



Hemiballism and chorea with acute/subacute onset: a retrospective series

Yeşim Güzey Aras¹ · Sena Boncuk Ulaş² · Türkan Acar¹ · Bilgehan Atılğan Acar¹

Received: 27 October 2022 / Accepted: 30 January 2023 / Published online: 7 February 2023
© The Author(s) under exclusive licence to Belgian Neurological Society 2023

Abstract

Introduction Chorea is a hyperkinetic movement disorder with sudden, irregular, random, dance-like involuntary movements, and ballism is usually one-sided, high-amplitude movements at the proximal of the extremities. In the etiology of acute chorea/hemiballismus, it is necessary to distinguish drugs first and then focus on metabolic causes. The most important etiological causes that may provoke acute/subacute onset chorea/hemiballismus are hypo-hyperglycemia and electrolyte disorders. In this study, we aim to present 19 patients who were admitted to our clinic with movement disorder with acute/subacute onset and diagnosed with chorea/hemiballismus.

Methods The study was completed with 19 patients. Routine biochemistry, HbA1c level, hemogram, sedimentation, CRP, hepatitis panels, detailed infective parameters, HIV, vitamin B12 level, folate levels, and thyroid function tests were studied. All patients underwent neuro-imaging.

Results 16(84.2%) were female and 3(15.8%) were male. The lowest age of the patients was 48 years, the highest age was 89 years, and the mean age was 72.21 years. Thirteen (68.42%) patients had a diagnosis of diabetes mellitus in their history. The blood glucose levels of these patients at the time of admission: the lowest was 99 mg/dl and the highest was 1200 mg/dl. HbA1c values of 11(84.61%) of the 13 patients were also found elevated. Thirteen (68.4%) patients had hemiballismus, 4(21.1%) patients had bilateral choreoathetosis in the four extremities, and 2(10.2%) patients had ballism limited to one upper extremity.

Conclusions Chorea/hemiballismus is a movement disorder that is rare and can occur due to a wide range of etiologies. The most common metabolic cause is NKHHS.

Introduction

Chorea is a hyperkinetic movement disorder (MD) with sudden, irregular, random, dance-like involuntary movements that affect any part of the body. Athetosis is a slower form of chorea and ballism is usually one-sided, high-amplitude

movements at the proximal of the extremities [1, 2]. They may appear as primary disorders or secondary to various medical situations [3]. Some endocrine diseases, liver and kidney failure, hyperglycemia, and electrolyte disorders could often present with MD. The pathophysiology of these disorders is complex and poorly understood. These disorders are seen as a result of dysfunction of the connections between the Basal Ganglia (BG)s and frontal motor cortex. In etiology, many conditions, such as infections, medications, and paraneoplastic syndromes, could appear, but cerebrovascular diseases and non-ketotic hyperglycemic hyperosmolar state (NKHHS) are the most often cause of them [3–6]. Also, advanced age, type 2 diabetes mellitus (DM), and female gender were shown as the risk factors for chorea/hemiballismus, which occurs as a rare complication of hyperglycemia [7, 8]. In the etiology of acute chorea/hemiballismus, it is necessary to distinguish drugs first and then focus on metabolic causes. The most important etiological causes that may provoke acute/subacute onset chorea/

✉ Sena Boncuk Ulaş
senaboncuk@gmail.com

Yeşim Güzey Aras
yesimguzeyaras@hotmail.com

Türkan Acar
tdeniz38@hotmail.com

Bilgehan Atılğan Acar
bilgehanacar@hotmail.com

¹ Department of Neurology, Sakarya University Faculty of Medicine, Sakarya, Turkey

² Department of Neurology, Edirne Keşan State Hospital, 22800 Keşan/Edirne, Turkey

hemiballismus are hypo-hyperglycemia and electrolyte disorders. Especially in NKHHS, the status starts mostly acute/subacute and progresses within days [3, 9].

In this study, we aim to present 19 patients who were admitted to our clinic with MD with the acute/subacute onset and diagnosed with chorea/hemiballismus in the last 3 years and discuss these patients in line with the literature.

