Heliyon 10 (2024) e26217

Contents lists available at ScienceDirect

Heliyon

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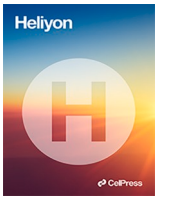

Case report

Gluteal compartment syndrome secondary to prolonged immobilization following carbon monoxide poisoning associated with leukoencephalopathy: A case report

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ARTICLE INFO

Keywords:
Case report
Gluteal compartment syndrome
Sciatic nerve palsy
Carbon monoxid poisoning and prolonged immobilization

ABSTRACT

Introduction: Gluteal compartment syndrome (GCS) is a rare diagnosis that results mostly from a non-traumatic etiology. We describe a case of a gluteal compartment syndrome, complicated with sciatic nerve palsy.

Case presentation: This paper reported a case of gluteal compartment syndrome caused by prolonged immobilization due to carbon monoxide poisoning. The case was complicated with sciatic nerve palsy and a sequala of leukoencephalopathy.

Discussion and conclusion: This case report highlighted the importance of having a high suspicion for gluteal compartment syndrome in patients with history of lying down with prolonged immobilization. The diagnosis can be made solely on clinical examination and a fasciotomy must be performed with no delay.

1. Introduction

Compartment syndrome (CS) is surgical emergency in which there is increased pressure within a limited place such as a fascial or osteofascial compartment, leading to local muscle ischemia and necrosis, with surgical decompression and fasciotomy being the gold standard treatment [1–3]. Multiple causes of this condition have been reported in the literature with trauma being the most common cause. However, multiple metabolic conditions such as diabetes mellitus, hypothyroidism, leukemia infiltration, nephrotic syndrome, rhabdomyolysis, use of anticoagulation and drug abuse resulting in prolonged immobilization, have been associated with the development of compartment syndrome [4–9].

Gluteal compartment syndrome (GCS) is a rare diagnosis and results mostly from a non-traumatic case such as prolonged surgery, substance abuse, or drug overdose [9–16]. Carbon monoxide poisoning as a cause of gluteal compartment syndrome is rarely reported in the literature [17,18]. We present an interesting case of a gluteal compartment syndrome, complicated with sciatic nerve palsy, caused by prolonged immobilization due to carbon monoxide poisoning associated with sequala of leukoencephalopathy changes.

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https://doi.org/10.1016/j.heliyon.2024.e26217
Received 24 December 2022; Received in revised form 7 February 2024; Accepted 8 February 2024
Available online 20 February 2024

2. Case presentation

A 40-year-old slim healthy female, working as a housemaid, was brought to the emergency department (ED) complaining of drowsiness and left hip pain which started in the afternoon one day following the use of burning charcoal in a closed room to keep herself warm at night. She woke up, after being down for over 16 hours, with severe left hip pain, which was associated with weakness and numbness. The patient denied any history of trauma, smoking, substance abuse, or medication administration.

Upon ED presentation the patient was found to be drowsy, but oriented to time, place, and person with Glasgow Coma Scale (GCS) of 15/15 with reactive pupils, her heart rate was 110 beats/minute, and Oxygen saturation on room air was 99% with normal respiratory rate and normal blood pressure and body mass index (BMI) of 23.2. Supplemental 100% oxygen was administered, which improved the patient's drowsiness.

Local examination of the left hip showed firm diffused swollen left gluteal area with intact skin [Fig. 4]. Patient complained of pain on passive movement of the hip. Stryker pressure gauge was not immediately available to record compartment pressure.

Compared to the contralateral side, the thigh circumference was greater by 3 cm. Neurovascular examination of the left lower limb showed decreased power of left hip and knee flexors and extensors with power 4/5, while ankle dorsiflexion, ankle plantarflexion and extensor halluces longus power were 0/5 with absent sensation over the dorsum of the foot.

Initial investigations showed: White Blood Cells (WBC): 21.0 × 10^3/μL, Hemoglobin (Hb): 18.6 g/dL, Potassium: 4.5 mmol/L, C-Reactive Protein (CRP): 100.6 mg/L, Procalcitonin: 1.23 ng/mL, Carboxyhemoblobin (CoHb): 1.8 %, Lactic acid (LA): 3.20 mmol/L, Serum creatinine: 81 μmol/L, Deranged liver enzymes – ALT: 571 U/L, AST: 952 U/L, Elevated Serum creatinine kinase (CK): >22000 U/L, and Serum Myoglobin: 12,911ng/mL suggesting rhabdomyolysis. Blood cultures ruled out infection. Radiographic examination of the left lower extremity ruled out any obvious fractures. Due to intermittent drowsiness of the patient, Computerized tomography (CT) scan of the head was done which showed hypodensities within the globus pallidus of both lentiform nucleus [Fig. 1].

