

to more typical meningothelial formations could be observed easily.<sup>2</sup> Sometimes the rhabdoid morphology of the tumour cells are focal or only seen after the first recurrence of a more typical meningioma. Our patient was initially misdiagnosed with a granulocytic sarcoma in the local hospital, possibly because only some tumour cells had rhabdoid features and some tumour cells nonspecifically stained for MPO. The differential diagnosis includes metastatic carcinoma or melanoma, primary dural-based sarcoma, and malignant glioma with dural invasion.<sup>2</sup> The diagnosis of the metastatic rhabdoid meningioma lesions in the liver posed a particular challenge, as there was only limited material available from the core needle biopsy and the tumour cells were entirely rhabdoid in appearance. Primary malignant rhabdoid tumour of the liver must be considered in the differential diagnosis. A total of 27 patients with malignant rhabdoid tumours of the liver have been reported<sup>7,8</sup> and most (89%) occurred in the first 2 years of life. The correct diagnosis of metastatic rhabdoid meningioma may be resolvable by a detailed history, careful clinical examination, immunohistochemical staining for vimentin and EMA, and electron microscopy of the tumour cells.

Rhabdoid meningiomas are highly aggressive and prone to recurrence and metastasis despite radical surgical excision and radiotherapy.<sup>8</sup> Perry et al.<sup>2</sup> reported 15 patients with rhabdoid meningioma of the CNS. Of these, 13 patients (87%) experienced at least one recurrence, eight (53%) died because of the disease, and two (13.3%) demonstrated metastasis to the lung and lymph nodes. A mitotic index greater than 4 per 10 high power fields and a MIB-1 labelling index greater than 4% were considered high proliferative indices based on their study.<sup>2</sup> Rhabdoid meningiomas have been reported to occur mainly in the cerebral convexities and the parasagittal region,<sup>5</sup> and an intraspinal rhabdoid meningioma

is highly unusual. Two patients with intraspinal rhabdoid meningiomas have been reported, but they both were primary cerebral rhabdoid meningiomas with leptomeningeal dissemination.<sup>9,10</sup> The present report is the first of a recurrent intraspinal rhabdoid meningioma with liver metastasis.

In summary, we have described a patient with a rare intraspinal rhabdoid meningioma that, despite radical surgical excision, radiotherapy, and chemotherapy, recurred and metastasized to the liver 22 months after initial presentation. Immunohistochemical staining of tumour cells for vimentin and EMA may aid in the diagnosis of this rare tumour.

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# An unusual presentation of hypokalemic paralysis with evolving pure motor hemiparesis

Yan-Ting Lu, Min-Yu Lan, Jia-Shou Liu, Wei-Hsi Chen\*

Department of Neurology, Chang Gung Memorial Hospital-Kaohsiung Medical Center, 123 Ta Pei Road, Niao Sung Hsiang, Kaohsiung 833, Taiwan  
College of Medicine, Chang Gung University, Kaohsiung, Taiwan

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## ABSTRACT

Pure motor hemiparesis is a vascular syndrome that is occasionally mimicked by central or spinal pathologies. However, metabolic neuromuscular disorders have not been reported to mimic this condition. We present a 52-year-old male patient with hypokalemic paralysis who presented with the early symptoms of acute-onset pure motor hemiparesis. Neurological examinations revealed right-sided weakness without bulbar, extraocular, or respiratory involvement. Ischemic stroke was initially diagnosed on the basis of the acute-onset unilateral motor deficit and the patient's history of hypertension, stroke, and previous cerebral infarctions. The right hemiparesis and weakness of the left limbs worsened on the day after admission. The patient's weakness rapidly reversed after correction of hypokalemia, and a diagnosis of hypokalemic paralysis was finally established. This unusual hemineurological presentation should alert medical personnel to the possibility of reversible metabolic neuromuscular disorders, thereby avoiding delayed diagnosis.