Methods

Patients who applied to Sakarya University Training and Research Hospital Neurology Clinic between March 2018 and March 2021 with acute/subacute onset MD were evaluated. Thirty patients that diagnosed with chorea/hemiballismus and followed up regularly in the MD outpatient clinic for at least 6 months were included in the study. Among these patients, those diagnosed with Idiopathic Parkinson's Disease and/or Parkinson's plus syndrome and those diagnosed with Huntington's Disease by genetic tests were not included in the study. In addition, patients who used multiple psychiatric drugs and were not followed up regularly in outpatient clinic were also excluded from the study. The study was completed with a total of 19 patients. The study was approved by the Sakarya University Ethics Committee on October 15, 2021, with the approval number E-71522473–050.01.04.74628–466. Their medical

histories, family histories, co-morbidities, and medications were investigated. Then, routine biochemistry, HbA1c level, hemogram, sedimentation, CRP, hepatitis panels, detailed infective parameters, HIV, vitamin B12-folate levels, and thyroid function tests were studied. All patients underwent neuro-imaging.

Results

Of the 19 patients, 16(84.2%) were female and 3(15.8%) were male. The lowest age of the patients was 48, the highest was 89, and the mean age was 72.21. Thirteen (68.42%) patients had a diagnosis of DM. The blood glucose levels of these patients at the time of admission; the lowest was 99 mg/dl and the highest was 1200 mg/dl. HbA1c values of 11(84.61%) of the 13 patients were also found elevated. In 3 (50%) of the 6 (31.57%) patients who did not have DM, blood glucose level was found to be above 120 mg/dl during admission. Of the 19 patients, 10 (52.3%) had hypertension (HT), 7 (36.8%) had cerebrovascular disease (CVD), 2 (10.5%) had congestive heart failure (CHF), 2 (10.5%) had malignancy, and 1 (5.3%) had chronic obstructive pulmonary disease (COPD). One (5.3%) patient had concomitant COVID-19 pneumonia (Table 1). Thirteen (68.4%) patients had hemiballismus, 4 (21.1%) patients

Table 1 Demographic data and medical history of patients

Patient	Age	Sex	DM	Blood glucose (mg/dL)	HbA1c (%)	Other co-morbidities
1	48	F	None	122	5,8	Breast cancer
2	55	F	Yes	312	8,3	CVD
3	69	F	Yes	344	15,7	None
4	89	M	Yes	720	15,7	CVD, prostat cancer
5	82	F	Yes	99	6,1	HT, CHF
6	70	M	Yes	109	5,5	CORD
7	79	F	None	95	5,8	HT, CVD
8	78	F	Yes	681	11,9	HT
9	71	F	Yes	132	6,2	COVID pneumonia
10	72	F	Yes	120	6,9	HT
11	89	M	None	171	6	HT, PTE
12	80	F	None	84	5,7	None
13	62	F	Yes	1200	21	HT
14	65	F	None	128	6	None
15	78	F	Yes	528	10,06	HT, CHF
16	61	F	Yes	304	10,2	HT, CVD
17	74	F	None	119	5,7	CVD, HT, Epilepsy
18	74	F	Yes	207	15	HT, CVD
19	76	F	Yes	800	8,6	CVD

DM diabetes mellitus, CVD cerebrovascular disease, HT hypertension, CHF congestive heart failure, CORD chronic obstructive respiratory disease, PTE pulmonary thromboembolism

had bilateral choreoathetosis in the four extremities, and 2 (10.2%) patients had ballism limited to one upper extremity.

Hemiballismus

In hemiballismus patients, in 8(61.53%) of 13 patients, no features were detected on Brain Computed Tomography (CT), while contralateral thalamic hematoma in 2(15.3%) patients, and contralateral hyperdensity in BG in 3 (23.07%) patients was seen on Brain CT. Cranial Magnetic Resonance Imaging (MRI) revealed no features in T1-weighted images in 10 (76.92%) of 13 patients. In 1 (7.69%) patient, contralateral BG hyperintensity was seen in the T1 MRI, while Cranial MRI was not performed in 2 (15.38%) cases with hematoma detected on Brain CT (Fig. 1). Valproic acid (VA) was started in 5 (38.46%) of 13 patients with hemiballismus, haloperidol in 3 (23.07%), and olanzapine was started in 1 (7.69%) patient. In 4 (30.7%) patients, the symptoms could not be controlled with monotherapy, and combination therapy was started. The combination of haloperidol and VA was used in 3 (23.07%) patients, and the combination of haloperidol, VA, and gabapentin was used in 1(7.69%) patient. In the etiology, 6 (46.15%) of these 13 patients

with hemiballismus had NKHHS, 2 (15.3%) had thalamic hematoma, and 5 (38.46%) had no etiological cause. Surveys of these patients, 11 (84.61%) had a complete recovery with treatment, 1 (7.69%) had secondary parkinsonism due to haloperidol without follow-up 6 months, and 1 (7.69%) died due to NKHHS.

