



Case Report

Cerebral Venous Thrombosis in Multiple Sclerosis After High Dose Intravenous Methylprednisolone Treatment: a Case Report

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Abstract

Cerebral venous thrombosis is a rare phenomenon that presents with a variety of manifestations which may be associated with different etiologic factors. Here we present a 24 years old woman with a history of known multiple sclerosis and recent intravenous methylprednisolone treatment and aim to discuss possible relationship between multiple sclerosis and cerebral venous thrombosis in current report.

Keywords: Cerebral VenousThrombosis, Multiple Sclerosis, Methylprednisolone, Anti Coagulant.

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Introduction

Cerebral sinus venous thrombosis is an uncommon condition with different causes and variable clinical presentation that affect cerebral venous drainage [1]. Several factors can lead to cerebral venous thrombosis including mechanical injuries, infectious, inflammatory, hormonal and hematologic disorders [2]. Relation of cerebral venous thrombosis and Multiple Sclerosis documented in patients with history of lumbar puncture and intravenous high dose methylprednisolone treatment in some studies [3, 4]. Here we present a case of definite multiple sclerosis that developed cerebral venous thrombosis after high dose intravenous corticosteroid therapy.

Case Report

A right-handed 24 years old woman with history of definite relapsing remitting MS for 4 years presented with numbness in lower limbs. After taking a precise history and detailed examination, brain MRI was performed and based on clinical and imaging findings the multiple sclerosis attack was confirmed so we initiated intravenous methylprednisolone at a dosage of 1 gr/day for 5 days. She had no history of known illness except multiple sclerosis from 4 years ago. There is no history of oral contraceptive consumption, previous corticosteroid intake (oral or intravenous) or lumbar puncture. In first visit she complained

numbness, paresthesia and reduced muscle strength in lower limbs. Systemic examination was unremarkable. Cranial nerve including fundoscopic exam and mental status was normal. We detected 3.5 muscle force in both legs. Deep tendon reflexes were hyperactive in patellar and Achilles tendons. Plantar reflexes were extensor in both sides. Sensation of pain and light touch was reduced in lower extremities. Cerebral MRI revealed hyper intense plaques on T2 and FLAIR sequences in periventricular and cervical spine with one ring enhancing lesion adjacent left posterior ventricle. Cervical plaques were non-enhancing (Figure 1). Lab data were normal.

The patient set on intravenous methylprednisolone treatment at a dosage of 1gr/day for five days (with divided dosage twice a day). Two days after treatment completion she complained severe headache with superiority in occipital regions accompanied nausea and vomiting without proper improvement with supportive treatment next day seizure complicated the patient general condition. Considering the cerebral venous thrombosis possibility brain MRI and MRV performed immediately. Thrombosis in superior sagittal sinus was determined (Figure 2, 3).

Therefore anticoagulant treatment initiated with the diagnosis of cerebral venous thrombosis. After 4 days headache was improved significantly and she discharged with oral anticoagulant. All lab tests that may precipitate the patient to

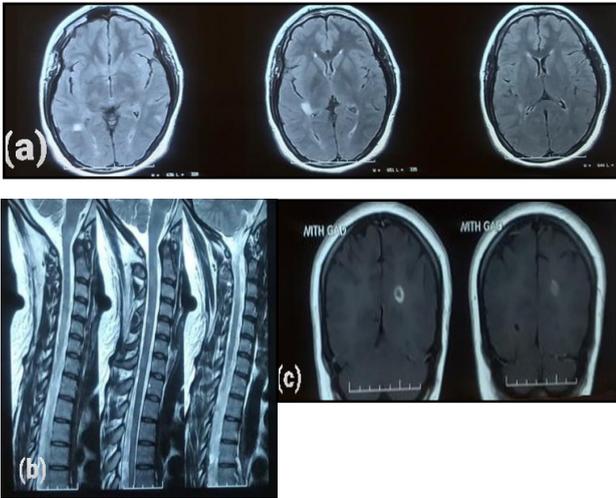


Figure 1. (a) Axial Flair MRI reveals hyper intense periventricular plaques in favor of MS lesions (b) cervical MRI demonstrates hyper signal plaques (c) MRI with gadolinium and gadolinium enhancing lesion.