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## 1. Introduction

Primary or secondary hypokalemia may trigger or precipitate periodic or non-periodic skeletal muscle weakness.<sup>1</sup> Generally,

\* Corresponding author. Tel.: +886 7 731 7123x3399; fax: +886 7 732 7385.  
E-mail address: brainhemostasis@yahoo.com (W.-H. Chen).

paralysis occurs rapidly, progresses gradually and distributes symmetrically, and it minimally involves bulbar and respiratory muscles. Although primary periodic paralysis is considered a benign neuromuscular disorder, there have been a few fatalities reported from secondary hypokalemic myopathy.<sup>2–4</sup> Therefore, a delay of diagnosis may increase the risk of damage to the patient. Here, we present a patient with hypokalemic neuromuscular disorder that mimicked pure motor hemiparesis.

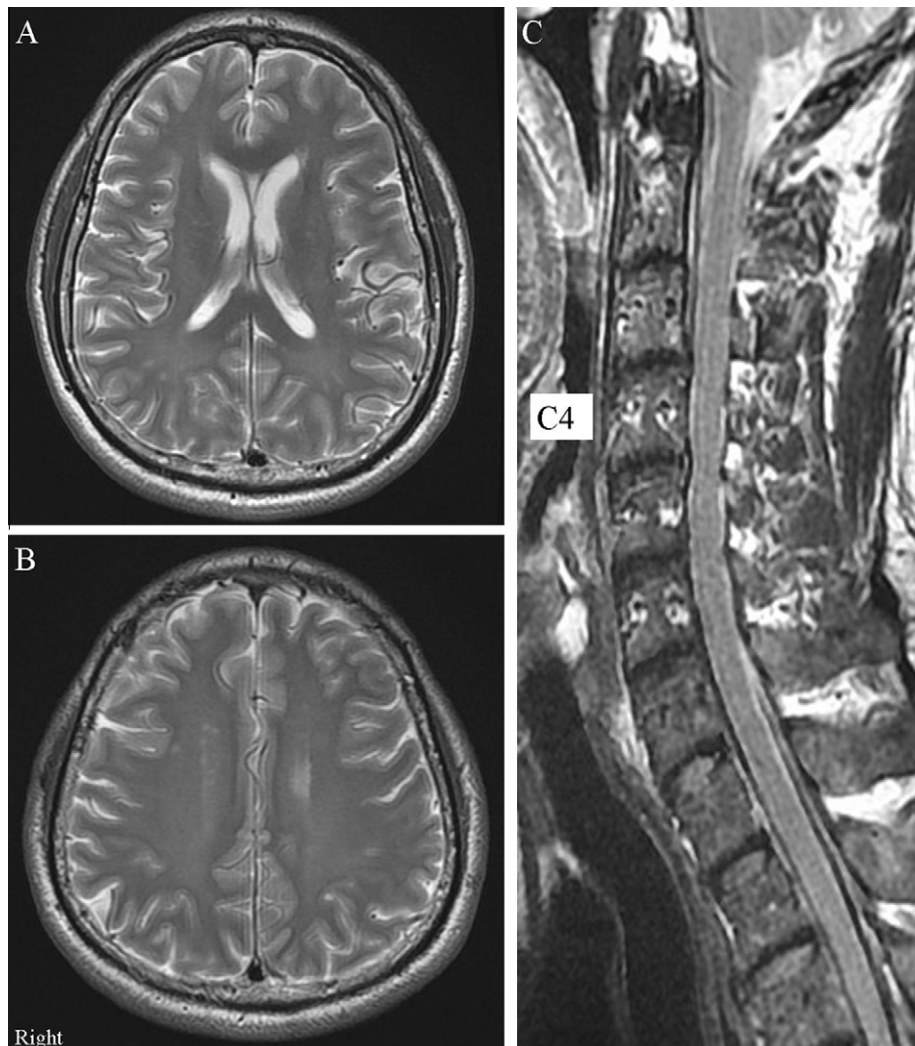
## 2. Case report

A 52-year-old man experienced sudden-onset mild clumsiness in the right arm and leg for 3 days. On the morning of the fourth day, the weakness in his right limbs exacerbated. He was unable to lift his right limbs, stand or walk and was admitted to our emergency department. He had previously had right hemiparesis due to ischemic stroke followed by complete recovery, and also hypertension and hypertriglyceridemia for 5 years. He denied any history of recent craniofacial trauma, a high calorie or carbohydrate diet, consuming beverages or alcohol, or an antecedent infection.

