Transient Achalasia of Esophagus in Tetanus

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Abstract

A case with tetanus presenting with progressive dysphagia due to lower esophageal dysfunction is described. A barium swallow at the onset of symptoms showed esophageal dilatation with a smooth tapering at the lower end suggestive of achalasia cardia. The patient recovered from tetanus over the ensuing three weeks; repeat barium swallow at this time was normal, suggesting that esophageal dysfunction was a manifestation of tetanus. (Indian J Gastroenterol 1992; 11: 139-140)

Key Words: Dysphagia, achalasia cardia.

Introduction

Oropharyngeal dysphagia due to tonic contraction of muscles of the jaws or pharynx is a presenting complaint in over 50% of cases with tetanus. However, dysphagia due to lower esophageal dysfunction has not been reported in this disease. We report a patient with tetanus who presented with dysphagia due to esophageal achalasia which improved completely on recovery from tetanus.

Case Report

A 56 year old woman was referred with progressive dysphagia of one week's duration. The symptoms started with a sensation of retrosternal sticking of solid food, but progressed over three days to include liquids as well. A barium swallow examination elsewhere showed dilatation of the middle and lower esophagus with a smooth tapering at the gastroesophageal junction (Fig 1). An upper gastrointestinal (UGI) endoscopy was attempted at the referring hospital but was abandoned because of severe pharyngeal spasm. Two days later, UGI endoscopy was attempted again at our center after intravenous diazepam. On insertion of the endoscope, the patient developed severe pharyngo-laryngeal spasm with respiratory arrest. Resuscitation was done with endotracheal intubation and ventilatory support; the spasm disappeared 30 min later and the patient was extubated.

Aesthetic aspects of this patient have been previously published elsewhere (Baronia AK, Singh PK, Dhiman RK. Intractable pharyngeal spasm following tracheal extubation in a patient with undiagnosed tetanus. Am J Gastroenterol 1991; 86: 1111.) The present publication highlights the esophageal involvement.

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Fig 1: Barium swallow showing dilatation of body of esophagus with a smooth tapering (beaking) at the lower end.

UGI endoscopy was successfully performed the next day under general anesthesia which was induced with sodium thiopentone and maintained with halothane and 60% N2O in oxygen. It did not show any stricture or growth at the lower end of the esophagus. On completion of the procedure, severe pharyngo-laryngeal spasm, cyanosis and bradycardia developed. Endotracheal intubation and controlled ventilation led to the patient regaining consciousness within a few minutes. Attempt at tracheal extubation was followed by similar laryngo-pharyngeal spasm and tonic convulsions leading to cardio-respiratory arrest. The patient was resuscitated successfully with external cardiac massage, endotracheal intubation and intermittent positive pressure ventilation. During intubation, trismus was noticed for the first time. On the following day, lock jaw and characteristic ‘risus sardonicus’ facial expression became apparent.

Neurological examination revealed generalized hypertonia, normal power in all the limbs, exaggerated deep tendon reflexes, bilateral ankle clonus and bilateral flexor plantar reflexes. Cranial nerves and sensory system were normal. A clinical diagnosis of tetanus was made. Retrospective enquiry revealed history of a deep laceration on the right upper arm four months ago. She had not received immunization against tetanus in the past.

The patient was treated for the next ten days with diazepam infusion, continuous muscular blockade using
pancuronium bromide, antibiotics, controlled ventilation and a single dose of human tetanus immunoglobulin. Severe intercurrent respiratory infection was treated with antibiotics, chest physiotherapy, tracheostomy, and ventilatory support. Lock jaw and rhabdomyolysis started regressing from day 5 onwards and disappeared completely by day 8. Pharyngeal-lymphoid spasms disappeared completely by day 10 following which pancuronium bromide and sedation were gradually withdrawn. Dysphagia was not observed after day 12, when ventilatory support was withdrawn. Repeat barium swallow on day 20 did not show esophageal dilatation or obstruction at the lower end (Fig 2). The patient is asymptomatic 12 months later.

Discussion

Our patient presented with dysphagia due to lower esophageal dysfunction - linctus described. Wang and Karmody reported a tetanus patient with progressive dysphagia due to complete obstruction at the level of the cricocele and spasm of the cricopharyngeus muscle that disappeared completely over the ensuing ten weeks. Watanabe et al described dysphagia in a patient with tetanus due to disturbance in the second stage of deglutition. The patient was unable to pass barium into the esophagus, and some barium entered into the trachea. The disturbance in swallowing occurred as the bolus was propelled from the oral cavity, and the larynx ascended to bring about closure of the epipharynx. The patient recovered completely in two weeks. Scholte et al reported a patient who presented with progressive dysphagia of 14 days' duration and later developed trismus. Barium swallow showed pooling of barium in the hypopharynx and a small amount of barium entering the trachea. An area of asymmetry was present in the hypopharynx, but no distal esophageal abnormality was observed. The patient recovered from tetanus and dysphagia resolved completely within five weeks. In all these cases dysphagia appeared to be due to disturbance in early stages of deglutition. Achalasia like presentation due to lower esophageal dysfunction, as observed in our case, has however not been previously reported.

Cranial nerve nuclei can be involved in tetanus. Baker showed destructive changes in the motor nuclei of the vagus nerve in postmortem brainstem biopsy of a patient dying of irregular heart and respiration not associated with spasms. Reversible localized nuclear changes have been postulated to explain bizarre ocular signs in several cases of transient ophthalmoplegia and facial nerve palsy in cerebral tetanus.

In patients with esophageal achalasia, degenerative changes have been noticed in the vagal dorsal motor nucleus in postmortem studies. Higgs et al produced a condition resembling human esophageal achalasia by electrolytic lesions in the feline dorsal motor nucleus and the nucleus ambiguus of dogs.

Achalasia of the esophagus in our patient might also have been due to an effect of tetanus toxin on the dorsal motor nucleus of the vagus nerve. Nuclear lesions of cranial nerves leading to their incoordinated activity present in varied forms ranging from hypertonicity of spasmolytic contractions to paresis or paralysis of the muscles supplied by them.

References