Atypical Guillain-Barré syndrome following typhoid vaccine

HISTORY

A 43 year old previously well Pakistani housewife presented with headache, facial tenderness and general weakness. Ten days previously she had visited her GP for a typhoid vaccine as she was due to visit Pakistan for the first time in four years. One day after receiving the vaccine she developed fever, lethargy and pain in her shoulder and left upper back muscles. A further 24 hours later she developed bilateral facial pain with tenderness of the ears and in the parotid and submandibular regions. Non-steroidals from her GP provided little benefit. Her face became weak and she was unable to close her eyes or mouth. Saliva pooled in her mouth and her speech became slurred. She had dry eyes but no diplopia or dysphagia. Nausea and vomiting precipitated her presentation to hospital. There were no respiratory, autonomic or limb features.

There was no history of tick bites or TB. Six months prior to the presenting complaint she had been seen with painful paraesthesia affecting her fingers and feet. Nerve conduction studies (NCS), ESR, rheumatoid factor and ANA were all normal. Her symptoms slowly resolved though she was left with some residual symptoms in her feet.

EXAMINATION

She had profound facial diplegia with severe tenderness and mild swelling in her parotid, submandibular and submental regions (see figures). There were no herpetic vesicles and her other cranial nerves were intact. Her back and shoulders were tender but there were no signs of inflammation. Her reflexes were symmetrical and brisk. Her feet were showed patchy pin-prick loss. Her forced vital capacity was 1.83l, limited by difficulty in forming a tight seal with her lips.

INVESTIGATIONS

Blood count, electrolytes, glucose, calcium, liver function, angiotensin converting enzyme and CRP were normal. ESR and creatine kinase were slightly raised at 12mm/hr and 183U/l respectively. CT head was normal. CSF was acellular with a normal glucose, but protein was raised at 1.44g/l. NCS demonstrated a mild demyelinating polyneuropathy. Antiganglioside antibodies were negative.
CXR and chest CT were normal. Campylobacter and Lyme serology were negative. Mumps serology was positive for IgG only.

DIAGNOSIS

Atypical Guillain Barré syndrome (GBS).

MANAGEMENT

As she was clinically stable iv immunoglobulin was witheld. Her arms and legs remained strong, her mobility being limited only by her back pain. She remained afebrile and her back and shoulder pain slowly improved. She was discharged home with gabapentin for her facial pain.

DISCUSSION

Facial diplegia has a number of causes, including bilateral Bell's palsy, sarcoidosis, Lyme disease, Hansen's disease, herpes zoster (Ramsay Hunt syndrome), brainstem encephalitis, HIV and GBS. Loss of deep tendon reflexes can help in distinguishing GBS as the underlying cause. The case presented here appears to be an exception to this rule. Susuki et. al. (2004) have previously reported two cases of facial diplegia with brisk reflexes as a GBS variant.
Vaccination is well reported as a cause of GBS, the influenza vaccine beng the commonest precipitant. Vaccination against hepatitis A and B, rabies and tetanus have also been reported as causes of GBS. This case appears to be the first report of GBS following typhoid vaccination.

REFERENCES

Blumenthal D, Prais D, Bron-Harlev E, Amir J. *Possible association of Guillain-Barre syndrome and hepatitis A vaccination.* Pediatr Infect Dis J.2004;23:586-8.

Chakravarty A. *Neurologic illness following post-exposure prophylaxis with purified chick embryo cell antirabies vaccine.* J Assoc Physicians India 2001; 49:927-8.

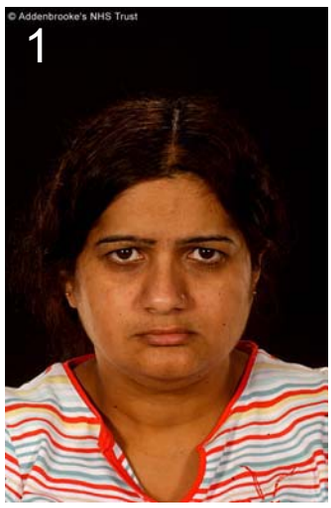
Newton N Jr, Janati A. *Guillain-Barre syndrome after vaccination with purified tetanus toxoid.* South Med J. 1997; 80:1053-4.

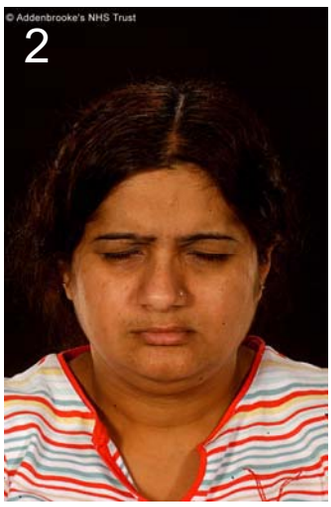
Sinsawaiwong S, Thampanitchawong P. *Guillain - Barre' syndrome following recombinant hepatitis B vaccine and literature review.* J Med Assoc Thai. 2000;83:1124-6.

Susuki K, Atsumi M, Koga M, Hirata K, Yuki N. *Acute facial diplegia and hyperreflexia. A Guillain-Barre syndrome variant.* Neurology 2004; 62:825-7.

Dr Jemeen Sreedharan is currently a neurology SHO at King's College Hospital, London. He trained there and at Addenbrookes. He has an interest in the neurogenetics of motor neurone disease and will be working with Prof Shaw on a PhD thesis

Correspondence to:
Alastair Wilkins, Case Report Coordinator, Addenbrooke's Hospital, Cambridge.
E-Mail. aw255@cam.ac.uk





FIGURES

1. patient asked to smile
2. patient asked to close eyes tight
3. demonstrating weakness of eye closure
4. patient asked to purse lips
5. swelling in parotid region