On presentation, his blood pressure was 150/90 mmHg, pulse rate 78 beats/min, body temperature 36.0 °C, and his respiratory

rate 13 breaths/min. Right flaccid hemiparesis (Medical Research Council [MRC] grade 2/5) was observed. The patient's tendon reflexes were symmetric and hyporeflexic, and he showed no bulbar or extraocular muscle weakness or myoedema. His brainstem reflexes, sensory function, and cerebellar function were preserved.

Stroke was considered the most likely diagnosis. His cranial CT scan showed no new abnormalities. However, multiple low-density areas compatible with the old infarctions were found in the brain parenchyma. An electrocardiogram revealed normal sinus rhythm with a mildly flattened T-wave. The results of biochemical, hematological, and muscle-enzyme assessments were within the reference ranges, except for the serum potassium ( $K^+$ ) level, which was 1.8 mEq/L (normal range: 3.0–4.8 mEq/L). Arterial blood gas analysis revealed metabolic alkalosis. The patient's right-side weakness worsened overnight. Subsequently, he developed weakness in the left lower limb (MRC grade 3/5). His cranial CT scan was repeated but it did not reveal any new consistent abnormality. On rechecking, the serum  $K^+$  and phosphate levels were 1.8 mEq/L and 1.8 mg/dL (normal range: 2.5–4.5 mg/dL), respectively. Hypokalemia, hypophosphatemia, and metabolic alkalosis were confirmed. Potassium and propranolol were administered, and the patient's weakness resolved rapidly. Brain MRI showed deep white matter ischemia (Fig. 1A, B), and MRI of the cervical spine revealed mild



**Fig. 1.** (A, B) Axial T2-weighted brain MRI showing deep white matter ischemia, and (C) sagittal T2-weighted cervical spine MRI showing spondylotic stenosis between C3 and C5.

**Table 1**  
Reports of hypokalemic monoparesis and hemiparesis.

	Lu et al. (present case)	2009 <sup>5</sup>	2008 <sup>6</sup>	1991 <sup>7</sup>
<i>Patient characteristics</i>				
Age (yrs), gender	52, M	28, M	86, F	42, F
Motor disability; initial (MRC grade, patient/normal)	Right UL + LL (2/5)	Right UL (1/5); right LL (2/5); left UL + LL (4/5)	Right UL + LL (2/5); left UL + LL (4/5); dropped head	Left UL + LL (4/5)
Tendon reflex	Hyporeflexia	Hyporeflexia	Hyporeflexia	Left areflexia, right hyporeflexia
Motor disability, ongoing	Left UL + LL (2/5)	NR	No	No
Tendon reflex, ongoing	No	NR	NR	NR
Muscle pain	No	No	No	Yes
Long tract sign	No	No	Chaddock sign	No
Other deficit	No	Cognitive impairment	No	Rhabdomyolysis
Serum K <sup>+</sup> level (mEq/L), minimum	1.8	1.7	1.6	1.5
Muscle enzymes	CK, myoglobin: normal; phosphate = 1.8 mg/dL	NR	CK = 559 IU/L	NR
Other abnormal laboratory tests	Metabolic alkalosis	NR	Metabolic alkalosis, albumin = 2.8 g/dL, calcium = 7.6 mg/dL	Metabolic alkalosis
Cardiac abnormality	No	NR	U wave	U wave
Brain MRI	Old lacunae	No abnormality	NR	NR
Spine MRI	Mild cervical spondylosis	Not done	Cervical spondylotic compression	NR
Underlying disease	Hypertension, hyperlipidemia, asymptomatic lacunae	Hyperthyroidism	Licorice (2.0 g/day) intake 4 years	Bisacodyl intake
Treatment	K supplement, propranolol	K supplement	K supplement, licorice withdrawal	K supplement, bisacodyl withdrawal
Outcome	Recovery	Recovery	Recovery	Recovery

CK: creatine kinase, F = female, K = potassium, LL = lower limb, M = male, Medical Research Council = MRC, NR = not reported, UL: upper limb.

spondylotic stenosis between the C3 and C5 (Fig. 1C). The patient's thyroid function, pituitary–adrenal axis function, renin–aldosterone axis function, autoimmune profile, and urine pH value were normal. Abdominal sonography revealed no adrenal mass. The patient's weakness was completely resolved on the fourth day, when his serum K<sup>+</sup> level was 2.7 mEq/L. Palpitation, hand tremor, and heat intolerance were entirely absent.