Bilateral choreoathetosis

In bilateral choreoathetosis cases, 3 (75%) of the 4 patients had no feature on Cranial MRI T1 and Brain CT, while in 1 (25%) patient, bilateral BG lesions were observed on both Brain CT and Cranial MRI T1 (Fig. 1). VA was used in 2 (50.0%) of 4 patients and haloperidol was used in 1 (25.0%) patient. In 1 (25.0%) patient, monotherapy was not sufficient, and thus, haloperidol, VA, levetiracetam, and gabapentin combination were administered. Looking at the etiology of these patients, NKHHS was present in 2 (50.0%) and no etiological causes in the other 2 (50.0%); however, one of them was diagnosed with COVID-19 pneumonia simultaneously with choreoathetosis. According to the survey, complete recovery was observed in 3 (50.0%) patients, whereas 1 (25.0%) patient died due to sepsis.

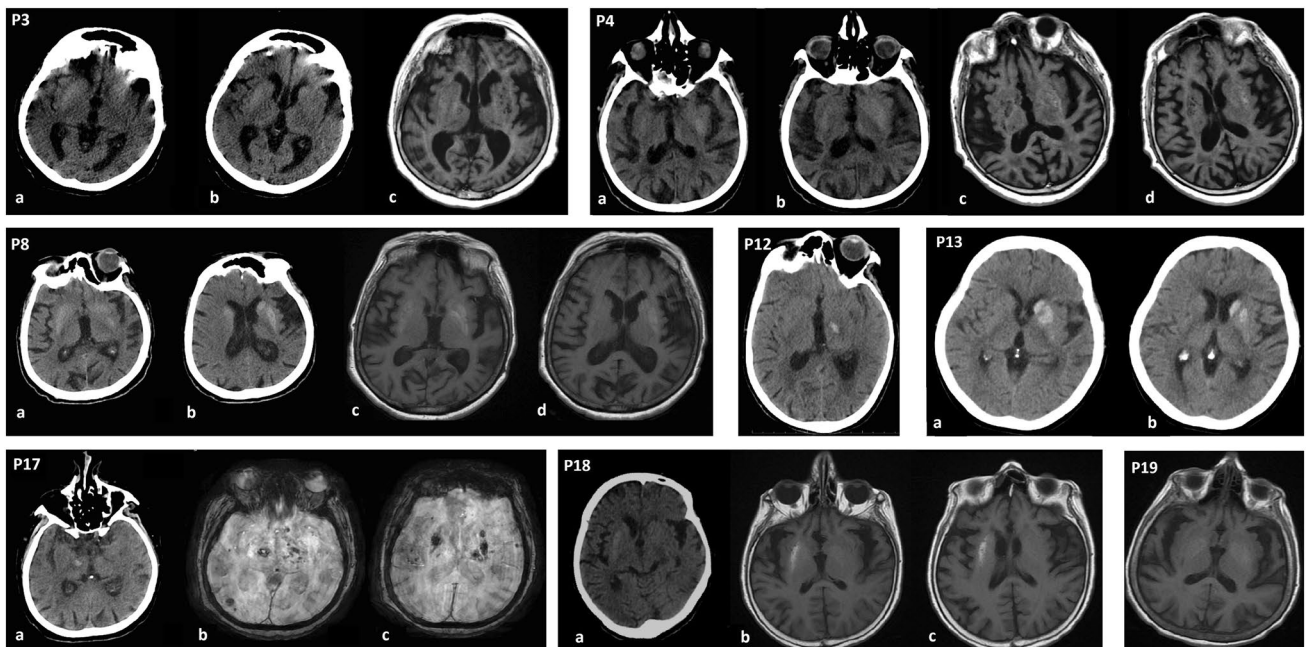


Fig. 1 **P3**: Lesion in the right BG; hyperdense in axial NCCT (a, b) and hyperintense in T1-weighted axial MRI (c), **P4**: lesions in bilateral BG; hyperdense in axial NCCT (a, b) and hyperintense in T1-weighted axial MRI (c, d) more prominent on the left, **P8**: lesions in bilateral putamen and head of caudate nucleus; hyperdense in axial NCCT (a, b) and hyperintense in T1-weighted axial MRI (c, d) more prominent on the left, **P12**: left thalamic hemorrhage in axial NCCT, **P13**: lesion in the left putamen and head of caudate nucleus; hyper-

