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Dysphagia

Case Reports

Dysphagia in Tetanus: Evaluation and Outcome

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Abstract. A 72-year-old man who contracted tetanus after a puncture wound presented with severe dysphagia in association with trismus, risus sardonicus, and nuchal rigidity. We describe his medical course and outcome, including repeated videofluoroscopic barium swallow examinations. We emphasize the value of videofluoroscopy for examining and managing dysphagia in patients with tetanus, in both the acute and chronic stages of this rare illness.

Key words: Tetanus – Barium swallow, tetanus.

Tetanus is an infectious disease affecting the neuromuscular system [1]. The cause of tetanus is the toxin of *Clostridium tetani*. Since the diagnosis of tetanus is made based entirely on clinical evidence [1–3], dysphagia is often an important initial sign of the disease. As many as 30% of patients with tetanus show acute dysphagia [2]. In addition to dysphagia, the clinical hallmarks of tetanus are trismus; stiffness of neck, back, legs, and/or abdominal muscles; generalized stiffness; and risus sardonicus [4].

Using the gastrointestinal radiologic barium swallow procedure, previous reports emphasized cricopharyngeal dysfunction [1], aspiration [5], and "total dysphagia" [6]. There are no reports of dysphagia in tetanus using Logeman's modified videofluoroscopic barium swallow procedure [7]. Furthermore, the literature lacks information on the resolution of dysphagia in patients with tetanus.

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Case Report

Medical Course

A 72-year-old man was admitted with trismus and dysphagia. Four days before admission he had difficulty opening his jaw to eat and later that day began to choke when swallowing. His local physician prescribed topical throat care but his symptoms progressed. He was hospitalized locally for treatment of dehydration, then referred to the Duke University Medical Center Otolaryngology Clinic. In addition to trismus and dysphagia, he complained of progressive nuchal rigidity, but denied fever. Indirect laryngoscopy was impossible because of severe trismus, but fiberoptic examination by nasal entry revealed no anatomical abnormality. He was therefore referred to the gastrointestinal radiology department for a barium swallow. This revealed pooling of contrast material in his valleculae with overspill into his trachea and minimal movement of barium into his esophagus. He was referred to the neurology service after the radiologist diagnosed possible bulbar or neuromotor palsy.

On examination, the patient gave a history of having punctured his right fifth finger with a rusty nail approximately 10 days before the onset of his symptoms. He had never served in the military and, to his knowledge, he had never received a tetanus immunization. His past medical history was positive only for a cataract extraction. His overall health had been excellent. He was taking no medications and, specifically, had taken no neuroleptics. His general physical examination was unremarkable and his puncture wound was well healed. His neurologic examination was notable for jaw opening limited to 1 cm. He showed increased intraoral sensitivity to stimulation. Saliva collected in the corners of his mouth and he had to expectorate to clear secretions. Risus sardonicus was observed intermittently. At admission, he could not flex or extend his neck secondary to muscle spasms in his posterior neck muscles, but had not yet developed generalized muscle spasms. The remainder of his neurologic examination was normal.

The patient was diagnosed as having tetanus with an initial cephalic presentation. The differential diagnosis at admission also included stiff-man syndrome, strychnine poisoning, and extrapyramidal rigidity with dystonic spasms, but these entities were excluded as his course evolved [8].

He was admitted to the intensive care unit, where he was electively intubated for airway protection. He received 6000 units of human tetanus hyperimmune globulin intramuscularly (IM), tetanus toxoid 0.5 ml IM, and penicillin G 1.2 mU intravenously (IV) every 6 h for 10 days.

An electromyographic examination performed on the day after admission showed continuous motor unit activity in both agonist and antagonist muscles of his neck. A lumbar puncture was normal. The following day he began to have frequent, brief, spontaneous, and stimulus-induced flexor spasms of his limbs with trunk extension and diaphragmatic spasms as well. His temperature rose to 38.6° C and tachycardia to 120 beats/min was noted. Over the ensuing week his spasms were treated with lorazepam IV, and he developed bilateral ptosis and ophthalmoplegia. His systolic blood pressure varied from 120 to 180 mmHg, but pharmacologic treatment was not necessary. He received supportive care including mechanical ventilation and enteral nutrition (Dubhoff tube feedings). Twelve days after admission he was extubated, although he continued to have intermittent jaw spasms with stimulation.