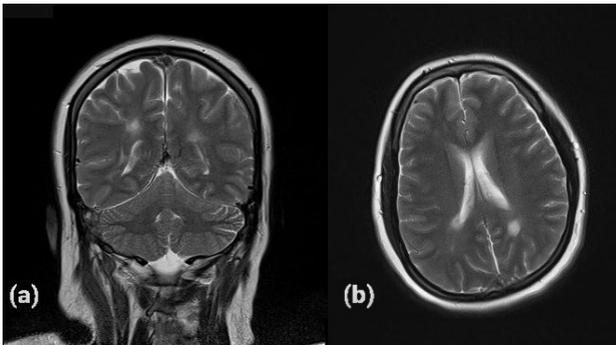


Figure 2. a) Coronal T2 MRI demonstrates signal change in superior sagittal sinus. (b) Superior sagittal sinus hyper signal flow in axial view, T2.

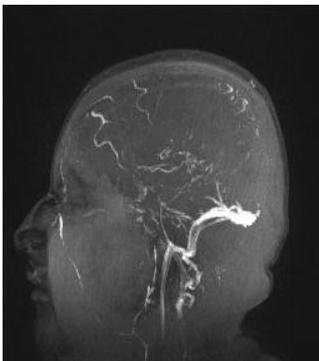


Figure 3. Increased signal in superior sagittal sinus in magnetic resonance venography.

thrombosis formation in venous sinuses were requested results were normal.

Discussion

Cerebral venous thrombosis (CVT) incidence is approximately 1-2% among all strokes and Young women are more disposable

to CVT, Based on thrombosis site symptoms are variable among patients, including raised intracranial pressure, headache, seizure and focal neurologic deficits [5]. There are a broad spectrum of CVT triggers such as infective causes, head injury, space occupying lesion, hormonal and endocrine causes, malignancies, severe dehydration, inflammatory diseases and several other causes. But one third of cases have no known trigger [6]. Here we discuss CVT and its possible causes in definite multiple sclerosis cases. CVT has an uncommon occurrence in multiple sclerosis patients; there is a possible relationship between lumbar puncture and treatment with high dose corticosteroid and increased risk of CVT development in multiple sclerosis patients [7]. Relation of Cushing syndrome , a state of high levels of corticosteroids in blood, and CVT development documented in some series, so we can conclude high dose corticosteroid can precipitate CVT occurrence in some patients [8].

The possible mechanism of clot formation in Cushing's patients can be explained as follows: hypercortisolism state associated with high levels of factor (F) VIII, IX and von Willebrand factor (VWF). Acute high-dose glucocorticoids accelerates synthesis of VWF and enhances the rapid activation of endothelial nitric oxide synthase (eNOS), which is a possible inhibitor of VWF secretion, overproduction of these procoagulatory factors make the vessels prone to clot formation, on the other hand glucocorticoids significantly increase levels of plasminogen activator inhibitor-1 (PAI-1), which can lead to fibrinogen diminished levels in serum so glucocorticoid-induced alterations in fibrinogen may contribute also to the thrombotic events [9].

The relation of CVT development and multiple sclerosis as an inflammatory disorder has not been proved in previous studies [10].

Therefore we can conclude in this case occurrence of CVT is associated with high dose of corticosteroid which can confirm by the followings: the patient developed headache immediately after end of treatment with corticosteroid, absence of any other trigger factor in our patient and proved association between high levels of corticosteroid and thrombosis considering the changes that corticosteroids make in serum coagulatory system and recorded DVT or pulmonary thromboembolism in Cushing's patients in recent studies. All of this findings points that intravenous methylprednisolone is main cause of thrombosis formation in superior sagittal sinus in this case.

Conflict of Interest

The authors declare that there is no conflict of interest.

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None.

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