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Movement disorders in adults with metabolic encephalopathy: A chart-based cross-sectional study

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Abstract

Background & Objectives: The objective of this study was to determine the prevalence and characteristics of movement disorders in adult patients diagnosed with various types of metabolic encephalopathies in the Philippines. *Methods:* This is a descriptive cross-sectional study based on a 7-year chart review of admitted adult patients diagnosed with any of the fifteen different types of metabolic encephalopathy who experienced movement disorders during their hospital stay. Movement disorders were characterized based on phenomenology, onset, and distribution. *Results:* Out of 9,773 neurologic referrals, 2,055 patients (17%) were diagnosed with metabolic encephalopathy, mainly the hypoxic-ischemic type. Only 4% (76) exhibited involuntary movements, primarily linked to hypoxic-ischemic encephalopathy. Three movement disorders were reported: myoclonus (64), tremor (10), and parkinsonism (2). The 6th decade was the most common age for these disorders, predominantly in males. Most cases appeared acutely within 7 days after onset of encephalopathy and were mostly diffuse and multifocal.
Conclusion: The findings highlight myoclonus as the predominant movement disorder in metabolic encephalopathy, particularly in hypoxic-ischemic and uremic types.

Keywords: Metabolic encephalopathy, movement disorders, myoclonus, hypoxic-ischemic encephalopathy, tremor, parkinsonism

INTRODUCTION

Movement disorders commonly result from a variety of neurodegenerative or structural brain diseases. However, they can also arise due to a broad spectrum of systemic diseases. When severe enough, these systemic conditions can lead to metabolic encephalopathy by disrupting the metabolic functions of nerve cells in the cerebral cortex and central brain nuclei.[1]

Metabolic encephalopathy is defined as a failure of some other organ system resulting in a global disturbance of cerebral function in the absence of a structural brain injury.[1] It can present as an acute or subacute onset of confusion, decreased consciousness, delirium, or coma, with fluctuating course and no consistent focal deficits.[2] Typically, loss of consciousness in these conditions correlates with decreased cerebral metabolism, and the rate of change in the underlying metabolic disturbance is as significant as its absolute level.[1] Movement disorders may be the harbinger of an underlying systemic disease or may emerge during the course of metabolic encephalopathy.[1,3]

Studying the co-existence of these two neurologic entities can provide new insights because each systemic disease can affect the brain in different ways. Unlike neurodegenerative movement disorders, those occurring in the setting of acquired metabolic encephalopathy secondary to systemic diseases are frequently amenable to causal treatment of the underlying condition. Hence, arriving at an early correct diagnosis is a key priority. These clinical clues could potentially play an important diagnostic or therapeutic roles especially in resource-limited settings, where detailed clinical assessment can compensate for limitations in diagnostic testing. Furthermore, this study could offer valuable insights into the Philippine context due to the scarcity of data on this topic. Thus, this study aims to determine the prevalence and characteristics of different movement disorders among various types of

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acquired metabolic encephalopathy.

This descriptive, cross-sectional, retrospective study was conducted at Makati Medical Center (MMC), involving a chart review of patients admitted from January 2017 to December 2023 at the intensive care unit (ICU) and inpatient wards/ private rooms. The subjects were patients diagnosed with any acquired metabolic encephalopathy who developed movement disorders. The independent variables were patients' age and sex, as well as the presence and specific types of metabolic encephalopathy. Age was classified into 10-year intervals (decades). Fifteen different types of metabolic encephalopathy were evaluated, including hypoxic-ischemic, hypercapnic, uremic, hepatic, hyperammonemic, hypo- and hyperglycemic, hypertensive, septic, hypo- and hypernatremic, hypo- and hypercalcemic and hypo- and hyperthyroid types. The dependent variables focused on the presence of movement disorders and their specific phenomenology, which included athetosis, ataxia, ballism, chorea, dystonia, myoclonus, parkinsonism, and tremor. The body distribution of involuntary movements was categorized as focal, multifocal and diffuse. Onset was classified into categories of less than 7 days, between 7 and 14 days, and more than 7 days.

