# Title- Post-traumatic tetra paresis with the trivial mechanism of injury myelitis vs embolic spinal cord infarction in a pre-school age child.

Krupa Torne[1], Georgina Harlow [2], Adam Thomas[3]

[1,] Department of Paediatric Neurology, Nottingham University Hospitals NHS Trust, Nottingham

[2]Department of Paediatric Intensive Care, Nottingham University Hospitals NHS Trust, Nottingham1

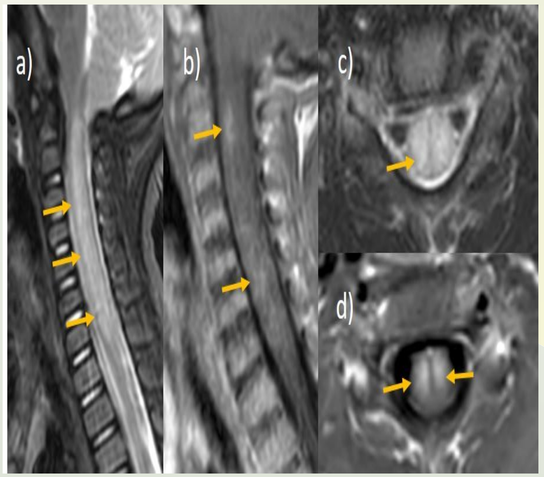
[3]Department of Paediatric Neuro Radiology, Nottingham University Hospitals NHS Trust, Nottingham

**Introduction-** Post-traumatic tetra paresis with the trivial mechanism of injury myelitis vs embolic spinal cord infarction in a pre-school age child.

**Methods-** A 3-year-old with chicken pox infection three weeks previously presented with reduced GCS after falling off a chair. Examination demonstrated flaccid tone in upper and lower limbs with absent reflexes power of 0/5, and loss of abdominal reflexes. Bladder and bowel dysfunction are present

Cranial CT, cervical spine x-ray and CSF were normal. Initial MRI showed longitudinally extensive cervical cord T2 hyperintensity involving the central grey matter with patchy enhancement (Fig 1).

**Results-**Instant neurological deficit, maturation of signal change and DWI findings suggested spinal cord infarction, most likely due to fibrocartilaginous disc embolization.



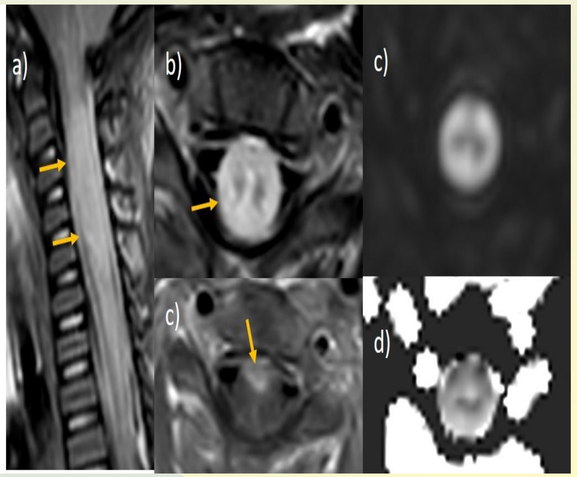




Fig 1: Sagittal STIR (a), post-contrast T1 (b), axial T2 (c) and post-contrast T1 (d) MRI showing extensive central cord T2 hyperintensity (a, c) with patchy intramedullary enhancement (b) centred around the central grey matter (d)

Repeat MRI after one week showed the evolution of T2 signal change from the centre of the cord to the periphery with diffusion restriction (Fig 2).

Fig 2: Sag STIR (a), axial T2 (b), T1 post-contrast (c), DWI (c) and ADC map (d) well-defined cervical cord signal abnormality (arrows, a) migrated to involve periphery of the spinal cord (arrow, b) with new peripheral patchy enhancement (arrow, c). DWI shows peripheral diffusion restriction.

**Conclusion-** Fibrocartilagenous disc material embolization should be considered in children with evidence of spinal cord infarction, despite the trivial mechanism of trauma and lack of any other associated traumatic imaging findings.

**References-** . Naiman JL, Donohue WL, Prichard JS. Fatal nucleus pulposus embolism of spinal cord after trauma. *Neurology* 1961;11:83–7. doi: 10.1212/WNL.11.1.83 [PubMed] [CrossRef] [Google Scholar]

2. Schreck RI, Manion WL, Kambin P, Sohn M. Nucleus pulposus pulmonary embolism. *A case report. Spine (Phila Pa 1976)* 1995;20(22):2463–6. doi: 10.1097/00007632-199511001-00016 [PubMed] [CrossRef] [Google Scholar]

3. Mateen FJ, Monrad PA, Hunderfund AN, Robertson CE, Sorenson EJ. Clinically suspected fibrocartilaginous embolism: clinical characteristics, treatments, and outcomes. *Eur J Neurol* 2011;18(2):218–25. doi: 10.1111/j.1468-1331.2010.03200.x [PubMed] [CrossRef] [Google Scholar]

4. Budka H, Perneczky A, Pusch S. Infarction of the spinal cord in the posterior spinal arterial supply area as a result of intervertebral disc embolism (author's transl). *Wien Klin Wochenschr* 1979;91(17):578–83. [PubMed] [Google Scholar]

**Disclosure-** none