Left gluteal ultrasound (USG) showed fluid collection in the posterolateral aspect of left thigh, in the intermuscular plane, measuring 3.1 × 0.5 cm [Fig. 2].

The patient was initially managed with conservative management, hydration with close monitoring of the thigh and gluteal compartments in addition to close assessment of the neurovascular status as the patient wanted to observe for improvement before surgical intervention [Fig. 3].

On reassessment, the patient's drowsiness improved. However, the left lower limb did not show any clinical improvement. Repeat USG of left gluteal region showed no significant changes. Based on the patient's history and assessment, the diagnosis of left gluteal compartment syndrome was made. Hence, the patient underwent fasciotomy of the left gluteal region and upper thigh.

Intra-operatively, the patient was positioned in right lateral decubitus position. Kocher Langenbach approach was utilized [Fig. 5]. The subcutaneous tissues and gluteal muscles appeared edematous with dark red discoloration of gluteus maximus and gluteus medius

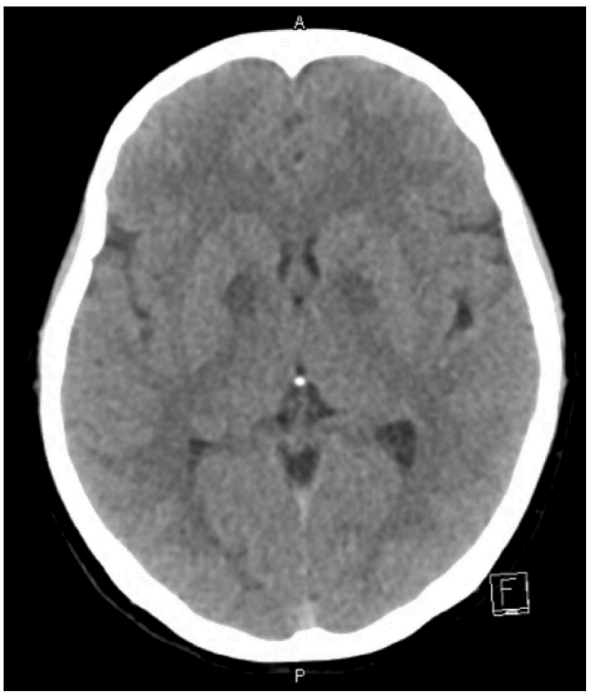

Fig. 1. Computerized tomography (CT) scan of the head showing hypodensities within the globus pallidus of both lentiform nucleus.

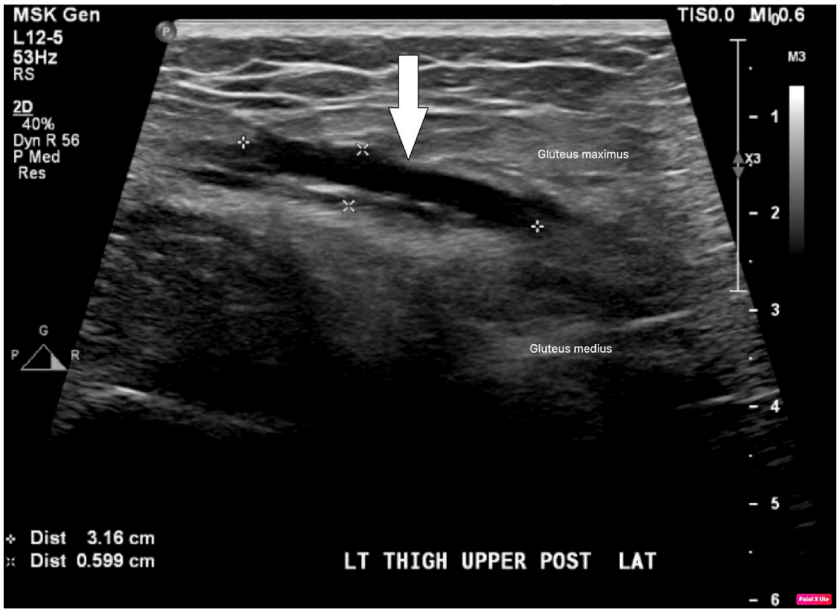

Fig. 2. Ultrasound image of left gluteal region showing a collection of 3.1 × 0.5 cm in the intermuscular plane.