METHODS

Chart review

Following approval from the Institutional Review Board of MMC (protocol number: MMCIRB2024-03-033), data collection commenced by initially reviewing the department's database. Patients diagnosed with any form of metabolic encephalopathy from 2017 to 2023 were identified. All charts were screened and chart review was performed from May 2024 to February 2025 after IRB approval. Informed consents were waived due to the retrospective nature of the study. Records were examined in both physical charts and electronic medical records, or a combination of both. In instances of multifactorial etiologies of metabolic encephalopathy, only the predominant type was used for categorization. There can be cases of multiple metabolic derangements in one patient, but one predominant type can contribute more to the encephalopathic state than the others. Additionally, the results of various diagnostic tests, including blood tests, brain imaging and electroencephalography (EEG), were reviewed to establish and confirm certain clinical parameters.

Study population

To prevent misrepresentation of the various types of movement disorders and metabolic encephalopathy, a total enumeration was conducted without using a specific sampling method. There were eight types of movement disorders and fifteen types of metabolic encephalopathy assessed. The inclusion and exclusion criteria were as follows:

Inclusion criteria: Patients referred to or admitted by the Neurology service with diagnosis of any acquired metabolic encephalopathy who subsequently developed new-onset movement disorders; Adult patients aged at least 19 years; Admission dates ranging from 2017 to 2023; Patients admitted at the intensive care unit and inpatient wards or private rooms; Individuals from any ethnic background.

Exclusion criteria: Pediatric patients aged 18 years or younger; Patients who had pre-existing movement disorders of any kind prior to hospitalization; Drug-induced movement disorders; Patients with pre-existing structural brain lesions (such as stroke, neoplasm, or trauma) that could produce movement disorders.

Statistical method

In this study, both the dependent and independent variables were categorical, represented as numbers and percentages displayed in tables and graphs. As this is a descriptive study, no comparisons between groups or any statistical analyses were conducted. The overall frequency of movement disorders was calculated over a 7-year period from 2017 to 2023, rather than on a yearly basis.

RESULTS

Types of metabolic encephalopathy

A review was conducted on the records of 2,055 metabolic encephalopathy patients from a total of 9,773 neurologic referrals between January 2017 and December 2023, yielding a prevalence rate of 17% (refer to Figure 1). Table 1 presents the frequencies of the different types of metabolic encephalopathy. The most common type identified was hypoxic-ischemic encephalopathy (HIE) at 31.14%. During their admission, only 4% (n=76) of patients developed movement disorders, while the vast majority (n=1979, 96%) did not (refer to Figure 2).

Table 1: Frequencies of metabolic encephalopathies

Types of metabolic encephalopathy	Number	Percentage
Hypoxic-ischemic	640	31.14%
Septic	554	26.96%
Hyponatremic	264	12.85%
Uremic	208	10.12%
Hepatic	134	6.52%
Hyperglycemic	69	3.36%
Hypercapnic	68	3.31%
Hypoglycemic	46	2.24%
Hypernatremic	28	1.36%
Hypertensive	25	1.22%
Hypercalcemic	7	0.34%
Hyperthyroid	6	0.29%
Hypocalcemic	4	0.19%
Hypothyroid	2	0.10%
Hyperammonemic	0	0%

Clinical and demographic profile of patients with metabolic encephalopathy who developed movement disorders

Table 2 outlines the clinical and demographic profiles of patients with metabolic encephalopathy who developed movement disorders during their admission, with prevalence of 4% (refer to Figure 2).

Metabolic encephalopathy with movement disorders (overall)

The most common age group was the 6th decade (n=23, 30.26%), while the least common was the 2nd decade (n=2, 2.63%). The majority of patients were males, making up 53.95% (n=41). HIE was the leading type of metabolic encephalopathy associated with involuntary movements, accounting for 52.63% (n=40). The 7-year review only identified myoclonus (n=64, 84%), tremor (n=10, 13%) and parkinsonism (n=2, 3%) as involuntary movements (refer to Figure 3). Most of these appeared within less than 7 days following the onset of encephalopathy (n=71, 93.42%). The most common distribution patterns observed were diffuse (39.47%) and multifocal (38.16%).