### 3. Discussion

Hypokalemic paralysis may lead to lethal cardiac dysrhythmia.<sup>2–4</sup> Therefore, early recognition and rapid correction of hypokalemia is essential. In our patient, the diagnostic dilemma was an acute onset of unilateral weakness, stroke risk factors, and a history of ischemic stroke with multiple old cerebral infarctions that reasonably indicated pure motor hemiparesis due to ischemia.

Only three patients with acute hypokalemic monoparesis or hemiparesis have been reported, excluding our patient (Table 1).<sup>5–7</sup> Although all four patients presented with monoparesis or hemiparesis, mild weakness in the “non-paralytic” limbs, which quickly progressed to tetraparesis, was detected in two patients.<sup>5,6</sup> Except for one patient who had long tract signs,<sup>6</sup> the other three patients had symmetric hyporeflexia and flaccidity in the involved limbs. Chaddock sign and Babinski's sign exhibit equal sensitivity in patients with corticospinal tract damage, but the false-positive rate for the Chaddock sign is higher than that for Babinski's sign in reported cases of hypokalemic paralysis.<sup>8,9</sup> The presence of Chaddock sign in the previously reported patients may have reflected an underlying upper motor neuron disorder. Symmetric hyporeflexia is a strong indicator of lower motor neuron involvement and an early neurological manifestation in these patients. Therefore, acute hypokalemic paralysis should be suspected in patients with monoparesis/hemiparesis with symmetric hyporeflexia despite the presence of long tract signs.

Hyperthyroidism<sup>10</sup> and licorice ingestion, which are well-established causes of acute hypokalemic paralysis, were reported in two cases.<sup>5,6</sup> However, no known cause for hypokalemia was identified in our patient. Less profound hemiparesis, normal consciousness,

normal blood glucose level, and the absence of autonomic symptoms exclude hypoglycemic hemiparesis. Our patient was taking amlopidine, but this does not disturb potassium hemostasis. However, his arterial blood gas examination indicated a metabolic alkalosis that may have led to potassium shift or loss. Unfortunately, we did not examine the transtubular potassium gradient in our patient on initial presentation.

The pathogenesis of unilateral weakness in patients with acute hypokalemic monoparesis or hemiparesis is unknown. Neuroglycopenic weakness may cause hypoglycemic hemiparesis because of the central dysfunction caused by focal dearrangements of cerebral autoregulation, vasospasm, and selective neuronal vulnerability. In our patient, hypokalemic weakness occurred on the right side, the same side as a previous stroke. Therefore, subclinical corticospinal tract damage may predispose to unilateral weakness in hypokalemic paralysis. A Chaddock sign reported with paralysis in a previous patient<sup>6</sup> also supports this probability. An evolutionary progression to tetraparesis without ongoing mental or other neural symptoms indicates a muscular disease in this circumstance.

Patients with hypokalemic paralysis rarely present with hemiparesis. Predisposing subclinical corticospinal tract involvement may account for this unusual hemineurological presentation. Therefore, this presentation should alert neurologists or emergency service physicians to reversible metabolic neuromuscular disorders, thereby avoiding a delayed diagnosis.

