Use of Robotic Gait Training in Hyperglycaemia-Induced Central Pontine Myelinolysis: A Case Report

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ABSTRACT

Objectives: To describe a rarely-seen cause of central pontine myelinolysis (CPM), and the benefits of application of robot-assisted gait retraining (RAGT) in a model of impairment-directed rehabilitation in the management of gait ataxia.
Study design: Case report.
Setting: Inpatient rehabilitation unit of tertiary hospital, Singapore.
Subjects: A 69-year-old male who presented with giddiness, cognitive impairment, and gait ataxia.
Methods: The patient underwent magnetic resonance imaging of the brain which revealed pontine hyper-intensity suggestive of CPM. His diagnostic work-up revealed significant hyperglycaemia.
Results: He underwent treatment for his metabolic derangements and was referred for rehabilitation. We added robot-assisted gait training in the treatment of his ataxia. He was able to ambulate independently subsequently and was discharged uneventfully.
Conclusion: CPM is an uncommon but disabling condition typically seen in rapid correction of hyponatraemia. In our patient this was caused by hyperglycaemia, which likely drives the same osmotic derangements that leads to the dramatic impairments that characterise this condition. Rehabilitation of hyperglycaemia-induced CPM has yet to be described but the condition is becoming more frequently recognised in clinical practice. In addition, given the benefits of RAGT in gait ataxia, there is great potential in the role that RAGT can play in the exploration of best practices beyond CPM in a model of impairment-directed therapy in future.

Keywords: myelinolysis, central pontine, robotics, gait disorders, rehabilitation

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Introduction

Central pontine myelinolysis (CPM) is an uncommon but potentially-fatal presentation of the osmotic demyelination syndrome characterised by oedema and apoptosis of pontine oligodendrocytes resulting from sudden osmolar shifts, such as from rapid correction of hyponatraemia though other associations include renal failure and alcoholism.[1,2] Clinical manifestations, severity, and outcomes are heterogeneous.[1] Hyperglycaemia-induced CPM has only been reported four times prior, and with gait disturbance as a feature.[2-5] We highlight the rare manifestation of hyperglycaemia-induced CPM, and explore the possible role that robot-assisted gait training (RAGT) can play as part of patient-centred and impairment-directed rehabilitation in CPM patients.

Case history

We present a case of a 69-year old Chinese male taxicab driver who was pre-morbidly well, community-ambulant, and lived alone, with a past medical history of hypertension, hyperlipidaemia, diabetes, and transient ischemic attack (TIA). He presented with a progressive one-month history of non-specific leg weakness of gradual onset, which resulted in him becoming homebound and needing to furniture-cruise. Worsening giddiness and swaying while walking led to his attendance at our emergency department. Prior to admission, he had stopped taking his oral diabetic medications for two months as he had run out of supply. Physical examination revealed mild right-sided hemiparesis and gait ataxia. He was forgetful but had normal affect, scoring nine out of ten on the Abbreviated Mental Test. Cranial nerve and sensory examinations were normal.

An urgent magnetic resonance imaging (MRI) scan of the brain on admission was performed for suspected stroke. No infarcts were observed, but new isolated T2-weighted signal hyper-intensity was observed in the central pons (Figure 1) suggestive of CPM. The imaging changes had not been observed a year before when he was diagnosed with TIA. His blood tests including serum sodium were unremarkable except for a serum glucose of 436 mg/dL (24.2 mmol/L) and HbA1c of 138 mmol/mol (14.8%). He was neither dehydrated nor hyponatraemic, and not in in diabetic ketoacidosis. A diagnosis of CPM was made on the basis of his clinical history, lack of physical findings to support alternative differential diagnoses, as well as the severity of his biochemi-