Metabolic encephalopathy with myoclonus

A total of 64 patients (84%) experienced myoclonus, with the most prevalent age group being the 6th decade (n=20, 31.25%). Majority were males (n=37, 57.81%). Myoclonus primarily developed following HIE (n=38, 59.38%). In HIE, 95% (38 out of 40 cases) of involuntary movements were myoclonus. A vast majority (n=60, 93.75%) had myoclonus within 7 days of onset. The distribution pattern was predominantly diffuse (n=26, 40.63%). Information regarding the body distribution of four cases was not documented in the records.

EEG findings in patients with myoclonus

Figures 4 and 5 show the EEG findings to help distinguish between epileptic and non-epileptic myoclonus. All EEG patterns of myoclonus cases were found to be abnormal. Among the 64 patients with myoclonus who underwent EEG testing, 17 of them (26%) exhibited epileptiform discharges. Additionally, from the 33 cases of post-hypoxic myoclonus, 9 patients (27%) also showed epileptiform discharges. In both groups, the predominant abnormal pattern was a slowing

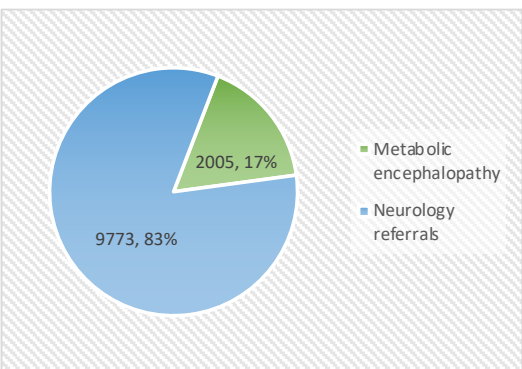

Figure 1. Frequency of metabolic encephalopathy among Neurology referrals (7-year period)

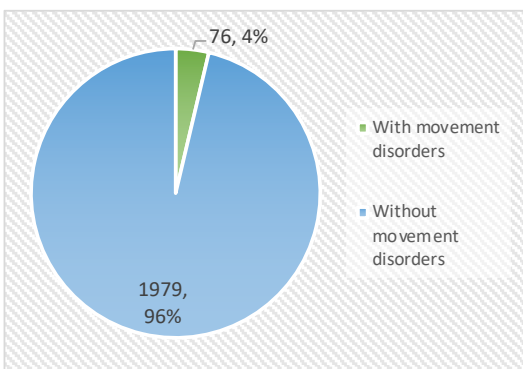

Figure 2. Frequency of movement disorders among patients with metabolic encephalopathy (7-year period)

Table 2: Clinical and demographic profile of adult patients with metabolic encephalopathy who developed movement disorders

