Journal of Neuroinfectious Diseases

Prasad A, et al., J Neuroinfect Dis 2016, 7:1
http://dx.doi.org/10.4172/2314-7326.1000203

Case Report Open Access

Acute Embolic Infarcts from *Alcaligenes Faecalis* Meningitis: A Case Report

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Rec date: Jan 06, 2016; **Acc date:** Feb 05, 2016; **Pub date:** Feb 08, 2016

Introduction

Cerebrovascular complications of meningitis include vasculitis, vasospasm, venous and arterial thrombosis, intracranial aneurysm formation, and rarely, subarachnoid hemorrhage [1-5]. Stroke may be the result of any one or combinations of these processes. In this case report, we describe a rare case of acute embolic infarcts from *Alcaligenes Faecalis* meningitis.

Case Description

A 44 y/o gentleman with past medical history of possible seizures not on any anti-epileptics came to the Emergency Department with headache, fever and confusion for the past two to three days. CT head did not reveal any acute intracranial process. The patient's labs showed leukocytosis with count of 20.6 and left shift. Soon after presentation, he had a witnessed generalized tonic, clonic seizure lasting 30 seconds. The patient was intubated for airway protection, placed on ASV ventilation mode and sedated with propofol. The patient was loaded with 1 gm of Levatiracetam and admitted to the Neuro-ICU service for further management.

The patient was noted to be febrile overnight. Blood cultures and lumbar puncture was performed and he was started on broad-spectrum antibiotics and acyclovir. The patient's respiratory status improved and he was extubated the following morning. CSF analysis revealed 73 leucocytes, 1580 erythrocytes with predominantly monocytes. CSF glucose was 36 and protein was 47. CSF cultures revealed growth of gram negative rod colonies which were later confirmed as *Alcaligenes Faecalis*. The patient was started on IV Meropenem for 21 days as per infectious disease recommendations. The patient on neurological examination was noted to be encephalopathic and had restriction of upward gaze.

CT angiogram of the head and neck revealed focal moderate stenosis of the left vertebral artery. MRI brain without contrast was performed and revealed foci of restricted diffusion within left inferior frontal lobe adjacent to the left frontal horn, the inferomedial right temporal lobe, and within the left thalamus extending to the left midbrain. The findings represented embolic infarcts.

A four-vessel cerebral angiography was performed to evaluate for CNS vasculitis. It revealed a 55 percent stenosis of left vertebral artery. An electroencephalogram was performed and did not reveal any epileptiform activity. Further hypercoagulable workup was negative apart from elevation of factor 8 activity. The patient was started on aspirin 325 mg for stroke prevention. The patient improved clinically and was discharged from the hospital to be followed with hematology for repeat Factor VIII Activity given that it had a twice fold increase in the acute phase of the stroke.

Discussion

The mechanism of stroke in this case includes thromboembolism in the setting of a severe, widespread vasculopathy due to *Alcaligenes faecalis* infection further complicated by stenosis of left vertebral artery. However, a primary source of infection was not identified. No prior case reports of *Alcaligenes faecalis* meningitis causing stroke were found in literature. Two fold increases in factor VIII activity raises the possibility of an underlying primary hypercoagulable state which may have been an additional contributing factor or a nonspecific finding. The role of *Alcaligenes faecalis* as a pathogen in meningitis and its cerebrovascular complications merits further study.

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