dense in axial NCCT (a, b), **P17**: right thalamic hemorrhage in axial NCCT (a), SWI sequence MRI shows bilateral BGs microhemorrhages in addition to the thalamus (b, c), **P18**: lesion in the right putamen; hyperdense in axial NCCT (a) and hyperintense in T1-weighted axial MRI (b, c), and **P19**: lesions in bilateral BGs; hyperintense in T1-weighted axial MRI. *P* patient, *BG* basal ganglia, *NCCT* non-contrast computed tomography, *MRI* magnetic resonance imaging (acquired with 1.5 T), *SWI* susceptibility-weighted imaging

Upper extremity ballism

In upper extremity ballism patients, 1 (50.0%) had no features on the Brain CT and Cranial MRI, while in the other patient had hyperdensity on the Brain CT and hyperintensity on the Cranial MRI T1 at the bilateral BGs were observed (Fig. 1). While haloperidol was started in 1(50.0%) patient, monotherapy was not sufficient in the other one, and haloperidol and VA combination therapy was administered. The etiology of these patients, NKHHS was detected in 1(50.0%), while breast cancer was detected in the other. According to the survey, 1(25.0%) completely recovered, and the other died due to sepsis (Table 2).

Discussion

Chorea/hemiballismus is a hyperkinetic MD characterized by short-term, random, irregular, involuntary movements caused by damage in the pathways in the BGs, leading to excessive dopamine activity. Chorea usually affects the distal of the extremities, rarely face and trunk, while ballism mostly affects the proximal of the extremities and has a wider amplitude [4]. The etiology of chorea/hemiballismus is quite diverse and can be seen as a result of metabolic diseases, hypoxic-ischemic events, vascular disorders, medications, toxins, infections, and systemic inflammatory, immunological diseases. Among the metabolic causes, the most common is NKHHS [10]. The acute/subacute presentation of chorea/hemiballismus is an essential finding for determining the etiology. In the acute onset, first NKHHS, and after other metabolic conditions, and drugs should be considered in the differential diagnosis [9, 11]. The age of onset of chorea/ballism and the acute or progressive form of onset may give an idea of the differential diagnosis. In terms of the age of onset, especially in Huntington's Disease, the onset of symptoms and the genetic diagnosis of patients occur at later ages. The way of onset is one of the most important determinants in differentiating the causes of neurodegenerative chorea. Especially, chronic chorea lasting months to years is usually associated with a neurodegenerative condition [12].

The incidence of NKHHS-related chorea/hemiballismus increases with female gender, advanced age, newly diagnosed type 2 DM, and non-well-regulated blood sugar. In a series of 15 patients of NKHHS-related chorea/ballism, Chen et al. found that 11 (73.3%) of the patients were female, 4 (26.7%) were male, while in our study, 16 (84.2%) of the patients were female, and 3 (15.8%) were male [1, 6]. In the same study, the average age was 54.1, and 10 (66.6%) of the 15 patients had a DM history, 5 (33.4%) had a newly diagnosed DM. In our study, the average age was 72.21, and DM was present in 13 (68.42%) of 19 patients, and NKHHS-related chorea/ballism was detected in 9 of them

[1]. The occurrence of NKHHS-related chorea/hemiballismus is most often acute/subacute. Considering the patient reports and patient series in the literature, it is seen that the chorea/hemiballismus appears in a few days to a few weeks [1, 4–7]. In our patients, the time of onset of symptoms was similar to the literature.