Clinical and demographic profile		Movement disorders (all cases) (n=76)		Myoclonus (n=64)		Tremor (n=10)		Parkinsonism (n=2)	
		Number	Percentage	Number	Percentage	Number	Percentage	Number	Percentage
Age (in years)	19-29	2	2.63%	1	1.56%	1	10%	0	0%
	30-39	6	7.89%	6	9.37%	0	0%	0	0%
	40-49	2	2.63%	2	3.13%	0	0%	0	0%
	50-59	8	10.53%	6	9.37%	2	20%	0	0%
	60-69	23	30.26%	20	31.25%	2	20%	1	50%
	70-79	11	14.47%	10	15.63%	1	10%	0	0%
	80-89	17	22.37%	13	20.31%	4	40%	0	0%
	⩾90	7	9.21%	6	9.37%	0	0%	1	50%
Sex	Male	41	53.95%	37	57.81%	4	40%	0	0%
	Female	35	46.05%	27	42.19%	6	60%	2	100%
Types of metabolic encephalopathy	Hypoxic-ischemic	40	52.63%	38	59.38%	2	20%	0	0%
	Uremic	16	21.05%	14	21.88%	2	20%	0	0%
	Septic	7	9.21%	3	4.69%	2	20%	2	100%
	Hyponatremic	4	5.26%	2	3.12%	2	20%	0	0%
	Hypercapnic	4	5.26%	4	6.25%	0	0%	0	0%
	Hepatic	1	1.32%	1	1.56%	0	0%	0	0%
	Hyperglycemic	1	1.32%	1	1.56%	0	0%	0	0%
	Hypernatremic	1	1.32%	1	1.56%	0	0%	0	0%
	Hypercalcemic	1	1.32%	0	0%	1	10%	0	0%
	Hyperthyroid	1	1.32%	0	0%	1	10%	0	0%
	Hypoglycemic	0	0%	0	0%	0	0%	0	0%
	Hypertensive	0	0%	0	0%	0	0%	0	0%
	Hypocalcemic	0	0%	0	0%	0	0%	0	0%
	Hypothyroid	0	0%	0	0%	0	0%	0	0%
	Hyperammonemic	0	0%	0	0%	0	0%	0	0%
Onset	<7 days	71	93.42%	60	93.75%	10	100%	1	50%
	7-14 days	4	5.26%	4	6.25%	0	0%	0	0%
	>14 days	1	1.32%	0	0%	0	0%	1	50%
Distribution	Focal	13	17.11%	13	20.31%	0	0%	0	0%
	Multifocal	29	39.47%	21	32.81%	8	80%	2	100%
	Diffuse	30	38.16%	26	40.63%	2	20%	0	0%

Note: Data were missing on the distribution of myoclonus in 4 patients

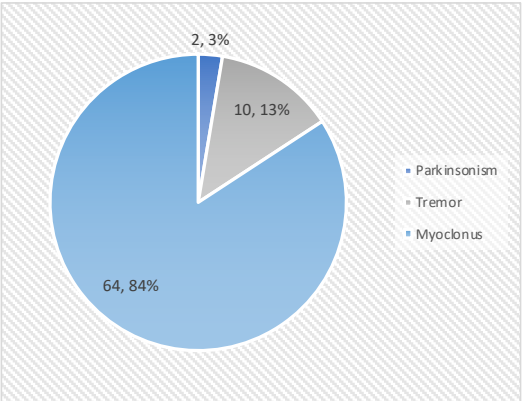

Figure 3. Frequency of movement disorders in patients with all-cause metabolic encephalopathies

of background activity. All EEG patterns with burst suppression were patients with post-hypoxic myoclonus.

Metabolic encephalopathy with tremor

A total of 10 patients (13%) were reported to have tremors, with the most common age group being the 8th decade (n=4, 40%). The majority were females (n=6, 60%). No predominating type of encephalopathy was seen. All ten cases (100%) developed tremors within 7 days of onset, with the majority exhibiting a multifocal pattern of distribution (n=8, 80%).

Metabolic encephalopathy with parkinsonism

Only two cases of parkinsonism were identified, occurring in patients from the 6th and 9th decades. Both cases developed parkinsonism following septic encephalopathy. One patient experienced tremors within less than 7 days, while the other case had tremors appearing more than 14 days after onset of encephalopathy. Both cases exhibited diffuse parkinsonism.

DISCUSSION

General insights on metabolic encephalopathy

All cases of metabolic encephalopathy, regardless of the underlying systemic cause, results from a disruption in the brain's attention and arousal centers, primarily the ascending reticular activating system (ARAS). The ARAS receives

