

## CASE REPORT

# Corticosteroid induced hyperosmolar hyperglycaemic state and hemiballismus

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## Abstract

We present an 89-year-old man with new onset of left-sided hemiballismus affecting his face, arm and leg. He was found to have hyperglycaemia with a glucose level of 20.2 mmol/l and had started prednisolone 3 months earlier for polymyalgia rheumatica. A T<sub>2</sub>-weighted magnetic resonance scan of the brain showed a hypointense lesion of the right lentiform nucleus. At follow-up, his symptoms had improved with treatment for diabetes mellitus. To our knowledge, this is the first patient to develop hemiballismus after starting corticosteroids.

## INTRODUCTION

Hemiballismus is a hyperkinetic movement disorder comprising large amplitude, from proximal to distal, flinging, throwing and involuntary movements on one side of the body [1]. Underlying causes include structural and vascular lesions of the basal ganglia or systemic causes such as hyperglycaemia, systemic lupus erythematosus, vasculitis, phenytoin use or electrolyte disturbances. Here, we report the first case, to our knowledge, of hemiballismus precipitated by corticosteroid-induced hyperglycaemia.

## CASE REPORT

An 89-year-old man presented to the emergency department with involuntary movements of his left arm and leg. His daughter noticed abnormal posturing of the left arm when walking 2 weeks earlier. Over the preceding week, the movements became more noticeable. There were no movements on the right side of the body. Three months earlier, he had been diagnosed with

polymyalgia rheumatica and started prednisolone 10 mg daily. He also had a past medical history of hypertension and osteoporosis for which he took amlodipine and calcium with cholecalciferol. There was no family history of neurological illness.

On examination, he had involuntary movements of his left arm and left leg which he could suppress briefly by holding on to a chair or his leg. The involuntary movements were most noticeable when he was agitated or walking (Supplementary Video 1). When walking he demonstrated abnormal posturing of the left hand with repetitive, stereotypical movements which seemed to involve the proximal and distal part of his limb. He tried to suppress the hand movements with holding the hand by his right hand or holding on to his leg with his left hand when sitting. The rest of the neurological examination was normal. Blood tests showed a glucose level of 20.2 mmol/l and glycated haemoglobin (HbA1c) of 12.7%. Full blood count, electrolytes, renal function, liver function, ferritin, caeruloplasmin, erythrocyte sedimentation rate and anti-nuclear antibodies were all normal. A computed tomography (CT) scan of the brain showed right basal ganglia hyperdensity initially reported as acute

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haemorrhage (Fig. 1). Magnetic resonance (MR) imaging of the brain 2 days after the CT scan showed hyperintensity of the right basal ganglia on  $T_1$ -weighted sequences and hypointensity on fluid-attenuated inversion recovery (FLAIR) sequences (Figs 2 and 3). There was no evidence of restricted diffusion (Fig. 4).

He was treated with insulin for hyperosmolar hyperglycaemic state and remained independently mobile despite ongoing involuntary movements. Upon follow-up 1 month later, his involuntary movements had improved but were still present. They did not interfere with his daily activities and he declined treatment.

## DISCUSSION

This patient had no previous history of diabetes or movement disorders. He developed hemiballismus within 3 months of starting prednisolone and was found to have developed diabetes mellitus.

Hyperglycaemia was first associated with hemiballismus in 1960, but thought to be a rare cause [2]. In a meta-analysis of 53 cases, 91% of cases were in people of Asian origin and the mean glucose level was 27 mmol/l with a mean HbA1c of 14.4% [3]. Case series suggest that two-thirds of those affected are women and it may be the first presentation of diabetes mellitus in 17–80% of patients [4]. Hyperglycaemia may also produce



Figure 1: Non-contrast axial CT image showing mild diffuse hyperdensity of the right basal ganglia without associated mass effect.

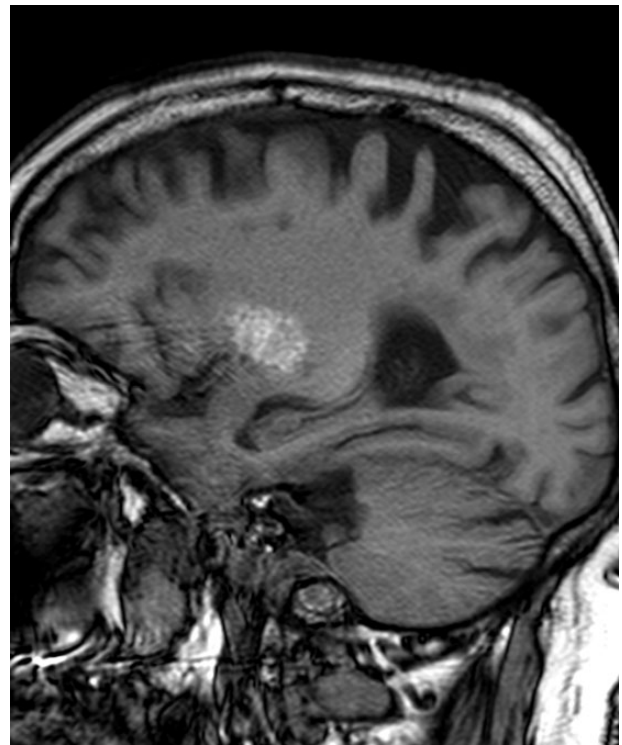


Figure 2: Non-contrast  $T_1$ -weighted sagittal MR image showing mild diffuse hyperintensity in the right basal ganglia.

