diabetes mellitus: An update on diagnosis and management. Clin Perinatol. 2018;45:41-59.
2. De Franco E, Flanagan SE, Houghton JAL, et al. The effect of early, comprehensive genomic testing on clinical care in neonatal diabetes: an international cohort study. Lancet. 2015;386:957-63.
3. Jain V, Satapathy A, Yadav J, et al. Clinical and molecular characterization of children with neonatal diabetes mellitus at a tertiary care center in northern India. Indian Pediatr. 2017;54:467-71.
4. Flannick J, Johansson S, Njølstad PR. Common and rare forms of diabetes mellitus: towards a continuum of diabetes subtypes. Nat Rev Endocrinol. 2016;12:394-406.
5. Jahnavi S, Poovazhagi V, Kanthimathi S, Gayathri V, Mohan V, Radha V. EIF2AK3 mutations in South Indian children with permanent neonatal diabetes mellitus associated with Wolcott-Rallison syndrome. Pediatr Diabetes. 2014;15:313-8.

Glossopharyngeal and Vagus Nerve Palsy in a Child With Scrub Typhus Meningitis

Scrub Typhus is an acute febrile illness caused by *Orientia tsutsugamushi,* an obligate intracellular Gram negative bacterium. The disease is endemic in Southeast Asia and Pacific Islands, and is prevalent in the Shivalik ranges from Kashmir to Assam, the Eastern and Western Ghats and the Vindhyachal and Satpura ranges in Central India [1]. The usual clinical features include fever, myalgia, headache, an eschar, regional or generalized lymphadenopathy and hepatosplenomegaly [1].

A five-year-old boy, from North 24 Parganas district of West Bengal, was referred with high grade fever for eleven days and severe headache and myalgia. On examination, he was haemodynamically stable and had generalized lymphadenopathy and hepatosplenomegaly. Other system examinations were normal. He was given supportive management with antipyretics.

A few hours after admission, he developed nasal intonation of voice with nasal regurgitation of food. Deviation of the uvula to the left and weakness of right palatal muscles was noted on examination, signifying palsy of right sided glossopharyngeal nerve and pharyngeal branch of vagus nerve. The possibility of involvement of other vagal branches was ruled out in the absence of dysphonia. Other cranial nerves and rest of the neurological examination were normal.

Complete blood counts showed a high (90%) neutrophilic differential leukocyte count. Acute inflammatory markers were raised but liver and renal function tests were normal. Work-up for etiology of fever was positive for scrub typhus IgM antibody (ELISA) which showed a five-fold rise subsequently. Cerebrospinal fluid revealed mononuclear pleocytosis with elevated protein but negative cultures. MRI brain was normal. He was started on oral azithromycin prescribed at 10 mg/kg once a day for 7 days. Child was afebrile within 30 hrs of the first dose. Physiotherapy of pharyngeal muscles was demonstrated and he was discharged with the advice to continue the same. There was no residual nerve palsy on follow-up after 4 weeks.

Glossopharyngeal and vagus nerve palsy have been associated with Varicella Zoster, Enterovirus and other pathogens [2]. However, palsy of these nerves due to *O. tsutsugamushi* has not been described earlier in the paediatric age group.

[1]The disease process is initiated by the bite of the mite. The pathogen multiplies at the bite site, forming an eschar, followed by proliferation of the organism in the endothelial cells of small vessels with perivascular infiltration of lymphocytes causing focal or disseminated vasculitis. The eschar is considered pathognomic but may be found in 7% to 80% of the patients [3]. Central nervous system involvement commonly manifests with altered sensorium due to aseptic meningitis or acute encephalomyelitis. Occasionally, seizures, intracerebral haemorrhage, cerebellitis, and rarely acute transverse myelitis, neuroleptic malignant syndrome, Gullain Barre syndrome or nerve palsy may be noted [4].

Cranial nerve involvement in scrub typhus may result from direct invasion of central nervous system by the bacteria leading to acute vasculitis or secondary immune reaction leading to vasculitis of vasa vasorum of nerve. There have been four earlier reported cases of abducens nerve palsy in scrub typhus, causing diplopia [3-5]. Multiple cranial nerve involvement, viz. 3rd, 7th, 9th, 10th and 12th have been earlier described in a case of scrub typhus meningitis and cerebellitis [6]. There were no residual deficits in any of the above cases.

The index case developed glossopharyngeal and vagus nerve (pharyngeal branch) palsy at the summit of symptoms. Although doxycycline is the recommended drug for treatment, he was treated with azithromycin, as use of doxycycline below 8 years of age is controversial. In an endemic country like India, scrub typhus should be kept as a differential in fever for more than 7 days with neurological deficits. Timely diagnosis and intervention can have complete resolution of neurological deficits.

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REFERENCES

1. Chakraborty S, Sarma N. Scrub typhus: An emerging threat. Indian J Dermatol. 2017;62:478-85.
2. Gunbey HP, Kutlar G, Aslan K, Sayit AT, Incesu L. Mag-

netic resonance imaging evidence of varicella zoster virus polyneuropathy: involvement of the glossopharyn-geal and vagus nerves associated with Ramsay Hunt syndrome. J Craniofac Surg. 2016;27:721-3.
3. Bhardwaj B, Panda P, Revannasiddaiah S, Bhardwaj H. Abducens nerve palsy in a patient with scrub typhus: a case report. Trop Biomed. 2013;30:706-9.
4. Rana A, Mahajan SK, Sharma A, Sharma S, Verma BS, Sharma A. Neurological manifestations of scrub typhus in adults. Trop Doct. 2017;47:22-5.
5. Ete T, Mishra J, Barman B, Mondal S, Sivam RK. Scrub typhus presenting with bilateral lateral rectus palsy in a female. J Clin Diagn Res. 2016;10:OD16-7.
6. Himral P, Sharma KN, Kudial S, Himral S. Scrub meningitis complicated by multiple cranial nerve palsies and cerebellitis. J Assoc Physicians India. 2019;67:88-9.

