and calcification of the basal ganglia but also to paroxysmal exercise-induced dystonia [22, 23].

Hyperparathyroidism

While the association of hyperparathyroidism with parkinsonism may be coincidental in some cases, parkinsonian features related to hyperparathyroidism may improve with correction of calcium and PTH levels or removal of parathyroid adenomas to a greater extent than dopaminergic medications [24]. Hyperparathyroidism may also worsen pre-existent parkinsonism [25].

Metabolic and Electrolyte Disorders

Electrolyte Disorders

Electrolytic disorders and acid-base disturbances may present with several neurological symptoms, which may affect the central and peripheral nervous systems. Besides encephalopathy, electrolyte and acid-base disturbances may cause different subtypes of movement disorders [3].

Osmotic demyelination syndromes, which include pontine and extrapontine myelinolysis, may affect several areas of the central nervous system as a result of fast correction of hyponatremia, liver disease, prolonged parenteral nutrition, alcohol abuse, and malnutrition [26]. Myelinolysis, particularly extrapontine, with basal ganglia involvement, is usually comprised by parkinsonism, chorea, dystonia, or myoclonus, along with neuropsychiatric symptoms [27–29]. The corresponding acute-onset parkinsonism may respond to levodopa, but most patients have residual deficits. Similarly, when the phenotype is chorea or athetosis, patients are often refractory to therapy [27]. This complication is suggested by neuroimaging demonstration of confluent areas of brainstem and basal ganglia T2-weighted and FLAIR hyperintensity (Fig. 1d–f).

Electrolyte disorders may present with a range of movement disorders (Table 1) [30]. Correction of the underlying electrolyte derangement reverses the associated neurological deficits. Disorders of potassium may not cause movement disorders but can induce severe weakness (technically, a loss of movement). Metabolic acidosis is suspected by the characteristic brain MRI abnormality in the basal ganglia: the fork sign (Fig. 2a) [31].

Glucose Metabolism

Chorea induced by non-ketotic hyperglycemia is associated with high serum glucose levels. After correction of the hyperglycemia, the improvement may occur weeks or months later, but in some cases, chorea may persist. The classic presentation is hemiballismus, which is a severe form of hemichorea (video—segment 3) [5, 32]. The corresponding brain MRI shows hyperintense signal in the striatum, which may also persist after the correction of the metabolic changes or even after