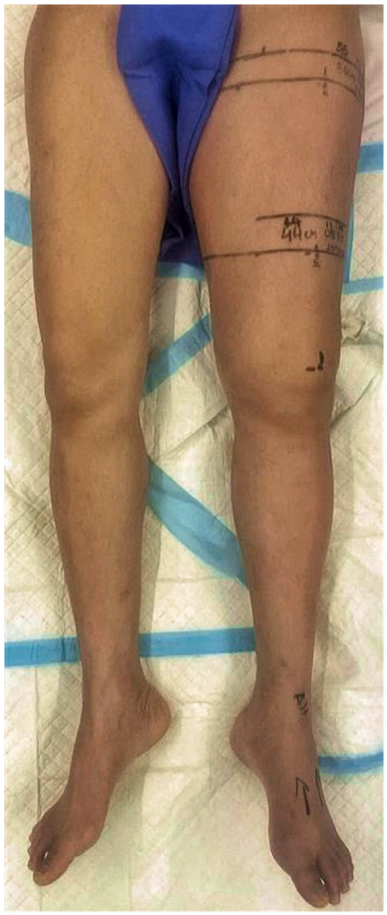

Fig. 3. Bilateral lower limbs comparing the Left thigh swelling with the uninvolved right thigh (Note the markings on the left thigh made for uniform serial thigh circumference measurements).

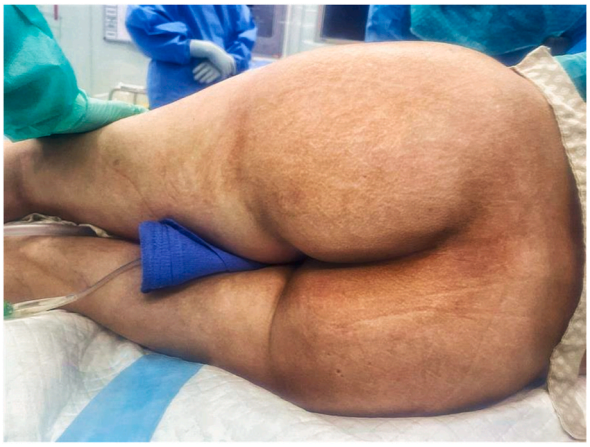

Fig. 4. Bilateral gluteal regions (Notice the swelling in the left gluteal region).

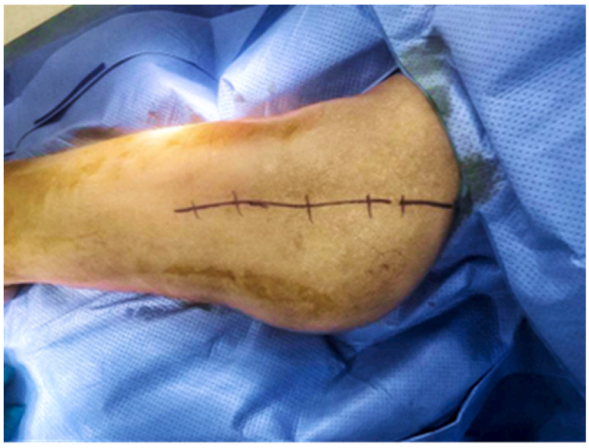

Fig. 5. Left thigh lateral incision with slight curve posteriorly.

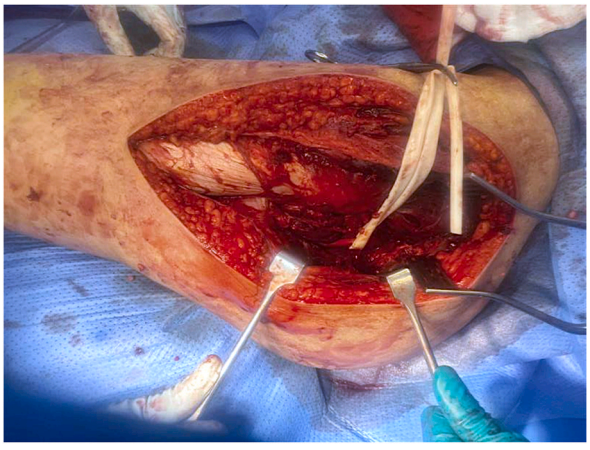

Fig. 6. Dark red discoloration of the Gluteus Medius and Gluteus Maximus muscles, with contused sciatic nerve.

muscles.

