Tick Induced Facial Palsy

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We report a 3-year old boy with acute onset of left sided facial palsy secondary to tick infestation in the left ear. On 7th day of follow-up, following tick removal, the facial palsy had resolved.

Key words: *Facial palsy, Tick infestation*.

Received: March 26, 2010; Initial Review: April 28, 010; Accepted: September 08, 2010

Ticks are the largest members of the order Acarina. They are vectors of many systemic diseases like rickettsial diseases, lyme disease, viral encephalitis, ehrlichiosis and babesiosis. Tick bites also lead to foreign body reactions, reactions to salivary secretions, reaction to injected toxins and hypersensitivity reactions, and neurological complications. We report tick induced isolated lower motor neuron (LMN) facial palsy in a toddler.

CASE REPORT

A 3-year old male child from rural background presented with history of deviation of angle of mouth towards right side and drooling of saliva of 1 day duration. There was history of pain in left ear and rubbing over left ear prior to onset of the symptoms. There was no history of ear discharge, fever, cold or cough. There was no past history of any significant ear, nose and throat diseases. He was born out of a non consanguineous marriage, was fourth in birth order with normal developmental milestones, and completely immunized. On examination, he was afebrile, conscious, but irritable**.** There was left sided isolated lower motor neuron facial palsy (Grade IV House and Brackmann classification). There were no other cranial nerve or motor deficits. The toddler resented handling of the left ear region and left pinna was tender. Upon otoscopic examination, the left ear canal was inflamed, swollen and clogged with blackish materials, resembling tick fecal particle. The ear was examined under anesthesia with a microscope. A tick was found near the tympanic membrane feeding on the canal wall. It was removed gently with a small crocodile forceps together with its fecal particle. There was a small perforation noted on the tympanic membrane following removal of the tick. The child was comfortable after the tick was removed. After 48 hours, the facial palsy considerably improved but child had otitis externa. He was treated with analgesics and oral amoxicillin-clavulanic acid and discharged on 4th day. On 7th day of follow up, there was no facial palsy, otitis externa improved and tympanic membrane was healing.

DISCUSSION

Ticks are obligate blood-sucking arachnids [1]. They infest dogs, cattle and other domestic animals, which can develop reversible respiratory paralysis if infestation is heavy and they may succumb to death if ticks are not removed urgently. In humans, most bites are painless as an anaesthetic and anticoagulant are introduced. Ticks are often seen or felt by the patients.

Diagnosis of intra-aural tick is straightforward. The tick may be found in the ear or evidence by the presence of feces of tick in the ear canal. An engorged full fed tick is easy to detect at its site of attachment. The unfed tick situated at the anterior deeper part of the external ear canal is not easy to see with an ordinary otoscope. The anterior bony hump of the ear canal may block the view to that particular area. Another obstacle to visualize ticks is the presence of excessive wax. The shiny appearance of the tick abdomen within the wax might be the only clue of its presence. The dark brown color of faces of tick might mix with earwax and would be difficult to differentiate [2]. In doubtful cases, examination under microscope is warranted.

Several theories may explain the pathophysiology of localized facial nerve palsy in an intra-aural tick infestation. It is likely that a presence of perforation in the tympanic membrane enable the tick saliva with toxin to enter the middle ear and reach the facial nerve probably through a natural dehiscence of the fallopian canal, causing paralysis [3,4]. In cases with intact tympanic membrane, direct extension of the inflammatory process to

the fallopian canal is via persistent dehiscence or direct invasion of the infectious organisms into the facial canal through the middle ear which results in edema of the inflamed nerve within the canal [5].

The tick produces toxin which interferes with liberation or synthesis of acetyl choline at the motor end plates of muscle fibres. Continuous secretion of toxin by the tick is necessary to produce paralysis. Recovery of paralysis occurs rapidly after removal of the tick.

Contributors: MMP and BNW reviewed the literature, managed the patient and drafted the manuscript. MMP will act as guarantor of the study. SSK collected data and helped in drafting the paper. SVP critically reviewed the article and helped in drafting the paper. The final manuscript was approved by all the authors.

Funding: None; *Competing interests:* None stated.

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Microvillous Inclusion Disease Diagnosed by Gastric Biopsy

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Received: June 22, 2010;
Initial Review: August 25, 2010;
Accepted: September 13, 2010.

Protracted diarrhea in neonates is uncommon and usually requires an intestinal biopsy for etiological diagnosis. Gastric biopsy has not been used in the routine diagnosis of this condition. We report the first documented patient with microvillous inclusion disease from India, where the diagnosis was established by a gastric biopsy.

Key words: *Diagnosis, Gastric biopsy, Infant, Microvillous inclusion disease*.

Neonatal onset of protracted diarrhea is rare and the differential diagnosis includes congenital carbohydrate malabsorption disorders, congenital ion transport defects, infectious or post infectious enteropathies, autoimmune or allergic enteropathies, IPEX syndrome, microvillus inclusion disease, and tufting enteropathy [1].

Microvillus inclusion disease (MVID) is an autosomal recessive disorder that presents in the neonatal period with severe secretory diarrhea and has no specific treatment and a high mortality [2]. The diagnosis of this condition is based on typical light and electron microscopic (EM) changes seen on small intestinal biopsies. We report the first child with MVID from India where the diagnosis was based on an antemortem gastric biopsy, confirmed later by a post mortem intestinal pathological examination.

CASE REPORT

This female baby was the third born child to healthy non consanguineous parents. The previous two babies were born by caesarean section (LSCS) at term gestation, developed profuse watery diarrhea on day three of life, and died on day three and eight of life, respectively with no specific diagnosis. The mother had an uneventful antenatal period and ultrasound done at 35 weeks gestation showed dilated bowel loops and increased amniotic fluid volume. She developed preterm prelabor rupture of membrane at 35 weeks and delivery was by LSCS in view of the previous two LSCS. This near term appropriate for gestational age girl baby, weighing 2320 g was admitted to nursery for observation in view of two previous early neonatal deaths. She was well for the first two days of life but developed profuse watery diarrhea with signs of severe