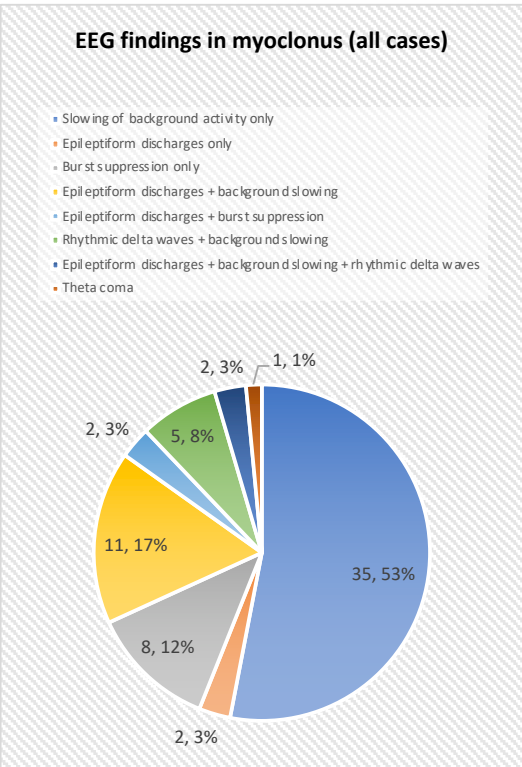

Figure 4. EEG findings in patients with myoclonus (64 out of 76 patients underwent EEG)

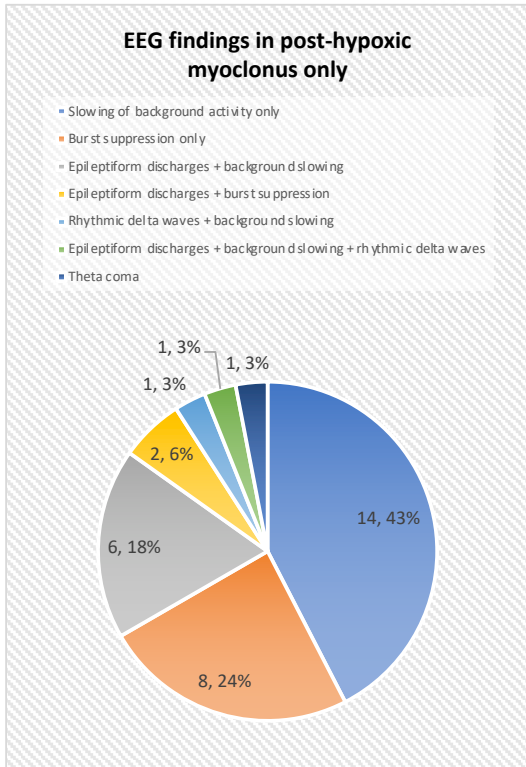

Figure 5. EEG findings in patients with post-hypoxic myoclonus (33 out of 38 patients underwent EEG)

extensive input from the cortex, deep cerebral nuclei, cingulate gyrus, spinal cord, visual and auditory centers, thalamus, hypothalamus, and hippocampus. Disruptions in these pathways can result in encephalopathy.[2] Some of these structures are also involved in the genesis of movement disorders. Hence, there is some degree of overlap in the affected anatomical structures.

In our study, the overall prevalence of metabolic encephalopathy is 17%. In comparison, another study revealed that the prevalence of metabolic encephalopathy was 70% of ICU patients, 16% of post-acute care patients, 10% of emergency department patients, and 42% of hospice patients.[4] We found that the most common type of metabolic encephalopathy was HIE, accounting for more than half of all encephalopathies. This supports the vulnerability of the brain due to its high metabolic demands, limited tolerance for hypoxia-ischemia, and sensitivity to reperfusion injury.[5-7] In another study, HIE accounts for 10-15% of causes of non-traumatic brain injury in emergency departments and acute hospitals.[7]

Movement disorders in metabolic encephalopathy

As expected, the low prevalence of movement disorders (4%) in encephalopathy is due to the fact that the encephalopathic state mainly occurs in cases of severe metabolic dysfunction. In comparison, another study found that the prevalence of movement disorders in neuro-metabolic disease is 29%.[8] However, their patient population was younger and did not consider the presence or absence of encephalopathy.

