



A CASE OF SCRUB TYPHUS PRESENTED WITH GUILLAIN BARRE SYNDROME: A RARE ASSOCIATION

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ABSTRACT Scrub typhus is a zoonosis and human are accidental hosts. It is caused by *Orientia tsutsugamushi* belongs to family rickettsiae. Its clinical presentation ranges from a mild, non specific febrile illness to a life threatening fatal condition and can affect almost every organ system. Guillain Barre Syndrome (GBS) is one of the rare complication following scrub typhus infection. Hence, we are reporting a case of 17 year-old male presenting to us with GBS following scrub typhus.

KEYWORDS : Scrub typhus, *Orientia tsutsugamushi*, rickettsiae, Guillain Barre Syndrome

INTRODUCTION:

Scrub Typhus is an important cause of acute febrile illness that is caused by *Orientia tsutsugamushi*¹. Scrub typhus is a zoonosis and human are accidental hosts². It is transmitted by bite of larval stage (chigger) of a trombiculid mite (*Leptotrombidium*), which serves as both vector and reservoir¹. Humans usually become infected when they accidentally encroach 'mite islands' that contains infected mites. Mite islands can range in size from a few inches to several meters with a wide range of vegetation types from scrub (tall-growing coarse grass) and primary forest to gardens, beaches, paddy fields, bamboo patches, sandy beaches, rain forests, alpine mountains and oil palm or rubber estates.³

More than 1 million infections occur each year, and it is estimated that more than 1 billion people are at risk. Scrub typhus occurs mostly in Asia, including areas delimited by Korea, Pakistan, and northern Australia¹. The clinical presentation of scrub typhus ranges from a mild, non specific febrile illness to a life threatening fatal condition and can affect almost every organ system². The neurological complications of scrub typhus include aseptic meningitis, meningoencephalitis, seizures, delirium, hearing loss, cerebellitis, myelitis. Guillain-Barre syndrome (GBS) may be one of the rare presentations seen following scrub typhus infection⁴. Hence we reporting a case of GBS following scrub typhus infection.

Case report

A 17-year old male from rural area presented to us with complaints of fever from 7 days, and weakness in both lower limbs from 1 day which was rapidly progressive and involved upper limbs also in a day. There was no history of trauma, recent vaccination, neck swelling, numbness and paresthesia, cranial nerve and bowel-bladder involvement. On examination patient found conscious and oriented, well nourished, eschar mark present over right thigh (figure-1). Nervous system examination revealed hypotonia and areflexia in all four limbs, power 2/5 in both lower limbs and 3/5 in both upper limbs and bilateral planter response was mute. Cranial nerve, Sensory system and cerebellar system examination was unremarkable and no signs of meningeal irritation were there. So we made the provisional diagnosis of fever without focus with acute flaccid paralysis with rapidly progressive, ascending, pure motor, areflexic quadriparesis, most probably GBS. For confirmation of the above diagnosis, the patient thoroughly investigated and following results were found complete blood count (Hb-13.8g/dL), total leucocyte count (TLC)-10800/mm³, platelet count-1,99,000/mm³, blood urea-63mg/dL, Serum creatinine-0.69mg/dL, sodium-134mEq/L, potassium-4.4mEq/L, liver function test (serum glutamic-oxaloacetic transaminase {SGOT}-139U/L, Serum glutamic-pyruvic transaminase {SGPT}-109U/L), Dengue rapid test, Malaria parasite quantitative Buffy coat test (MPQBC), typhi dot IgM, Venereal Disease Research Laboratory (VDRL), hepatitis B surface antigen (HBsAg), human immunodeficiency

virus (HIV), Chest X-ray and ECG were found normal. IgM ELISA for scrub typhus was positive. Patient underwent lumbar puncture and cerebrospinal fluid (CSF) examination revealed albuminocytological dissociation (protein-109mg/dL, cell count-0 cells/mm³). Nerve conduction study was done and revealed prolonged distal latency with reduced amplitude and normal conduction velocity suggestive of both demyelinating and motor axonal neuropathy.



Figure-1. Eschar mark on right thigh

DISCUSSION

Scrub Typhus is an important cause of acute febrile illness that is caused by *Orientia tsutsugamushi*¹. There is wide range of neurological manifestations reported with scrub typhus which includes delirium, myelitis, cerebral haemorrhage, hearing loss, 6th and 7th nerve palsy, trigeminal neuralgia, opsoclonus, acute disseminated encephalomyelitis and GBS⁴.

Guillain-Barré syndrome (GBS) is an autoimmune disorder that is thought to be a postinfectious polyneuropathy, involving mainly motor but also sensory and sometimes autonomic nerves. This syndrome affects people of all ages and is not hereditary. The onset of weakness usually follows a nonspecific gastrointestinal or respiratory infection by approximately 10 days. The original infection might have caused only gastrointestinal (especially *Campylobacter jejuni*, but also *Helicobacter pylori*), respiratory tract (especially *Mycoplasma pneumoniae*), or systemic (Zika virus) symptoms⁵.

GBS may follow administration of vaccines against rabies, influenza, and conjugated meningococcal vaccine, particularly serogroup C. Other infectious precursors of GBS include mononucleosis, Lyme disease, cytomegalovirus, and the Zika virus. Subtypes of GBS include an acute inflammatory demyelinating polyneuropathy and an acute motor axonal neuropathy; these are distinguished by findings on nerve conduction studies and an associated pattern of antiganglioside antibodies. Localized forms of GBS also occur and include a pattern of facial diplegia with paresthesias and a pattern of pharyngeal-cervical-

brachial weakness. Miller-Fisher syndrome (MFS) is an uncommon GBS variant associated with acute external (and occasionally internal) ophthalmoplegia, ataxia, and areflexia. The 6th cranial nerve is most often involved in MFS⁵.

GBS is one of the rarest neurological complication following scrub typhus. The exact mechanism of GBS is unknown but it might possibly occur as a result of cell-mediated immunological response to non-self antigen that misdirects to host nerve tissue via a resemblance of epitope mechanism, called molecular mimicry. O. tsutsugamushi antibody or antigens presented on infected cells are suspected to activate mimicry on myelin cells or peripheral nerve axons, which elicits immune reactions similar to autoimmune diseases⁴.

Diagnosis of GBS is mainly based on clinical and laboratory findings. The following is the criteria for diagnosis of GBS(Asbury criteria)⁶.

Required

- Progressive weakness
- Areflexia
- Duration <4 weeks
- Exclude other causes(vasculitis,toxins,porphyria).

Supportive

- Symmetrical weakness
- Mild sensory involvement
- Cranial nerve involvement
- Absence of fever
- Typical CSF finding (albumin-cytological dissociation)
- Nerve conduction study suggestive of demyelination

CONCLUSION

Guillain -Barre syndrome is a rare neurological complication of scrub typhus. It should always be considered if a patient of acute febrile illness during infection or during recovery phase develops progressive areflexic paralysis.

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