Tuberculosis in Catastrophic Antiphospholipid Antibody Syndrome

Antiphospholipid syndrome (APS) describes patients with antibodies targeting phospholipid molecules causing recurrent arteriovenous thromboses, fetal losses, thrombocytopenia along with antiphospholipid antibodies viz. lupus anticoagulant and the anticardiolipin antibodies [1]. It can be either primary, or secondary that is triggered by infections or malignancies [2]. Catastrophic APS (CAPS), also known as Asherson syndrome, is a rare accelerated variant characterized by the rapid appearance/progression of more than three organ thromboses, with microangiopathy leading to multiorgan failure. We describe a child with pulmonary tuberculosis triggering CAPS.

A 14-year-old girl presented with increasing pyrexia and cough for 3 weeks. Mild left upper/middle zone crepitations were present with no respiratory distress. Investigations on admission revealed anemia (hemoglobin 8.5 g/dL), leucocytosis (leucocyte count 13×10^9/L, polymorphs 85%), thrombocytopenia (platelet count 45×10^9/L), with mildly elevated C reactive protein (10 mg/dL) and erythrocyte sedimentation rate (ESR 40 mm/h). Blood cultures were sterile. With an initial chest roentgenogram revealing pneumonitis, she was commenced on antibiotics (amoxicillin and clavulanic acid) with no response. Worsening in the productive cough with persistent fever, despite 7 days of antibiotics, prompted a computed tomography (CT) scan, which revealed a left upper consolidation with cavitatory changes and necrotic mediastinal lymphadenopathy. Sputum tested positive for *Mycobacterium tuberculosis* and anti-tubercular therapy (ATT) was commenced.

While on ATT, she developed severe bifrontal headache, vomiting and worsening respiratory distress. Magnetic resonance imaging of brain was performed, which was suggestive of thrombosis of superior sagittal venous sinus, right transverse sinus and sigmoid sinus with thrombus extending to proximal internal jugular vein. Repeat investigations revealed anemia, falling leucocyte counts (5.8×10^9/L), thrombocytopenia (platelet count 60×10^9/L), increasing CRP (107mg/dL), high ESR (120 mm/h) and transaminitis. Activated partial thromboplastin time (aPTT) was prolonged. Thrombophilia work-up was suggestive of antiphospholipid antibody (APLA) positivity [Lupus anticoagulant positive; Prolonged Russel viper venom time (RVVT), 78.5 (control- 48.3) seconds; anti-cardiolipin (ACL) IgG, 15 U/mL (normal range 0-12.5 GPL U/mL); Beta 2 glycoprotein 1 IgG, 30 U/mL (control 12 U/mL)]. Protein C, Protein S, anti-thrombin levels were unremarkable. Hyperfibrinogenemia (450 mg/dL), hypocomplementemia (C3, 80.8mg/dL and C4 9.2 mg/dL), positive direct coombs test, elevated serum lactate dehydrogenase (600 U/L), high serum ferritin (800 ng/mL) and significant proteinuria (spot urine protein creatinine ratio 1.2) were other positive findings. Peripheral smear ruled out schistocytes. Antinuclear antibody (ANA), Rheumatoid factor and anti-neutrophil cytoplasmic antibodies (ANCA) were negative. A CT angiogram revealed bilateral segmental pulmonary thromboembolism, ground glass changes and diffuse alveolar hemorrhage (DAH) with multiple splenic infarcts. With imaging and laboratory evidence of progressive multiorgan dysfunction, a diagnosis of CAPS triggered by pulmonary tuberculosis was made.

Child was started on parenteral methyl prednisolone followed by oral steroids and anticoagulation with low molecular weight heparin, while continuing ATT. Fever and cough resolved over the next 2 and 4 weeks, respectively. Steroids were tapered over 3 months. Repeat APLA work-up after 12 weeks confirmed APLA positivity. Anticoagulation was continued for 6 months. APLA tested negative at 6 months ruling out primary APS and establishing tuberculosis as the etiology. Repeat neuroimaging at 6 months showed significant resolution of the thrombus.

CAPS is a rare life-threatening autoimmune disease defined by definite criteria [3]. The diagnostic urgency of CAPS lies in a canonical onset of multiorgan thromboses/dysfunction, thrombotic microangiopathies (TMAs), systemic inflammatory response syndrome (SIRS) mimicking septicemia and a high mortality rate. Skin infections (18%) and human immunodeficiency virus infection (17%) are the most common associated infections [4]. ACL antibodies have been reported in a proportion of tuberculosis patients compared to normal controls [5]. Pneumonitis in the lower respiratory tract and systemic inflammation causes endothelial activation along with a reduction in the anti-coagulant mechanisms, impaired fibrinolysis with resultant hypercoagulopathy and pulmonary and systemic thrombin generation [6]. Besides, molecular mimicry between *M. tuberculosis* and β-2GPI molecule has also been proposed for the development of CAPS. In the index child, presence of such rapidly progressive deterioration with microangiopathy and thromboses (with APLA positivity) in the