Our study underscored the susceptibility of the aging brain, evidenced by the high prevalence of metabolic encephalopathy in the elderly population. The highest frequency of patients was observed in the 6th, 7th and 8th decades. Aging is marked by the buildup of molecular and cellular damage throughout an organism's life, resulting in physical decline and a higher risk of disease. The failure to repair this damage results in impaired physiological functions, ultimately contributing to disease and death.[9-10] The sharp decline in the 9th decade likely indicates the mortality of individuals who reach that age, compounded by the smaller size of the Philippine population in the oldest age groups.[11]

Myoclonus was the most commonly found involuntary movement in our study. This differs from another study that identified myoclonus as the second most common after tremor-like movements.[12] Like in overall cases of metabolic encephalopathy, HIE was the most prevalent type in patients with involuntary movements. Some regions of the brain are more susceptible to hypoxia, particularly the gray matter and areas supplied by the distal branches of deep and superficial penetrating blood vessels, such as the upper brainstem, cerebellum, and subcortical structures.[13]

In our study, a vast majority of patients with involuntary movements presented acutely within 7 days of encephalopathy onset, with most showing multifocal and diffuse patterns. It indicates the expansive and immediate damage to the brain.

Myoclonus in metabolic encephalopathy

Myoclonus mainly arises in the cerebral cortex, brainstem or spinal cord.[14] A close relationship between myoclonus and hypoxic brain injury was noted. In our study, majority (59.38%) of myoclonus cases were post-hypoxic. In the literature, post-hypoxic myoclonus is typically classified as either acute or chronic, depending on the onset of myoclonus. Acute post-hypoxic myoclonus usually manifests within 12 to 48 hours following hypoxic brain injury[15], thought to be cortical or subcortical in origin, and also possibly involving the brainstem and spinal cord.[15-17] Chronic post-hypoxic myoclonus, also known as Lance-Adams Syndrome, is action- and startle-sensitive, appearing days to weeks after coma resolution. It is primarily cortical in origin but can also be subcortical.[18]

The second most common type of encephalopathy found in our patients with myoclonus was the uremic type. In the literature, myoclonus is frequently observed in uremic states.[19] Myoclonus is more prominent in uremia compared to most other metabolic encephalopathies.[20] This is a separate entity from myoclonus associated with dialysis encephalopathy, which results from aluminum toxicity or rapid reduction of uremia.[21]

In a minority of cases, myoclonus was co-existent with hypercapnic (6.25%), septic (4.69%), hyponatremic (3.12%), hypernatremic (1.56%), hepatic (1.56%) and hyperglycemic (1.56%) encephalopathies. They have distinct pathomechanisms, but they all cause encephalopathy by disrupting the metabolic functions of nerve cells in the brain. Only a few cases of myoclonus have been reported in hypercapnic encephalopathy[22-24], while it is infrequent in septic-associated encephalopathy.[25-29] Myoclonus is common in hypernatremic

encephalopathy, but rare in hyponatremia[19]; although they can be seen in cases of osmotic demyelinating syndrome (ODS).[30,31] Positive or negative myoclonus (asterixis) is commonly observed in hepatic encephalopathy, and its progression corresponds to the development of the confusional state.[32] Although chorea and ballism are well-recognized in some cases of non-ketotic hyperglycemic states, there have been reported cases of myoclonus in hyperglycemic encephalopathy.[20,33-34]

Epileptic vs non-epileptic myoclonus

Movement disorders (especially myoclonus) and epilepsy are overlapping nosological entities that manifest along both a phenomenological and a pathophysiological continuum.[35] In one study, 29% of seizures fulfilled the diagnostic criteria for a defined movement disorder. The most common was myoclonus at 35%, which was expected given their cortical origin.[36] Therefore, EEG is essential for differentiating myoclonic jerks associated with electrographic discharges from non-epileptic myoclonus, which are common in metabolic encephalopathy.[37]

In our study, 27% of all patients with myoclonus and 25% of post-hypoxic myoclonic jerks exhibited epileptiform discharges. Comparing it to other studies, epileptiform discharges are found in 33-55% of cases with post-hypoxic myoclonus.[15,16,38,39] EEG findings in acute post-hypoxic myoclonus can include burst suppression, spike-wave activity, diffuse slow background, generalized periodic discharges, and alpha coma.[18] The EEG background may vary from suppression to a completely continuous pattern.[40] In some instances, epileptiform activity may not be exhibited on EEG, suggesting that the myoclonus is likely of subcortical origin.[41] Occasionally, a coma pattern may be displayed, indicating that the myoclonus originates from the brain stem and results from cortical inhibition or brain stem release.[42] Literature on the EEG findings of myoclonus in non-hypoxic encephalopathies is lacking.