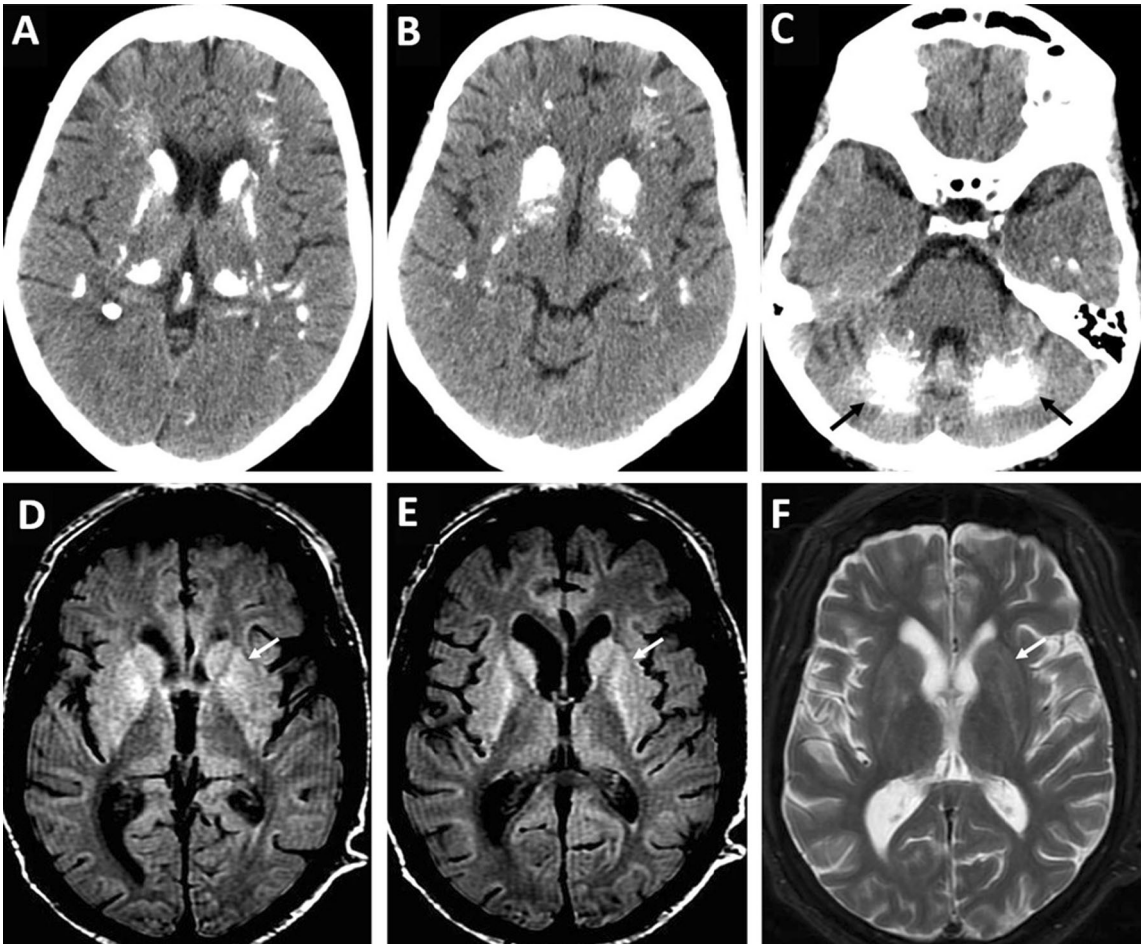

Fig. 1 Neuroimaging of selected metabolic disorders (1/2). Diffuse bilateral calcification in the basal ganglia, thalamus (**a**, **b**), and dentate nucleus (**c**) on head CT in patient with primary hypoparathyroidism. Courtesy of Dr. Miryam Carecchio, Department of Neuroscience, University of Padua, Padua, Italy. Extrapontine myelinolysis. Axial FLAIR (**d**, **e**) and T2-weighted (**f**) brain MRI sequencing shows symmetrical hyperintense signal in basal ganglia and thalamus in patient with extrapontine myelinolysis brought on by fast correction of hyponatremia

the clinical recovery (Fig. 2b) [33]. The pathophysiology of this condition is not yet completely understood, but petechial microhemorrhage has been documented [34]. Also, hemichorea-hemiballismus may be associated with increased $GABA_B$ receptor-mediated activity in the motor cortex [35]. Although correction of glucose levels often suffices, some patients may require dopamine blocking agents (neuroleptics) for short-term symptomatic control [5].

Chorea, myoclonus, and paroxysmal dyskinesias have been rarely reported in hypoglycemia [36].

Metabolic Disorders Associated with Organ Failure

Hepatic Failure

Acquired hepatolenticular degeneration refers to a syndrome with cognitive, motor, and psychiatric abnormalities related to liver insufficiency of any cause. Symptoms may occur insidiously or subacutely, within a few weeks or months. Involvement of the basal ganglia in acquired hepatocerebral degeneration may cause chorea, dystonia, tremor, parkinsonism, and myoclonus [37, 38]. Orobuccolingual dyskinesias are frequently observed [39]. Most commonly, patients with hepatic encephalopathy present with asterixis, a form of negative postural myoclonus due to transient lapses in tone (video—segment 4). It is caused by portal-systemic shunting leading to excessive concentration of manganese in the globus pallidum [40]. The manganese deposition can be suspected by a hyperintense signal in the basal ganglia in T1-weighted brain MRI sequences (Fig. 2c). Improvement of the movement disorders related to hepatic failure requires treatment of hepatic encephalopathy, which may include liver transplantation, as appropriate [41]. It is imperative to distinguish acquired hepatocerebral degeneration from Wilson's disease, since both have similar phenotype, but different causes and treatments [42].