The pathophysiology of NKHHS-related chorea/hemiballismus is not clear. Several hypotheses were proposed related to this condition. The first hypothesis is the depletion of gamma-aminobutyric acid (GABA) as an alternative energy source in hyperglycemia, and the appearance of thalamic disinhibition and hyperkinetic movements in combination with a deficiency of GABA. Other hypotheses are that hyperglycemia directly affects brain metabolism, disrupts blood–brain barrier permeability, and hyperglycemia-related ischemia and microhemorrhages are seen in BGs [4, 7, 8]. However, these hypotheses are insufficient to explain the pathophysiology. While especially in metabolic disorders, the effect on BG is expected to be bilateral, these hypotheses cannot explain why the manifestation is mostly seen unilaterally.

Considering the neuro-imaging in NKHHS-related chorea/hemiballismus, it is seen that studies in the literature recommend using Brain CT and Cranial MRI T1 as a diagnostic criterion [13, 14]. Mostly expected, hyperdensity on Brain CT and hyperintensity on Cranial MRI T1 is observed in the contralateral BG. In rare patients, ipsilateral or bilateral BG may also be affected [13]. However, it was also found in the literature that there are no features in neuro-imaging in some of the patients of NKHHS-related chorea/ballism [1, 6]. We also found features in neuro-imaging in 5 of 9 patients with NKHHS-related chorea/hemiballismus in line with the literature. In 3 of these 5 patients, we detected hyperdensity on Brain CT and hyperintensity on Cranial MRI T1 in the contralateral of the affected extremity. In one of the other 2 patients with ballism in the right upper extremity, hyperintensity in bilateral BGs on Brain CT and hyperintensity in contralateral BG on Cranial MRI T1 were observed. In the other patient with bilateral choreoathetosis, bilateral BG lesions were observed on both Brain CT and Cranial MRI. We did not detect any features on the Brain CT and Cranial MRI of the remaining 4 patients with NKHHS-related chorea/hemiballismus.

In the etiology, there are many reasons for the development of acute/subacute chorea/hemiballismus, other than NKHHS. In our study, 9 patients were presented with the NKHHS. Looking at the literature, it seems that the development of acute MD due to ischemic/hemorrhagic stroke is rare and the rate is %1–4 of all strokes [15]. A study that screened 1500 patients with stroke between 1990–1999 reported that 56 patients developed post-stroke MD, and 20 (35.7%) of these had chorea [16]. In our study, consistent with the literature, we detected contralateral thalamic hematoma in only 2 patients.

Table 2 Characteristics, onset, imaging findings, treatments, etiology, and survey of patients

Patient	Chorea/ballism characteristics	Onset to hospital	CT	MRI T1	Treatment	Etiology	Survey
1	Ballism of left upper limb	One day	Unremarkable	Unremarkable	Haloperidol	Malignancy	Doing well
2	Left HB	Several days	Unremarkable	Unremarkable	Olanzapine	NKHHS	Doing well
3	Left HB	Three days	Hyperdense in right BG	Unremarkable	VA	NKHHS	Seconder parkinsonism because of haloperidol (no follow-up for six months)
4	Ballism of right upper limb	Several days	Hyperdense in bilateral BG	Hyperintense in left BG	Multi-treatment*	NKHHS	Exitus
5	Left HB	Several months	Unremarkable	Unremarkable	Haloperidol	Undetermined	Doing well
6	Left HB	One week	Unremarkable	Unremarkable	VA	Undetermined	Doing well
7	Right HB	Several days	Unremarkable	Unremarkable	VA	Undetermined	Doing well
8	Choreoathetosis of bilateral upper and lower limbs	Ten days	Hyperdense in bilateral BG	Hyperintense in bilateral BG	Multi-treatment**	NKHHS	Exitus
9	Choreoathetosis of bilateral upper and lower limbs	One day	Unremarkable	Unremarkable	Haloperidol	Covid pneumonia	Doing well
10	Choreoathetosis of bilateral upper and lower limbs	Two days	Unremarkable	Unremarkable	VA	Undetermined	Doing well
11	Right HB	Two months	Unremarkable	Unremarkable	VA	Undetermined	Doing well
12	Right HB	Two days	Acute left thalamic hemorrhage	-	Multi-treatment***	Thalamic hemorrhage	Doing well
13	Right HB	One month	Hyperdense in left BG	Unremarkable	Multi-treatment*	NKHHS	Exitus
14	Right HB	Several days	Unremarkable	Unremarkable	Haloperidol	Undetermined	Doing well
15	Choreoathetosis of bilateral upper and lower limbs	Several days	Unremarkable	Unremarkable	VA	NKHHS	Doing well
16	Right HB	One day	Unremarkable	Unremarkable	VA	NKHHS	Doing well
17	Left HB	One day	Acute right thalamic hemorrhage	-	Haloperidol	Thalamic hemorrhage	Doing well
18	Left HB	Two weeks	Hyperdense in right BG	Hyperintense in the right BG	Multi-treatment*	NKHHS	Doing well
19	Right HB	Three months	Unremarkable	Unremarkable	Multi-treatment*	NKHHS	Doing well