Tremor in metabolic encephalopathy

Depending on the specific types of tremor, the pathophysiological mechanisms are driven by abnormal oscillations of the basal ganglia, thalamus, and cerebellum and disconnection of the cerebellum from other brain regions.[43] In our study, we did not identify a predominant type of encephalopathy associated with tremors. This is in contrast to myoclonus, which was primarily observed in hypoxic-ischemic and uremic encephalopathies. According to the literature, tremor is frequently seen in hyperthyroid and uremic states.[19] Postural and action tremors in the upper limbs can occur in hyperthyroidism, either alone or alongside systemic symptoms such as tachycardia, diarrhea, and sweating.[44] Tremor is infrequent in hypercalcemic and hyponatremic states.[19,28,45-47] Tremor has also been reported in ODS.[48] Cases of post-hypoxic tremor have also been described.[49] In a larger study, acute hypoxia was demonstrated to increase tremor frequency in the 6–12 Hz range.[50] In septic encephalopathy, tremor is relatively infrequent.[29] One case was reported during septic encephalopathy from COVID-19 infection.[51]

Parkinsonism in metabolic encephalopathy

Parkinsonism mainly results from the degeneration of dopaminergic neurons in the pars compacta of the substantia nigra (SNc) and the resulting reduction in the levels of dopamine in the striatum, the main synaptic target of SNc axons.[52] In our study, two cases of parkinsonism were noted in septic encephalopathy. Infectious causes are less common etiologies of secondary parkinsonism.[53] Parkinsonism resulting from the loss of dopaminergic neurons due to an infectious process develops rapidly, in contrast to the gradual and progressive nature of idiopathic Parkinson's disease.[54] The relationship between parkinsonism and the important viral and bacterial pathogens has been described.[55] During sepsis, the area postrema facilitates the entry of circulating inflammatory mediators into the brainstem, leading to neuroinflammation.[56,57] Parkinsonism may then arise from a direct loss of dopaminergic neurons.[58] Since the central nervous system is not an immune-privileged organ, it is likely that innate immune responses in the brain could be affected for an extended duration after sepsis.[59]

As for the limitations of this study, first, this study is limited on its retrospective nature involving a chart-based review. Generalizability was restricted due to the small sample size of patients who presented with movement disorders, as well as the exclusion of pediatric patients and since it was a single-center study. Movement disorders were diagnosed and described by their overseeing physicians based on clinical evaluation, many of them were not movement specialists. Reliance on clinical judgement for diagnosing movement disorders may introduce observer bias. Furthermore, only patients who

were admitted were included in the study. Those who arrived at the emergency room with metabolic encephalopathy and involuntary movements but were not admitted for reasons such as death, refusal, or transfer were excluded. This study also failed to look at delayed-onset movement disorders among patients who survived and were discharged. Other types of metabolic encephalopathy were not considered, such as drugs or toxins, withdrawal states, nutritional/vitamin deficiencies, and imbalances in magnesium and phosphorus.

In conclusion, this study provides important insights into the prevalence and characteristics of movement disorders in the background of metabolic encephalopathy. The findings highlight myoclonus as the predominant movement disorder in metabolic encephalopathy, particularly associated with the hypoxic-ischemic and uremic types. This may allow clinicians to anticipate the development of acute-onset movement disorders, especially in elderly group. The acute onset of involuntary movements underscores the rapid neurological impact of these conditions. The presence of movement disorders may serve as a guide to the diagnosis of the underlying systemic disease. They may potentially become more informative than the symptoms referable to the organ primarily involved. Given the limited existing data on this topic in the Philippine context, these findings underscore the need for increased awareness and further research into the relationship between metabolic encephalopathy and movement disorders, ultimately contributing to improved diagnosis and management of affected patients.

DISCLOSURE

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Conflicts of interest: None.

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