[Fig. 6]. There was hematoma surrounding the sciatic nerve and it was evacuated while protecting the sciatic nerve. Release of tensor fascia lata, Gluteus maximus and Gluteus medius was done by incising the intermuscular fascia and passing between the

intermuscular planes.

Fasciotomy and decompression of the anterior and posterior thigh compartments was done. The surgical wound was irrigated. Skin and subcutaneous tissue were partially approximated using non-absorbable monofilament nylon sutures [Fig. 7]. Dressings included moist normal saline gauze.

Postoperatively the left lower limb was positioned in hip extension and knee flexion to relax the sciatic nerve. On postoperative day one, the swelling was reduced. Sensation over the dorsum of the foot was still diminished with flickering movement of the toes. The power of dorsiflexion in ankle and toes improved to 1/5. Dressing was soaked with serosanguinous discharge and changed. On postoperative day 2, the thigh swelling had improved significantly allowing bedside fasciotomy wound closure. Then, the patient started to regain her foot sensation.

On the fourth postoperative day, the labs improved – Potassium: 3.7 mmol/L, ALT: 324 U/L, AST: 457 U/L, CK: 14,261 U/L and myoglobin: 167 ng/mL. On postoperative day seven, wound closure was completed with nylon 0 monofilament sutures. On post-wound closure day one, toes dorsiflexion power improved to 2/5, ankle plantar flexion power was 2/5, and ankle dorsiflexion power was 1/5. Patient started ambulation with left lower limb partial weight bearing.

On post-wound closure day three, the patient had improved sensation over the heel and medial arch, power of both the ankle and toes dorsiflexion improved to 3/5. The patient was clinically doing well, hence discharged. An early clinic follow-up and physical therapy appointments were arranged. However, the patient missed her appointments.

Two weeks after being discharged from hospital, the patient came to outpatient clinic, the surgical wound was healed, and the sutures were removed. The left foot sensation was intact, with ankle dorsiflexion and plantarflexion power of 3/5, and full power of 5/5 in the knee flexion and extension.

Seven weeks following the incident, the patient was brought to the emergency department with complaints of personality changes, memory loss and drowsiness. On examination, she was found to be conscious, oriented to place and person, while she appeared lethargic. She had hypokalemia (3.3 mMol/L), which was corrected after administration of 30 mEq potassium chloride. While the rest of her lab results were within the normal.

She was admitted under the medical team and underwent investigation including brain magnetic resonant imaging (MRI), which revealed hypoxic leukoencephalopathy in bilateral centrum semiovale and periventricular area, globus pallidum. The patient was diagnosed as a case of Carbon monoxide poisoning - neuropsychiatric sequelae. The patient was asymptomatic when assessed by the medical team. However, in absence treatable symptoms, she was discharged without medications.

During the second month after the operation, she was seen twice in outpatient physiotherapy department two weeks apart. The left ankle and toes dorsiflexion power improved to 4/5. A month later, the patient travelled to her home country.

3. Ethical approval

The reported case was approved by the Medical Research Centre at Hamad Medical Corporation, Doha, Qatar. Reference number: MRC04-22-831.

4. Informed consent

A written informed consent was obtained from the patient for publication of this case report and it's images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

4. Discussion

The gluteal muscles are enclosed by the fascia lata of the thigh, which splits the gluteal region into three distinctive compartments. It surrounds the gluteus maximus muscle posteriorly and the tensor fascia lata muscle anteriorly, whereas the combined fascia between these muscles overlies the gluteus medius and minimus. This fascia doesn't contain the sciatic nerve within it except in cases where the nerve perforates the piriformis muscles, however the nerve is vulnerable to compression by the swelling of these adjacent muscles leading to neurological compromise [19,20]. These muscles function as three separated compartments, hence it is crucial when surgical treatment is considered adequate decompression of each compartment separately should take place [21].

