A Rare Case of Spontaneous Subdural Hemorrhage in Dengue Fever That Mimics a Tumor on MRI: A Case Report

Deepak Ranade, MD., Ankit Patel, MD., Bhagirath More, MD., Apurva Lachake, MD.

Department of Neurosurgery, Dr. D.Y. Patil Medical College, Hospital and Research Centre, Pimpri, Pune, India.

ABSTRACT

Background Date: In all cases of spontaneous spinal hemorrhage (epidural, subdural, and intramedullary hemorrhage), spinal subdural hemorrhage is extremely rare. Bleeding diathesis is a commonly associated complication of Dengue fever along with multisystemic complications, such as renal toxicity, heart failure, shock, and electrolyte abnormalities. Dengue fever presenting as a neurological complication is extremely rare, <1% of patients.

Study Design: A case report.

Purpose: To report a rare case of dengue fever with spontaneous subdural hematoma (SDH) and subarachnoid hemorrhage (SAH) in the spine.

Case Report: A 52-year-old female patient presented with acute onset of progressive bilateral lower limb weakness accompanied with difficulty in micturition and headache for a 5-day duration. She also had a history of fever prior to lower limb weakness and headache. Clinical examination revealed grade 2 motor power in both lower limbs, absent deep tendon reflexes, and equivocal Babinski’s reflex. There was no definite sensory deficit.

Results: Patient improved postoperatively after hematoma evacuation.

Conclusion: Spontaneous SDH with SAH can be a rare presentation of dengue fever. Prompt intervention is very important to prevent irreversible neurological deficits. (2021ESJ241)

Keywords: SDH, SAH, dengue fever, spine, conus, hematoma.
INTRODUCTION

In all cases of spontaneous spinal hemorrhage (epidural, subdural, and intramedullary hemorrhage), spinal subdural hemorrhage is extremely rare. Bleeding diathesis is a commonly associated complication of dengue fever along with multisystemic complications, such as renal toxicity, heart failure, shock, and electrolyte abnormalities. Dengue fever presenting as a neurological complication is extremely rare, <1% of patients. Several pathophysiological mechanisms have been hypothesized, including direct CNS viral entry, metabolic disturbances impairing CNS function, hemorrhage, CNS inflammation, and demyelination due to virus-generated autoimmune reactions.

In this report, we present an uncommon case of paraparesis caused by spontaneous SSDH and SAH as a presentation of dengue hemorrhagic fever.

Case Presentation

A 52-year-old female patient presented with sudden onset of progressive bilateral lower limb weakness accompanied by difficulty in micturition and headache for 5 days. She also had a history of fever prior to lower limb weakness and headache. She had been diagnosed with dengue fever 1 week prior to presentation. Clinical examination revealed grade 2 motor power in both lower limbs, absent deep tendon reflexes and equivocal Babinski's reflexes. There was no definite sensory deficit. Her hematological investigations revealed the following: Hb, 13.4; WBCs, 12900; PLT count, 88000. Her blood coagulation profile and biochemistry parameters were within normal levels to her age. Dengue IgM was positive. MRI spine revealed a well-defined intradural extramedullary mass size of $14 \times 10 \times 13$ (CC $\times$ AP $\times$ T) mms at the level of T12–L1 vertebra on the right anterolateral aspect of conus medullaris. It was compressing the conus causing adjacent cord edema. Radiologically, the lesion was hyperintense on T2W and isointense on T1W with no postgadolinium contrast enhancement. There were T2 hypointensity within the thecal sac and fluid-fluid levels due to subarachnoid hemorrhage (Figures 1,2A). A hemilaminectomy was performed, and after durotomy, minimal SAH was noted along with partially clotted blood in the subdural spaces that has been evacuated (Figure 3). Postoperative MRI confirmed the complete removal of hematoma (Figure 2B). Two weeks after surgery, her motor power had recovered to grade 5, along with a full recovery of bladder incontinence.

Figure 1. (A) Preoperative MRI lumbosacral spine T1WI axial and sagittal images showing heterogenous intensities lesion intradural with outer hyperintensity and inner hypointensity at T12-L1 level. (B) Preoperative MRI lumbosacral spine T1WI with IV gadolinium contrast injection axial and sagittal images, showing no contrast enhancement of the lesion.
Figure 2. (A) Preoperative MRI lumbosacral spine T2WI axial and sagittal images showing focal hyperintense lesion found at the level of T12 and L1 with cauda equina compression. (B) Postoperative MRI lumbosacral spine T2WI axial and sagittal images showing normal anatomy of the cauda equina and total lesion disappearance.

Figure 3. (A) Intraoperative microscopic view after the durotomy showing the SAH. (B) Intraoperative microscopic view showing SDH clots compressing the cauda equina.

DISCUSSION

This case illustrated a nontraumatic “spontaneous" SSDH with SAH associated with dengue fever in which thrombocytopenia was not severe. A review of the literature did not report any case of SSDH with SAH as a presenting feature of dengue fever. Extramedullary spontaneous SDH is commonly seen in the thoracic segment and usually presents with sudden back pain that radiates to the upper or lower extremities or the trunk. This causes sensory and motor deficits that are progressive and can be severe. The duration from the onset of back pain to the development of paraplegia is 10 to 26 hours. In general, the MRI is helpful
in terms of differentiating between tumors and vascular malformations as the underlying etiology. Usually, these subdural lesions are convex and crescentic in appearance and tend to be around the spinal cord with delineation of the dural sac and intact epidural fat. Upward and downward extension is frequent, with extension to the posterior cranial fossa in some cases. However, the unusual imaging appearance of this subdural lesion (focal and independent from the dura) misled the initial radiological diagnosis to be a spinal tumoral hematoma or spinal subdural mass apoplexy.

The exact pathophysiology of spontaneous SDH is still not clear. The spinal subdural space is avascular. This means that a hemorrhage in the subdural space might be secondary to a subarachnoid source and a rupture of the arachnoid membrane. The spinal SDH may persist (usually in a ventral portion) following rapid resolution of SAH by dilution with CSF.

The postulated mechanism is a sudden increase in the pressure of the intraspinal vessels consequent to an acute rise of intra-abdominal or intrathoracic pressure. Secondary to the increased pressure following minor spinal trauma or sudden physical activity, the inner luminal pressure of the venous structures located in the subarachnoid area increases. When there is a concomitant drop in the cerebrospinal fluid pressure below the intraluminal venous pressure, it results in the breach of the integrity of the valveless radiculomedullary veins, which traverses the spinal subarachnoid space.

However, a contradictory hypothesis exists. Morandi et al. emphasized that the thin and delicate extra-arachnoid veins in the inner surface of the dura would lead to a SDH. Hung et al. hypothesized that the rise in the intracranial pressure could also increase the shearing force between the spinal subarachnoid and the subdural spaces, causing the inner dura to tear and bleed. Moreover, Schwartz et al. described the acute spinal SDH as being extra-arachnoidal, adding that it appeared to be between two layers of the dura, which suggests that a potential intradural space could be the source of the bleeding.

CONCLUSION

Spontaneous SDH with SAH can be a rare presentation of dengue fever. Prompt intervention is very important to prevent irreversible neurological deficits. Acute neurological deficits in cases of dengue fever should raise a high index of suspicion to diagnose this relatively rare manifestation.

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النتائج:

يمكن أن يكون النزف العفوي تحت الام العنكبوتية و الام الجافية عرضًا نادرًا لحمى الضنك. التدخل الفوري مهم جدا لمنع العجز العصبي الذي لا رجعة فيه.