HB hemiballismus, *NKHHS* non-ketotic hyperglycemic hyperosmolar state, *BG* basal ganglion, *VA* valproic acid

*Haloperidol, valproic acid

**Haloperidol, valproic acid, levetiracetam, gabapentin

***Haloperidol, valproic acid, gabapentin

There are also patient reports in the literature that contralateral hemiballismus due to thalamic hematoma. The occurrence of hemiballismus due to thalamic involvement without the direct involvement of BGs was associated with the interruption of afferent and efferent pathways of BGs [17]. In the remaining

8 patients, we did not detect any etiological factors. In these patients, we also did not observe any neuro-imaging findings. In one of these patients, breast cancer was diagnosed 6 months after discharge. When we look at the literature, it is seen that paraneoplasia is also present in the etiology, especially

in adults. Patients of small cell lung cancer were reported mostly. However, in paraneoplasia-associated MD, the survey is expected to be poor, while in our patient, the symptoms were controlled with monotherapy in a short time, and did not recur during follow-ups. Therefore, the co-occurrence of malignancy and the ballism is considered incidental [18]. Moreover, one of these 8 patients was found to have an acute onset bilateral choreoathetosis concurrent with COVID-19 pneumonia. Looking at the literature, COVID-19-associated acute chorea is quite rare. In some patient reports and a few newly published studies, it has been stated that movement disorders can be seen with and/or after COVID-19, and chorea can also be detected [19–22]. The absence of any other risk factor in the etiology, the absence of features in the neuro-imaging, and the simultaneous onset of symptoms suggested that COVID-19 pneumonia may be a possible etiological factor in the choreoathetosis in this patient.

In treatment, first, the underlying cause should be determined and treated [23]. NKHHS-induced chorea/hemiballismus is often benign and responds quickly to strict control of blood sugar. However, until DM regulation is achieved, medical treatment can be started for symptom control [3]. In the literature, it was noted that NKHHS-induced chorea/hemiballismus was completely treated within 6 months, often with blood sugar regulation and medication [1]. In treatment, dopamine-depleting therapy (neuroleptics) and antiepileptics, especially VA, are recommended [23]. When looking at the studies, it is seen that neuroleptics, especially haloperidol and VA from antiepileptics, are preferred first [1, 5, 6]. However, the use of neuroleptics has been limited due to side effects such as secondary parkinsonism. In our study, similar to the literature, we preferred VA in 7 (36.84%), neuroleptic in 6 (31.57%), and combinations of neuroleptic and antiepileptics in 6 (31.57%) patients. Tetrabenazine, which is mainly used in the treatment of Huntington's chorea, can also be used in the treatment of other choreas besides Huntington's. There are publications in the literature, showing that tetrabenazine can also be used in the treatment of hemiballismus due to NKHHS [23, 24]. However, we did not use tetrabenazine in any of our patients, especially since it is difficult to provide in our country. We think that tetrabenazine can also be used as a treatment option in resistant chorea/hemiballismus that cannot be controlled with the other treatments mentioned. According to the surveys, only 3 (15.78%) patients died, and 1 (5.26%) developed secondary parkinsonism. In the other 15 (78.94%), complete recovery was observed at 6 months of follow-up.