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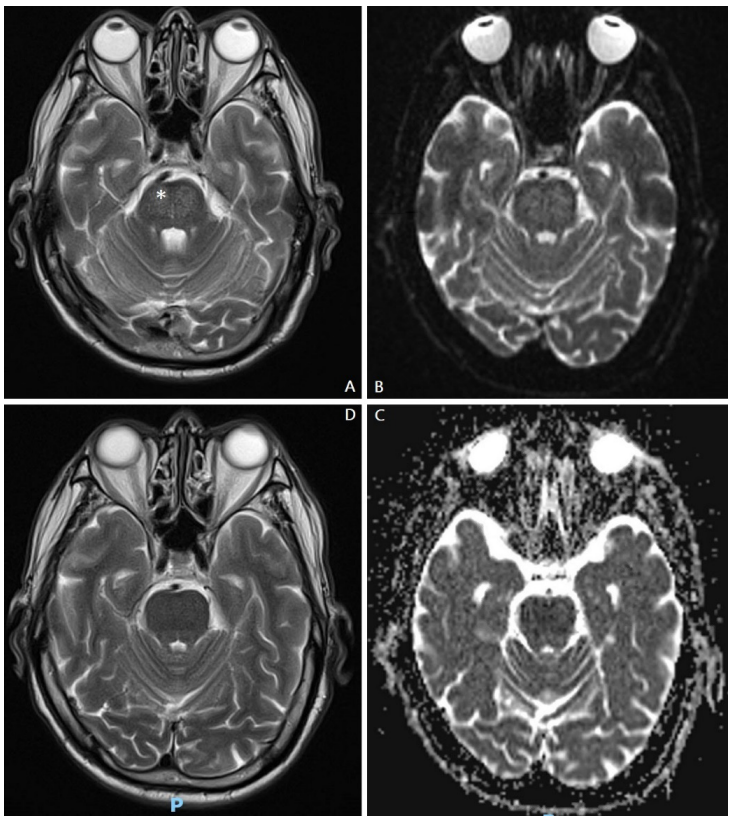

Figure 1. MRI scans of the patient's brain showing (A) T2-weighted signal hyper-intensity in the central pons suggestive of CPM; (B) similar increased signal shown in diffusion-weighted imaging (DWI); (C) signal iso-intensity on apparent diffusion coefficient (ADC) mapping which ruled out an infarct; (D) comparison with the patient's previous MRI findings a year before, showing normal T2-weighted signal, when he presented for a separate neurological complaint (TIA); * represents the lesion of interest.

cal and radiological findings.

He received insulin and was referred for rehabilitation after two days, with transfer to our unit on the fourth day of admission. With correction of his hyperglycaemia, both his weakness and memory had recovered (28/28 on the Mini-Mental State Examination (Chinese)), but despite two days of therapy in the general ward, his standing balance remained poor with retropulsion. He needed assistance to ambulate and perform lower body activities. Quality-of-life (QoL) assessment via the EQ-5D-5L instrument revealed problems in the mobility, self-care, and usual activities domains (health state 23311). He self-scored 50 on the visual analogue scale (EQ VAS).

He underwent conventional physiotherapy and occupational therapy for gait and cognitive training. Conventional gait training in our unit includes both static and dynamic stepping exercises as well as balance perturbation tasks, with promotion to functional walking for performing activities of daily living or going to the inpatient gym. Cognitive training is titrated towards the patients' specific deficits - in his case this memory training through simulation of home tasks.

To address his main impairment of gait ataxia we prescribed overground RAGT with the Andago® V2.0 (Hocoma AG, Switzerland) on the basis of facilitating massed practice. We referred him to ophthalmology for diabetic eye screening and treatment of cataracts. We incidentally diagnosed osteoporosis on bone mineral densitometry and counselled him for anti-resorptive treatment.