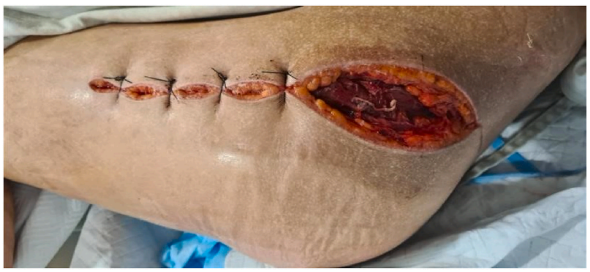

Fig. 7. Postoperative image after wound approximation.

Atraumatic gluteal compartment syndrome (GCS) is rare occurrence, it is hypothesized that prolonged pressure due to resting on hard uneven surfaces in an unusual position can lead to gluteal muscles swelling and edema, resulting in increased intra-compartmental pressure [22]. This can occur in multiple conditions where the patient consciousness or orientation has been altered, such as drug abuse, alcohol intoxication, carbon monoxide poisoning, and surgical positioning [10,15–18,23]. Due to the large muscle mass GCS is frequently accompanied by crush syndrome, which describes systemic manifestations of rhabdomyolysis and multiple organ failure alongside compartment syndrome [3,11,12,14,24,25]. Therefore, when it is diagnosed or in question, laboratory investigation including renal function, CPK, electrolytes, and electrocardiography should be conducted to rule out crush syndrome, as early diagnosis can allow adequate fluid resuscitation to prevent secondary acute renal failure [26,27].

Given the seriousness of GCS it is crucial to identify early and late manifestations. Diagnosis can be based on clinical signs. Usually, the patient will have pain out of proportion, pain with passive stretch, localized hard swelling, and may have associated peripheral neuropathy [28]. In doubtful cases, diagnosis can be made by measurement of the compartment pressure utilizing a wick catheter [28,29]. Hence the nervous system is the most susceptible to hypoxia, a compartmental pressure capable of affecting tissue perfusion is usually within 30 mm of Hg of diastolic pressure. Hargens et al. showed that the time required to produce peripheral nerve conduction block is inversely proportional to the intra-compartmental pressure and usually this occurs when the difference between the compartment pressure and the diastolic pressure difference is less than 30 mm of Hg [28,30,31].

Our patient had a history of CO intoxication and presented as a case of gluteal compartment syndrome with peripheral neuropathy. This presentation can be multifactorial, the contributing factors could be CO intoxication secondary to its effect on skeletal muscles and prolonged immobilization while lying down. Carbone monoxide effect on skeletal muscles has been reported in the literature. It usually leads to local muscle swelling, which may be the cause of increased the intra-compartmental pressure. This in turn will cause tissue hypo-perfusion and muscle necrosis and eventually rhabdomyolysis occurs, and patient might develop acute renal failure [17,18]. Peripheral neuropathy is a rare associated complication, and its prevalence has been reported to be 0.84% in 2360 patients diagnosed with Carbon monoxide intoxication [32].

This has been contributed to multiple causes including toxicity to CO intoxication itself, nerve compression secondary to local muscles swelling, and ischemia to the nerve due to hypoxia. However, in most cases neurological deficit is known to show complete recovery within a few months [32,33]. On the other hand, there have been reports of peripheral neuropathy secondary to CO intoxication with no local muscles swelling or compartment syndrome [34].

Carbon monoxide-induced rhabdomyolysis leading to compartment syndrome in the buttocks is rare, with few cases reported in the literature [35–37]. Due to its highly deleterious effects, our brain tissue is sensitive to carbon monoxide toxicity. Carbon monoxide poisoning on brain tissue commonly manifests as bilateral damage to the basal ganglia and cerebral cortex [38]. Lee et al. reported a patient with rhabdomyolysis and gluteal compartment syndrome after carbon monoxide intoxication. MRI of the brain was done for this patient and showed bilateral hyperintensities in the globus pallidus and cerebellum [Figs. 8 and 9]. To the best of our knowledge, no other studies in the orthopedic literature reported on carbon monoxide poisoning on brain tissue in patients with gluteal compartment syndrome. Therefore, improved awareness of the neurological sequelae associated with carbon monoxide is paramount to provide early management and follow-up for such patients.

This case report is a rare condition, surgeons and physicians commonly do not have enough awareness of such condition, that is why usually the diagnosis is delayed or even mistaken for gluteal abscess or DVT [14]. The gold standard management of compartment syndrome is urgent surgical fasciotomy and decompression; in case of GCS there is no defined standard surgical approach, regardless of the approach the main goal of the surgery is to decompress all three compartments with or without sciatic nerve neurolysis to prevent post fasciotomy inflammatory adhesions [12,28,39,40].