Conclusions

Chorea/hemiballismus is a rare MD and can occur due to a wide range of etiologies. The most common metabolic cause is NKHHS. Therefore, blood sugar and HbA1c values

should be examined in every patient especially acute/subacute chorea/ hemiballismus, with or without a DM diagnosis. Brain CT and Cranial MRI should be evaluated for differential diagnosis, but also be noted that in some patients, they can be completely normal. With neurological examination, laboratory tests, and imaging methods, a diagnosis can be made and etiology can be determined in most patients. In our study, despite modern neuro-imaging methods and laboratory tests, etiological causes were not detected in about 30% of patients, similar to the literature. It is believed that multicenter studies are needed to clarify the group whose etiology cannot be determined.

Author contributions YGA: data curation, formal analysis, investigation, methodology, and writing—review & editing. SBU: data curation, formal analysis, investigation, methodology, and writing. TA: data curation, formal analysis, investigation, methodology, and writing—review and editing. BAA: data curation, formal analysis, investigation, methodology, and writing—review & editing.

Data availability The datasets analysed during the current study are available from the corresponding author on reasonable request.

Declarations

Conflict of interest The authors declare that they have no conflict of interest.

Ethical approval This article was approved by the ethics committee of Sakarya University Faculty of Medicine on 25.10.2021 with the number E-71522473-050.01.04-74628-466.

Informed consent Informed consent is not required.

References

- Chen C, Zheng H, Yang L, Hu Z (2014) Chorea-ballism associated with ketotic hyperglycemia. *Neurol Sci* 35(12):1851–1855. <https://doi.org/10.1007/s10072-014-1968-1>
- Pedroso JL, Barsottini OG, Espay AJ (2019) Movement disorders in metabolic disorders. *Curr Neurol Neurosci Rep* 19(2):7. <https://doi.org/10.1007/s11910-019-0921-3>
- Raza HK, Jing J, Cui G, Liang X, Hua F, Zhang Z, Tang H, Shi H, Chen H (2017) Hemichorea caused by nonketotic hyperosmolar state: a case report and review of the literature. *Somatosens Mot Res* 34(1):44–46. <https://doi.org/10.1080/08990220.2016.1278205>
- Ticona J, Zaccone V, Zaman U, Kashani D, Chung Z, McFarlane IM (2020) Hemichorea-hemiballismus as an unusual presentation of hyperosmolar hyperglycemic syndrome. *Am J Med Case Rep* 8(6):159–161 (Epub 2020 Apr 5. PMID: 32432159; PMCID: PMC7236990)
- Marques JS, Monteiro N, Nunes A, Machado J, Olivério J, Martins AS, Correia A (2018) Hyperglycaemic hemichorea. *Eur J Case Rep Intern Med*. 5(4):000807. https://doi.org/10.12890/2018_000807
- Ryan C, Ahlskog JE, Savica R (2018) Hyperglycemic chorea/ ballism ascertained over 15 years at a referral medical center.