He remained clinically-stable and participated well in therapy. After a week in our unit which incorporated seven daily 30-minute sessions of RAGT (Figure 2), he made significant gains in both his gait speed and stability, and subsequently mobilised independently. His functional ambulation category had improved from 1 on admission, to 2 on transfer to rehabilitation, to 5 at the end of his RAGT training. EQ VAS remained at 50 but his health state had improved to 11111. He was discharged home to independent living with outpatient follow-up. At the time of discharge his other mobility indicators were: 315 m on the six-minute walk test (level of assistance 7, no assistive devices, increased from 240 m when measured earlier during his stay in our unit), 10 seconds to complete the Timed Up and Go test, and a score of 53/56 on the Berg Balance Scale (nearly-full functional balance). These had not been charted on admission as they were not routine measurements in our unit for a non-stroke patient.

Discussion

The clinical trajectory of hyperglycaemia-induced CPM varies, from inpatient recovery to intensive care admission, though symptom resolution typically occurs over one to eight weeks.[2,3] The intensity and method of management in such patients also remains heterogeneous, ranging from impairment-directed rehabilitative therapy to purely supportive manage-

Figure 2. Our patient using the robotic gait trainer on the day of his discharge.

ment followed by referral to nursing facility.[3] In our patient his gait improved over the course of a week which was thankfully considered short and he was able to go home.

Just as in treatment of hyponatraemia where there is a need to avoid rapid overcorrection of the patient's serum sodium, we found it important to be wary of large corrections in his diabetic control. Although fluctuations occurred these were generally acceptable and his clinical condition did not deteriorate.

Multi-disciplinary management of CPM is crucial especially in patients with more severe features, to facilitate impairment-directed interventions such as tone management, wheelchair customisation, and communication aids.[6] Our patient's main issues were gait ataxia and memory dysfunction.

His rapid cognitive recovery after reversal of hyperglycaemia suggests delirium rather than a true neurocognitive manifestation of CPM. Management of gait ataxia traditionally comprises balance training, developing postural control, specific gait training, and the use of compensatory orthotics and aids, though treadmill training has not previously shown significant benefits.[7] Using RAGT in the management of gait ataxia in CPM is novel – as an adjunct to conventional physiotherapy in stroke there is moderate evidence that it increases the chance of independent walking (number needed to treat = 7).[8] RAGT also improves gait and endurance in other cardiopulmonary and musculoskeletal conditions. Although there are no precedents in CPM, we applied this understanding in our approach to his gait disturbance.[9] The Andago® is described as a patient-guided suspension system under new classifications of lower limb rehabilitation robotics - these confer mobility to harnessed patients who can then safely practice free-range walking and make errors in a falls-safe environment.[10] The robot leverages on the concepts of intrinsic learning and massed practice to facilitate and accelerate the process of gait retraining in all different phases of gait – when tethered to the robot patients are able to increase the number of steps taken during a therapy session and recognise their own gait abnormalities. The addition of a safety harness helps to prevent falls and promotes confidence, and the mobility of the device allows patients to explore the environment, allowing "real walking", which can motivate participation.[11] Finally, the device also features partial weight supports which allow patients with truncal and lower limb weakness to use it for gait practice. In our patient this translated into a 32% increase in walking speed and also improved balance to the level of unsupervised community mobility.

In CPM there often is a need to evaluate dysphagia and speech but he demonstrated sufficient function in this area and hence we focused more on the gait ataxia.[6] Finally, screening for mood and QoL is essential due to the various activity and participation barriers that can arise – these were addressed through improvement of his walking function and although his gait speed was still considered slow (0.53 m/s), he had regained confidence in his own mobility by discharge.

CPM is increasingly becoming recognised in patients at earlier, less-lethal stages and consequently more of such patients may require rehabilitation services.[1] In addition, given the benefits of RAGT in a model of impairment-directed rehabilitation, there is great potential in its application to a wide variety of other neurological conditions beyond CPM with manifestations of gait ataxia. While we continue to develop best practices in this field, it may prove worthwhile to explore the burgeoning role that robotic therapies could yet play.

Disclosures

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Written informed consent was obtained from the patient for publication of this case report, available on request.

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