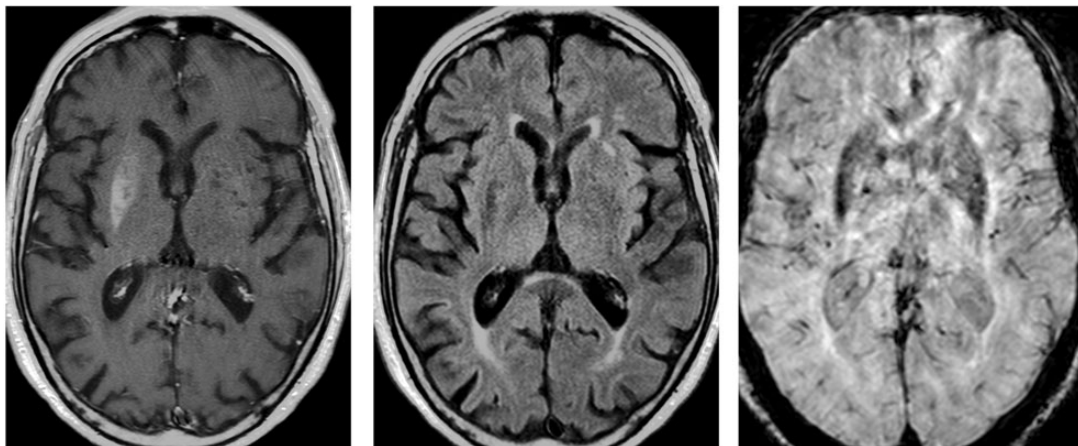


Figure 3: Axial MR images showing (left) no further enhancement of hyperintensity on post-contrast  $T_1$ -weighted scan, (middle) moderate to marked hypointensity on  $T_2$  fluid-attenuated inversion recovery scan and (right) mild susceptibility on susceptibility-weighted (VEN BOLD) scan.

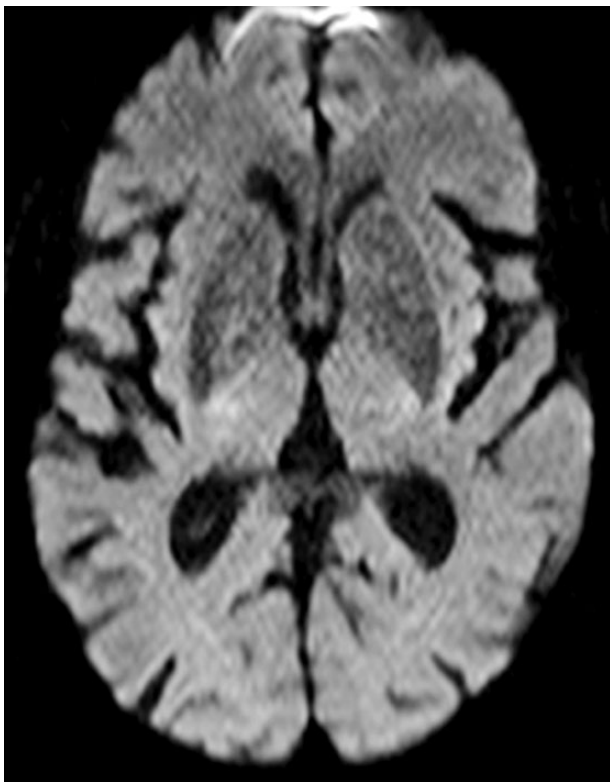


Figure 4: Axial diffusion-weighted MR imaging sequence showing no evidence of restricted diffusion.

bilateral ballismus in 11% of cases and facial involvement in 29% of cases [3]. It has also been reported to cause stereotypies [5].

The pathophysiology is unclear, but it is thought that the common mechanism for hemiballismus is impaired globus pallidus firing and subsequent loss of inhibition of thalamocortical pathways, causing excessive movement [1]. A number of other basal ganglia structures modulate globus pallidus firing including the subthalamic nucleus, caudate and putamen. Hyperglycaemia, in particular, is associated with putaminal lesions on MR imaging. Basal ganglia structures may be especially vulnerable to vasogenic oedema, excitotoxicity or anaerobic respiration in hyperosmolar hyperglycaemic states.

Most cases of hyperglycaemia-induced hemiballismus recover fully within months [3]. Symptomatic treatment may be provided by tetrabenazine, and refractory hemiballismus due to stroke has been treated with deep brain stimulation [6].

In conclusion, our report shows that corticosteroids can precipitate hemiballismus, which is most likely due to steroid-induced diabetes mellitus and a hyperosmolar hyperglycaemic state.

No ethical approval was required for this case report. Fully informed written consent was obtained from the patient in this case report in accordance with our local health district guidelines. K.C. and M.K. are guarantors for this case report.

## SUPPLEMENTARY MATERIAL

Supplementary material is available at *Oxford Medical Case Reports* online.

## CONFLICT OF INTEREST STATEMENT

None declared.

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