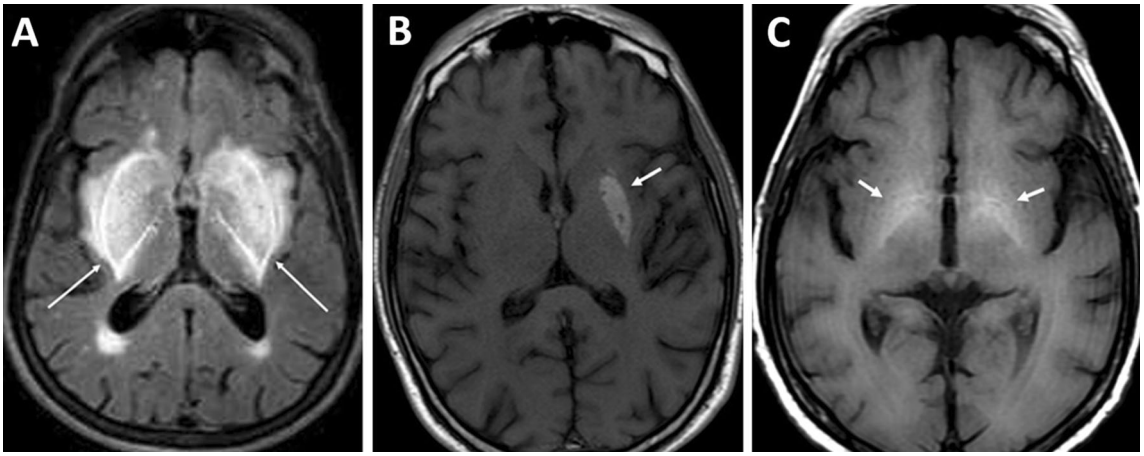

Fig. 2 Neuroimaging of selected metabolic disorders (1/2). Axial FLAIR brain MRI shows bilateral symmetrical hyperintense signal in the basal ganglia that delineate the lentiform nucleus (fork sign) (**a**), characteristic of metabolic acidosis. Axial T1-weighted brain MRI shows hyperintense signal in the left striatum in a patient with right-sided hemichorea/hemiballismus due to non-ketotic hyperglycemia in a patient (**b**). Courtesy of Dr. Victor Hugo Rocha Marussi, Department of Radiology, Hospital Beneficência Portuguesa, SP, Brazil. Axial T1-weighted brain MRI shows symmetrical hyperintense signal in basal ganglia, suggestive of manganese deposition (**c**) in a patient with acquired hepatolenticular degeneration

Renal Failure

Neurological symptoms are frequent in patients with acute or chronic renal failure, including encephalopathy, seizures, and movement disorders [6, 43]. Patients with chronic renal failure often develop only subtle neurological symptoms. Several movement disorders have been reported in patients with severe uremia and end-stage renal disease, particularly those with diabetic nephropathy. The most common are myoclonus, tremor, and asterixis, usually associated to encephalopathy and seizures (video—segment 5). Chorea is rarely observed in uremia, mostly restricted to elderly patients [6, 44].

Uremia is capable of inducing secondary restless legs syndrome (RLS) and is frequently associated with neuropathy. The frequency of RLS in patients with renal failure is around 20 to 40%, when compared to the general population. Pathophysiological mechanisms involved remain poorly understood but involve iron dysfunction and dopaminergic dysfunction. Dopaminergic replacement therapy, standard treatment for idiopathic RLS, can be futile in cases associated with renal failure or iron deficiency [45]. Correction of these abnormalities becomes necessary for symptomatic control.

The association between myoclonus and renal failure may include an important differential diagnosis (Table 2). Hemodialysis patients may present with transient dysarthria and, occasionally, seizures. This results from transient osmotic imbalance during dialysis and is associated with hyperintense signal in the basal ganglia. Slower and longer sessions appear to prevent this phenomenon [46]. Patients with uremic encephalopathy consistently improve after dialysis or renal transplantation [5].

A very rare autosomal recessive disease called *action myoclonus-renal failure* syndrome is caused by a loss-of-function mutation of the *SCARB2* gene [47]. Myoclonus persists after renal transplantation in these patients.