- Parkinsonism Relat Disord 48:97–100. <https://doi.org/10.1016/j.parkreldis.2017.12.032>
7. Cho HS, Hong CT, Chan L (2018) Hemichorea after hyperglycemia correction: a case report and a short review of hyperglycemia-related hemichorea at the euglycemic state. *Medicine (Baltimore)* 97(10):e0076. <https://doi.org/10.1097/MD.00000000000010076>
 8. Wang W, Tang X, Feng H, Sun F, Liu L, Rajah GB, Yu F (2020) Clinical manifestation of non-ketotic hyperglycemia chorea: a case report and literature review. *Medicine (Baltimore)* 99(22):e19801. <https://doi.org/10.1097/MD.00000000000019801>
 9. Yu JH, Weng YM (2009) Acute chorea as a presentation of Graves disease: case report and review. *Am J Emerg Med* 27(3):369.e1–369.e3. <https://doi.org/10.1016/j.ajem.2008.05.031>. (PMID: 19328390)
 10. Awasthi D, Tiwari AK, Upadhyaya A, Singh B, Tomar GS (2012) Ketotic hyperglycemia with movement disorder. *J Emerg Trauma Shock* 5(1):90–91. <https://doi.org/10.4103/0974-2700.93095>
 11. D'Angelo R, Rinaldi R, Pinardi F, Guarino M (2013) Acute chorea-dystonia heralding diabetes mellitus. *BMJ Case Rep*. <https://doi.org/10.1136/bcr-2013-009221>. (PMID: 24000205; PMCID: PMC3794330)
 12. Mestre TA (2016) Chorea. *Continuum (Minneap Minn)* 22:1186–1207. <https://doi.org/10.1212/CON.0000000000000349>. (PMID: 27495204)
 13. Takamatsu K (2014) Diabetic chorea. *Brain Nerve* 66(2):121–128 (Japanese. PMID: 24523310)
 14. Chua CB, Sun CK, Hsu CW, Tai YC, Liang CY, Tsai IT (2020) “Diabetic striatopathy”: clinical presentations, controversy, pathogenesis, treatments, and outcomes. *Sci Rep* 10(1):1594. <https://doi.org/10.1038/s41598-020-58555-w>
 15. Suri R, Rodriguez-Porcel F, Donohue K, Jesse E, Lovera L, Dwivedi AK, Espay AJ (2018) Post-stroke movement disorders: the clinical, neuroanatomic, and demographic portrait of 284 published cases. *J Stroke Cerebrovasc Dis* 27(9):2388–2397. <https://doi.org/10.1016/j.jstrokecerebrovasdis.2018.04.028>. (Epub 2018 May 21 PMID: 29793802)
 16. Alarcón F, Zijlmans JC, Dueñas G, Cevallos N (2004) Post-stroke movement disorders: report of 56 patients. *J Neurol Neurosurg Psychiatry* 75(11):1568–1574. <https://doi.org/10.1136/jnnp.2003.011874>
 17. Alagöz AN, Alaçam Köksal S, Acar BA, Acar T, Bölük A (2016) Talamik Hemorajiye Bağlı Gelişen Hemiballismus: Olgu Sunumu. *Türk Beyin Damar Hastalıklar Dergisi* 22(1):21–24. <https://doi.org/10.5505/tbdhd.2016.77598>
 18. Kyle K, Bordelon Y, Venna N, Linnoila J (2022) Autoimmune and paraneoplastic chorea: a review of the literature. *Front Neurol* 13:829076. <https://doi.org/10.3389/fneur.2022.829076>
 19. Brandão PRP, Grippe TC, Pereira DA, Munhoz RP, Cardoso F (2021) New-onset movement disorders associated with COVID-19. *Tremor Other Hyperkinet Mov (N Y)* 8(11):26. <https://doi.org/10.5334/tohm.595>
 20. Byrnes S, Bisen M, Syed B, Huda S, Siddique Z, Sampat P, Russo R, Oueida Z, Johri G, Dargon I (2020) COVID-19 encephalopathy masquerading as substance withdrawal. *J Med Virol* 92(11):2376–2378. <https://doi.org/10.1002/jmv.26065>
 21. Schneider SA, Hennig A, Martino D (2022) Relationship between COVID-19 and movement disorders: a narrative review. *Eur J Neurol* 29(4):1243–1253. <https://doi.org/10.1111/ene.15217>. (Epub 2021 Dec 31 PMID: 34918437)
 22. Ray STJ, Abdel-Mannan O, Sa M, Fuller C, Wood GK et al (2021) Neurological manifestations of SARS-CoV-2 infection in hospitalised children and adolescents in the UK: a prospective national cohort study. *Lancet Child Adolesc Health* 5(9):631–641. [https://doi.org/10.1016/S2352-4642\(21\)00193-0](https://doi.org/10.1016/S2352-4642(21)00193-0). (Epub 2021 Jul 15. Erratum in: *Lancet Child Adolesc Health*. 2021 Jul 28;: Erratum in: *Lancet Child Adolesc Health*. 2021 Dec;5(12):e46. PMID: 34273304; PMCID: PMC8279959)
 23. Bashir H, Jankovic J (2018) Treatment options for chorea. *Expert Rev Neurother* 18(1):51–63. <https://doi.org/10.1080/14737175.2018.1403899>
 24. Jaafar J, Rahman RA, Draman N, Yunus NA (2018) Hemiballismus in uncontrolled diabetes mellitus. *Korean J Fam Med* 39(3):200–203. <https://doi.org/10.4082/kjfm.2018.39.3.200>. (PMID: 29788710; PMCID: PMC5975992)

Publisher's Note Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.

Springer Nature or its licensor (e.g. a society or other partner) holds exclusive rights to this article under a publishing agreement with the author(s) or other rightsholder(s); author self-archiving of the accepted manuscript version of this article is solely governed by the terms of such publishing agreement and applicable law.