

Case Report

Acute transverse myelitis following typhoid vaccination

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KEYWORDS: acute transverse myelitis, vaccination

INTRODUCTION:

Transverse myelitis is an acute spinal cord demyelination giving rise to dysaesthesia across chest or abdomen with sphincter dysfunction following viral, bacterial infection or vaccination. Annually millions of active immunization with vaccines are carried out globally with occasional case reports of transverse myelitis as adverse reactions¹. Acute transverse myelitis (ATM) following rabies, rubella, measles², influenza, polio, cholera, diphtheria, pertussis and tetanus³ has been reported in the literature but typhoid vaccine (ViCPS) induced acute myelitis has not been documented in the recent literature. We report a case of typhoid vaccine induced ATM.

CASE REPORT:

A 19-year-old man presented with paraesthesia and weakness in both legs 5 days after intramuscular ViCPS typhoid vaccine [Aventis, Pasteur]. The day after vaccination he developed fever, malaise and pain and swelling at the injection site (right deltoid). On the 5th day post-injection, he developed sensory loss of all modalities (pain, touch, temperature, vibration and joint sensation) from his lower abdomen to both legs. There was gradual, progressive, symmetrical weakness of both legs - initially flaccid - followed by spastic paraplegia with retention of urine and overflow dribbling. Lower limb power was reduced to 3/5 with bilateral hypertonia after a week of onset of weakness. Abdominal reflexes were absent. Deep tendon reflexes of the lower limbs were exaggerated with a prominent ankle clonus. Both plantars were extensor. Other systems were normal. Investigations revealed a normal haemogram, biochemistry, chest skiagram and electrocardiogram. Cerebrospinal fluid: 5 lymphocytes/mm³, 80mg/dl protein, 44mg/dl sugar [blood sugar 110mg/dl] and sterile culture. Oligoclonal bands were not detected. Spinal X-rays, nerve conduction study and visual evoked potentials were normal. Other relevant investigations including ANF, anti-dsDNA antibody, VDRL and p24 antigen were negative. To exclude compressive myelopathy, a magnetic resonance imaging with gadolinium contrast was done and revealed a centrally located high intensity signal in the T2 weighted film extending over several spinal segments at T12-L4 level (Fig 1a & 1b).

Intravenous pulse methylprednisolone [1000mg] was given for 3 days followed by oral prednisolone tapering over 2 weeks. The patient started feeling better after receiving active aerobic physiotherapy and was subsequently sent to a neurology centre.

DISCUSSION:

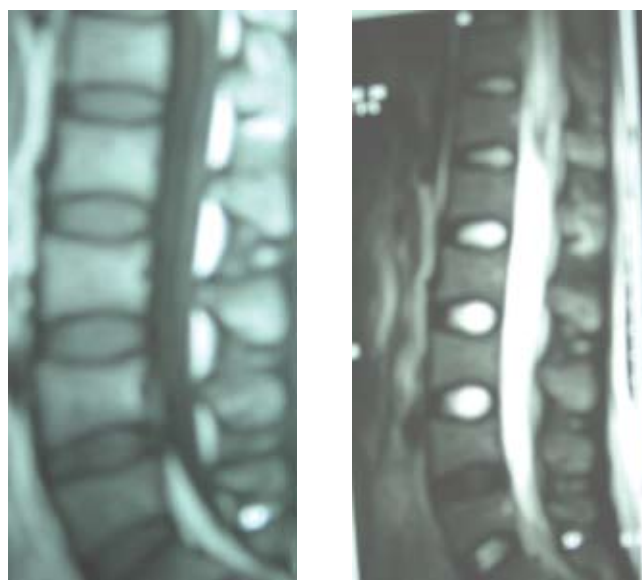


Fig 1a. T1WT: Low signal

Fig 1b. T2WT: High signal

In 1990 D'Costa reported a case of ATM following cholera, typhoid and polio vaccination⁴ and in 1955 McKelvy reported a case following TAB inoculation⁵.

Our case is similar as evidenced by paraesthesia and leg weakness with upgoing plantars, exaggerated reflexes, loss of all modalities of sensation from lower abdomen to both feet and urinary retention. Here lower spinal demyelination involved pyramidal and lateral spinothalamic tracts, posterior column and the autonomic nervous system. Neurological complications of vaccination, namely, convulsion, coma, hemiparesis, paraparesis, blindness and even multiple cranial nerve palsies⁶ mostly occurred within 24 hours of vaccination. In our case, symptoms appeared 5 days after vaccination and complete neurological deficit was established within 7 days. It fulfilled the proposed diagnostic criteria and nosology of acute transverse myelitis as described by the Transverse Myelitis Consortium Working Group⁷. Nucleus pulposus embolism (NPE) has bimodal peak of 22 and 60 years, presents with sudden, severe pain in the neck and interscapular region (70%) and rapid onset (minutes to hours) paralysis. These features distinguish it from the present ATM.

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Though influenza-like side effects are commonly seen after vaccination, there was no temporal evidence of upper respiratory viral infection at the time of vaccination. We suggest that TAB vaccination triggered the development of ATM; post-vaccination fever may have played secondary role in pathogenesis of ATM. The pathogenesis of ATM is unclear. Immunologically mediated direct insult on the central nervous system by antigen-antibody reaction has been postulated⁸.

Other aetiologies of ATM (including systemic lupus, sarcoid, Sjogren syndrome, Bechet disease, syphilis, Lyme disease and HIV) have been excluded by history and laboratory tests. The spinal form of multiple sclerosis was excluded by contrast enhanced MR imaging which revealed the centrally located high signal intensity occupied more than two thirds of the cross-sectional area of the cord. There were no peripheral plaques. The peripheral contrast enhancement of high intensity signal excludes multiple sclerosis in which enhancement in the central zone of peripherally located high signal intensity on T2 weighted images are seen⁹.

The best treatment often depends on a timely and accurate diagnosis. Identification of the aetiology may suggest some medical treatment, whereas no clearly established treatment currently exists for idiopathic ATM. Establishment of a diagnostic algorithm will likely lead to improved care, although it is recognized that the entire evaluation may not need to be performed for each patient. Prognosis of vaccine induced transverse myelitis is unsatisfactory and tends to linger for prolonged period with residual paralysis.

Typhoid vaccine-induced myelitis has not been reported in Nepal though the prevalence of typhoid is high. It is pertinent to mention that in this era of prevention we should be cautious about rare uncommon complication of vaccination against common diseases and to follow the universal rule of avoidance of vaccination during intercurrent illness.

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The Authors have no conflict of interest to declare

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