Funding statement

The Open Access funding was supported by the Qatar National Library. This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Data availability statement

Data will be made available on request.

Additional information

No additional information is available for this paper.

CRediT authorship contribution statement

Mohammad Shujauddin: Writing – review & editing, Writing – original draft, Data curation, Conceptualization. **Ashraf T. Hantouly:** Writing – review & editing, Writing – original draft, Supervision, Methodology, Conceptualization. **Isam Moghamis:** Writing – original draft, Data curation. **Osama Alzobi:** Writing – review & editing, Writing – original draft. **Fuad Vayalil Mazhar:** Writing – review & editing, Supervision, Conceptualization. **Mohamed Maged Mekhaimar:** Writing – review & editing, Supervision, Conceptualization.

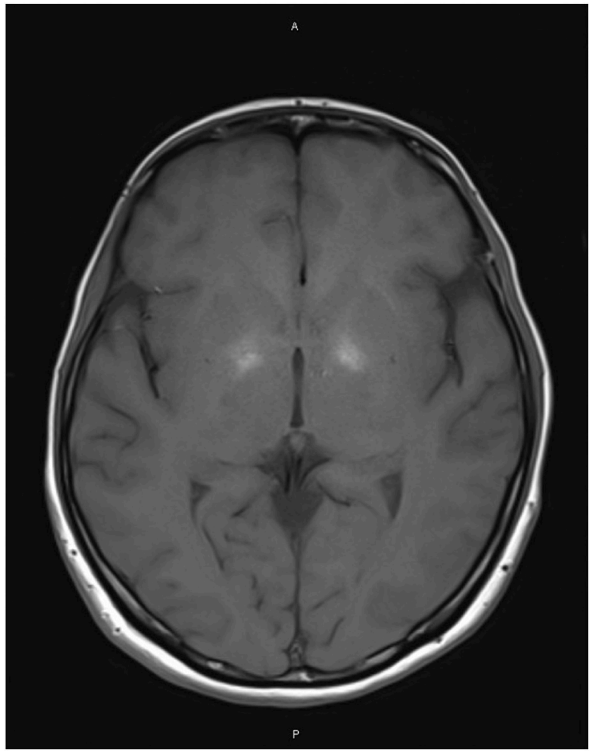

Fig. 8. MRI brain T2 sequence showing hyper-intensities in the centrum semiovale and periventricular region.

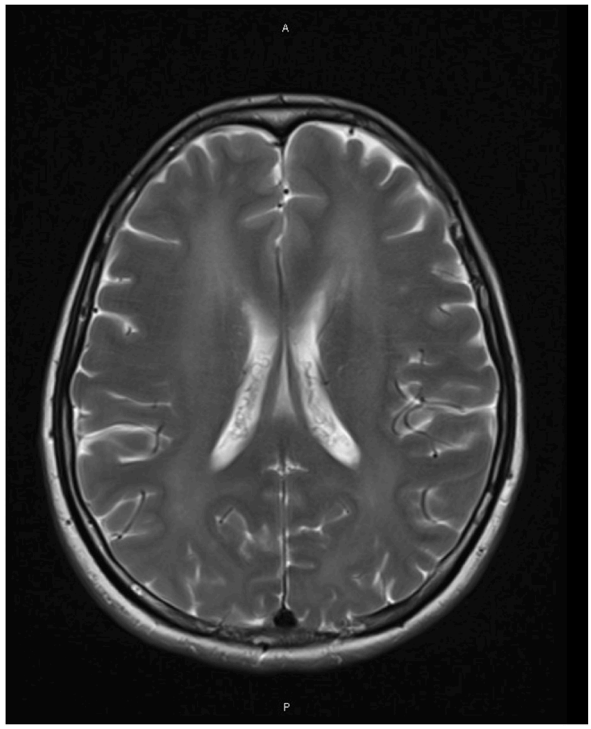

Fig. 9. MRI brain T1 sequence showing hyper-intensities in the bilateral globus pallidum.

Declaration of competing interest

The authors declare the following financial interests/personal relationships which may be considered as potential competing interests:

Mohammad Shujauddin reports article publishing charges was provided by Qatar National Library.

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