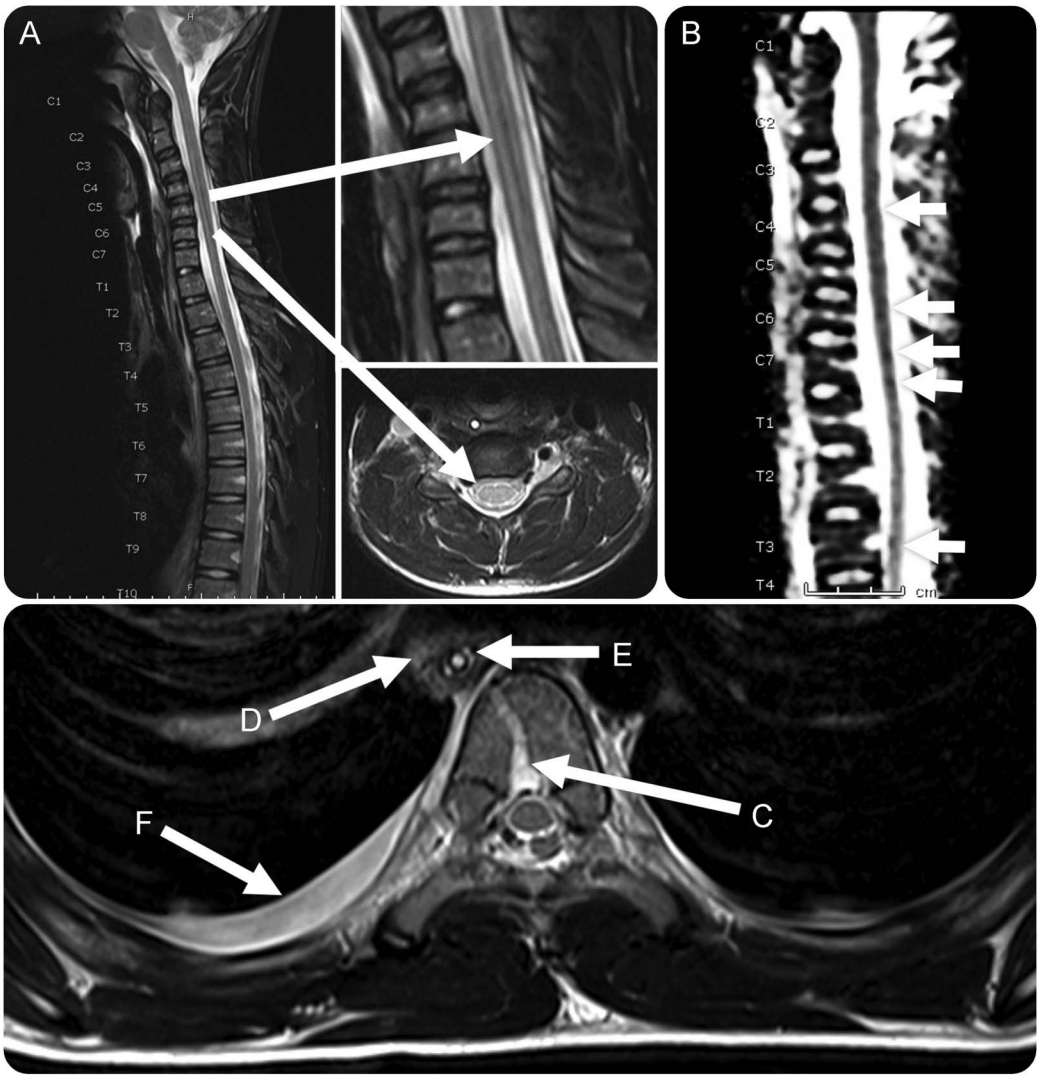
Acute spinal cord syndrome secondary to venous congestion

Figure T2 and apparent diffusion coefficient sequence MRI cervical and upper thoracic spine



Evidence of subtle T2 hyperintensity throughout the spinal cord (A) with apparent diffusion coefficient changes (B) secondary to venous congestion of the basilar vertebral plexus (C). Associated mediastinitis (D) evidenced by thickened oesophageal wall (E) with intra-luminal nasogastric tube in situ, peri-oesophageal inflammation and pleural effusions (F) is seen.

A 13-year-old boy developed lower limb flaccid paralysis 24 hours after accidental ingestion of an odorless and tasteless potassium hydroxide liquid. He had absent abdominal reflexes, brisk deep tendon reflexes, and no sensation distal to the umbilicus. MRI demonstrated T2 hyperintensities throughout the spinal cord and engorgement of basilar vertebral venous plexus (figure). He made a full recovery within 48 hours of empirical IV methylprednisolone.

Caustic ingestion can cause esophageal liquefactive necrosis and perforation leading to mediastinitis and venous congestion.[1,2] Acute spinal cord syndrome secondary to venous congestion is an infrequently reported and poorly understood entity.

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