Cardiac Arrest and Brain Hypoxia

Anoxia after cardiac arrest or related to insufficient cerebral blood flow (e.g., septic shock) induces a global brain injury, presenting as diffuse encephalopathy. Pathophysiological mechanisms involve not only neuronal and glial damage but also excitatory brain injury. A glutamate efflux increases intracellular calcium and cytokines that lead to proinflammatory states. The most susceptible brain regions affected in anoxia are hippocampus, basal ganglia, Purkinje cells, and neocortex [48].

Several movement disorders may occur after cardiac arrest, related to hypoxic or anoxic complications. Although myoclonus is the most common, patients may also present chorea, parkinsonism, tremor, dystonia, and tics (video—segment 6) [49]. Patients with myoclonus after cardiac arrest must undergo electroencephalogram to evaluate for focal myoclonic seizures, which would require selected antiepileptic drugs, sodium valproate, levetiracetam, and/or clonazepam [50, 51].

Lance-Adams syndrome is characterized by chronic myoclonus, ataxia, and cognitive impairment that appear days or weeks after cardiac arrest and disabling action myoclonus is common [52]. These patients tend have had preexistent hypercapnia due to respiratory failure (from severe pneumonia or asthma, for instance), which explains that this complication is uncommon after cardiac arrest not preceded by respiratory insufficiency, even if it has been rarely reported after "brief hypoxemia" (e.g., after a surgery or anesthesia) without cardiac arrest [53].

Table 2 Differential diagnosis when myoclonus and renal failure coexist

	Cause	Symptoms	Treatment
Uremic encephalopathy	Renal failure	Myoclonus	Dialysis or renal transplantation
Dialysis encephalopathy	Aluminum toxicity, rapid uremia reduction	Myoclonus, speech disturbance, and seizures	Avoid aluminum-containing dialytic fluids, slow correction of uremia
Drug toxicity (iatrogenic)	Myoclonus-inducing drugs in renal failure[a]	Myoclonic encephalopathy	Avoid acyclovir, ciprofloxacine, dobutamine, cephalosporins, and gabapentin in uremic patients
Action myoclonus-renal failure syndrome	*SCRBA2* mutations (autosomal recessive)	Myoclonus without encephalopathy; proteinuric glomerulonephropathy	First line: levetiracetam, topiramate, zonisamide; second line: clonazepam

[a] Drugs that induce myoclonus in the context of renal insufficiency include acyclovir, ciprofloxacine, dobutamine, cephalosporins, and gabapentin

Post-pump chorea usually occurs after cardiopulmonary bypass or after congenital heart surgery in children (video—segment 7). Orofacial dyskinesias, hypotonia, ballismus, and supranuclear gaze palsy may be present in these patients [54, 55].

Miscellaneous Disorders

Polycythemia

Polycythemia rubra vera is a myeloproliferative disease characterized by increased red blood cells. Neurological manifestations may include headache, vertigo, paresthesias, visual deficits, ischemic stroke, or tinnitus. Rarely, polycythemia rubra vera may present with movement disorders, most commonly chorea [56]. Chorea restricted to the orofaciolingual region or to a hemibody (hemichorea) may also be part of the phenotypic spectrum [56, 57]. Chorea has also been reported associated with secondary causes of polycythemia [58].

Iron Dysregulation

Several neurodegenerative diseases, such as neurodegeneration with brain iron accumulation (NBIA) and Friedreich ataxia, are associated with dysregulation of brain iron metabolism [59]. Hemochromatosis, a genetic disease, may increase iron content in basal ganglia but is unlikely to cause movement disorders [60].

Conclusions

Abnormal movements associated with metabolic disorders are common in daily clinical practice. Because of the therapeutic implications, general neurologists, clinicians, and hospitalists must be aware about the subtypes of movement disorders and the most common metabolic conditions associated with these neurological manifestations. Movement disorders may be the most prominent neurological symptoms in metabolic abnormalities. Treatment of the underlying metabolic disorders usually improves the associated abnormal movements.

Compliance with Ethical Standards

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