

Case Report

Spinal Cord Infarction with Markedly Elevated Protein in CSF Mimicking Inflammatory Disease

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Case Presentation

Information pertaining to the case was obtained via retrospective review of electronic medical records.

51 year lady with HTN, HLD, TIA, DM, multi vessel CAD s/p CABG on 6/20/16 followed by spinal infarct with resultant paraplegia and bowel bladder incontinence was transferred from Rehabilitation to ED with complaints of chest tightness across her entire chest. Pt denies having SOB and Pts chest pain resolved on its own. Neurology was consulted due to her paraplegia [1-4].

Neurological examination, there is a sensory level at T5 level. Patellar and Achilles reflexes absent, plantar response is mute bilaterally.

Investigation

Patient was evaluated with extensive work up for inflammatory cause.

Spinal tap showed 27 nucleated cell, lymphocyte 69% and 24 monocytes.

Protein 460, glucose 90, oligoclonal bands CSF 0, serum oligoclonal 2, VDRL CSF non reactive, no malignant cell seen. Serum NMO was negative WNV, EBV and VZV PCR, HSV 1 & 2 not detected. HIV



Figure 1: MRI T Spine DWI sequence with extensive DWI changes in lower cervical and extensive thoracic cord.

Abstract

Spinal cord infarction following cardiac surgery from hypoperfusion is one of the mechanisms for cord infarction. It can mimic NMO because of extensive cord involvement and acute presentation.

Keywords: Spinal cord infarction post CABG; Neuromyelitis optica

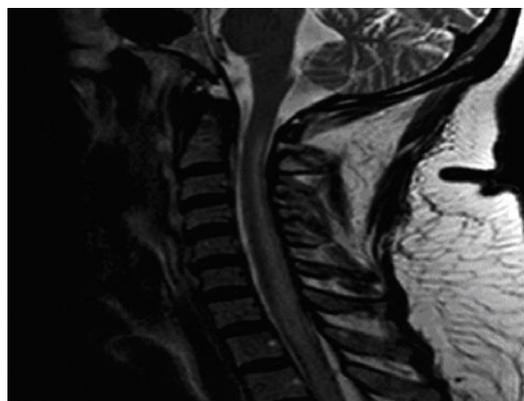


Figure 2: MRI C spine T2 sequence showing hyper intensity longitudinal extensive.

AG/AB combo non reactive, hepatitis panel negative Mycobacterium TB PCR not detected. ENA, anti dsDNA non detected, ANA weakly positive 1:80 speckled pattern.

Imaging

Gallium scan did not show any evidence to suggest adenopathy for sarcoidosis.

MRI T spine - Abnormal intramedullary T2 signal throughout the entire thoracic spinal cord, unchanged from prior exam with areas of enhancement.

MRI brain - several discrete small foci of bright T2 / FLAIR signal are scattered in the cerebral white matter with no mass effect or abnormal enhancement.

Discussion

Previous study has shown elevated protein up to 75 mg/dl following spinal cord infarction. Our case showed increase CSF protein of 460 mg following spinal cord infarction. All the inflammatory cause for extensive spinal cord T2 hyperintense signal was excluded by extensive imaging and laboratory work up.

References

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