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## MRI appearance of a cerebral cavernous malformation in the caudate nucleus before and after chorea onset

Kazutaka Kobayashi<sup>a,b,c</sup>, Naoki Aoyama<sup>a,c</sup>, Jun Sasaki<sup>a,d</sup>, Hideki Oshima<sup>a,b</sup>, Chikashi Fukaya<sup>a,b,\*</sup>, Takamitsu Yamamoto<sup>a,b</sup>, Yoichi Katayama<sup>a,b</sup>

<sup>a</sup> Division of Neurosurgery, Department of Neurological Surgery, Nihon University School of Medicine, Tokyo, Japan

<sup>b</sup> Division of Applied System Neuroscience, Department of Advanced Medical Science, Nihon University School of Medicine, 30-1 Oyaguchi Kamimachi, Itabashi-ku, Tokyo 173-8610, Japan

<sup>c</sup> Department of Neurological Surgery, Social Insurance Yokohama Central Hospital, Yokohama, Japan

<sup>d</sup> Department of Radiology, Social Insurance Yokohama Central Hospital, Yokohama, Japan

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### ABSTRACT

Movement disorders associated with cerebral cavernous malformations (CM) are seldom reported, and chorea, in particular, is rarely associated with a CM located in the caudate nucleus. Here we report a 78-year-old female patient with chorea, who presented with choreiform movements due to a CM in the contralateral caudate nucleus. A brain MRI was obtained and compared with that obtained before the onset of chorea. The new images did not reveal further extralesional hemorrhage from the CM when compared with the previous images. The choreiform movements showed spontaneous improvement and then disappeared completely. We reviewed previous reports of patients with chorea associated with a CM, and conclude that CM located in the caudate nucleus can cause chorea.

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### 1. Introduction

A cerebral cavernous malformation (CM) is a well-described vascular malformation of the central nervous system. A CM consists of a well-circumscribed collection of dilated, thin-walled vascular channels lined by simple endothelium and thin, fibrous adventitia, and contains blood products at various stages of evolution.<sup>1</sup> Although hemorrhage associated with a CM produces extralesional blood and often leads to focal neurological deficit, a focal neurological deficit correlated with the anatomic location of a CM can occur without any evidence of extralesional blood. Movement disorders associated with CM are seldom reported,<sup>2,3</sup> and chorea, in particular, is rarely associated with a CM located in the caudate nucleus.<sup>4–7</sup> We describe a patient with chorea caused by a CM in the contralateral caudate nucleus, and review reports of chorea associated with CM.

### 2. Case report

A 78-year-old female presented with choreiform movements of her right upper extremity. She did not have a prior history of abnormal movements and was not undergoing any medical treatment. She did not have any risk factors for vascular disease, including long-term hypertension, diabetes or smoking. She also had no family history of movement disorders. Her general physical examination and blood examination results were completely normal

with no signs of infection. Neurological examination revealed choreic movements of the right upper extremity. Her cognitive function, muscle strength, sensation and cerebellar function were all normal; no pathological reflexes were observed.

A CT scan conducted at another hospital when the patient was approximately 50 years old had incidentally revealed a lesion in her brain (image not available); she was symptom-free at that time. When she was 75 years old, an MRI was performed at another hospital to investigate the lesion (Fig. 1A). The MRI revealed a 2-cm-diameter, roughly oval-shaped lesion that occupied the head of the left caudate nucleus and extended into the rostral part of the left lenticular nucleus. T2-weighted MRI revealed a focus of high signal intensity within a hypointense lesion (Fig. 1A, left), while T1-weighted MRI showed that the lesion had isointense foci superimposed on a hypointense background (Fig. 1A, right). No associated mass effect was observed. These brain MRI findings suggested that the lesion was a CM.

The patient visited our hospital for treatment of the involuntary movements that she had been experiencing for 3 days. A new MRI was obtained and compared with the previous images. Although there were no obvious changes in the T2-weighted MRI (Fig. 1A and B, left), a comparison of the T1-weighted MRI revealed that a previously isointense area lesion had become hyperintense at presentation of the chorea. No extralesional hemorrhage, edema, or increase in diameter of the CM was observed on comparison of the previous images with those at presentation (Fig. 1A and B, right). There were no other abnormal magnetic resonance intensity changes in the brain, such as those suggestive of encephalitis. Although medical treatment for the involuntary movements was

\* Corresponding author. Tel.: +81 3 3972 8111; fax: +81 3 3554 0425.

E-mail address: chikashi@med.nihon-u.ac.jp (C. Fukaya).