Swallowing Evaluations

The baseline barium swallow study on the day of admission revealed loss of coordinated neuromotor pharyngeal activity with extensive aspiration. Once the patient was medically stable (on day 23), a speech/language pathology consultation was obtained to determine if oral feedings could resume. A clinical examination (Table 1) was notable for showing decreased strength of the tongue and jaw and a hypersensitive gag reflex bilaterally. His voice was perceived as mildly harsh and breathy in quality, but otherwise speech was within normal limits. A bedside examination of swallowing (using small sips of water) caused significant coughing after each bolus. The patient complained of occasional choking, excessive oral secretions, and a need to clear his throat frequently. On follow-up examination at 15 months (Table 1), he had no complaints regarding swallowing, was on a regular diet, and was maintaining his weight. His oral-facial examination was normal except for hypersensitive gag bilaterally. Dysphonia had cleared and his speech quality was within normal limits.

Three videofluoroscopic barium swallow studies were performed, as summarized in Table 2. The first examination showed severe dysphagia. Minimal amounts of barium entered the esophagus and extensive aspiration occurred. The second examination showed moderate dysphagia. The oral preparatory phase was normal, but reflex initiation was mildly delayed at the level of the vallecular spaces. During the pharyngeal phase of swallowing, small amounts of both liquid barium (barium sulfate) and "paste" barium (Esophotrast) entered the laryngeal vestibule. A vigorous reflexive cough cleared the laryngeal vestibule of material. No aspiration occurred. After the swallow, bilateral residue of paste barium was seen in the vallecular spaces and pyriform sinuses (slightly more on the left than on the right). Multiple sips of liquid from a cup facilitated clearance of the pharyngeal residue. The criopharyngeus opened well.

At a 15-month follow-up examination, abnormal swallowing persisted. The oral preparatory and reflex initiation phases of the swallow were now within normal limits, and there was no laryngeal vestibule penetration. However, moderate pharyngeal weakness persisted, as evidenced by residue of both paste barium and paste-coated cookie boluses bilaterally in the vallecular spaces and unilaterally in the left pyriform sinus. Alternation of liquids with solids continued to facilitate clearance of the pharyngeal residue.

Management

Following completion of the barium swallow study on day 23, oral feeding recommendations included soft dietary texture, small boluses, alternating liquid with solid boluses, and taking multiple swallows to facilitate pharyngeal clearing. Frequent gentle throat clearing was encouraged. On follow-up examination, he was tolerating a regular diet. He offered no complaint about pharyngeal residue after swallowing. Nevertheless, continued alternation of liquids between solid food boluses was recommended to ensure clearance of food from the pharynx.

Table 1. Clinical evaluations of swallowing[a]

	August, 1986	November, 1987
Subjective complaint	Choking	None
Motor exam[b]		
Jaw	±	+
Lips	+	+
Tongue	±	+
Velum	+	+
Sensory exam[c]		
Tongue	+	+
Lips/Face	+	+
Gag reflex	±[d]	±[d]

[a] On admission, 7/28/86, severe trismus precluded oral examination.
[b] Isolated movements.
[c] Light touch stimulation.
[d] Hypersensitive gag bilaterally.
+, Intact; ±, moderately impaired.

Table 2. Videofluoroscopic barium swallowing evaluations[a]

	August, 1986[b]	November, 1987[b]
Oral preparatory phase	Normal	Normal
Reflex initiation phase	Mild delay	Normal
Pharyngeal phase	Moderate residue	Moderate residue
Aspiration	None, but laryngeal vestibule penetrated[c]	None
Cricopharyngeal relaxation	Normal	Normal

[a] On the admission (7/24/86) barium swallow study, liquid barium (barium sulfate) only was given. Findings were severe trismus, pooling of barium in the valleculae, overspill into the trachea with gross aspiration, and minimal movement of barium into the esophagus.
[b] Logemann procedure [6].
[c] Vigorous cough protected the trachea.

Discussion

Tetanus is a rare and often fatal disease. The average annual incidence rate for 1982–1984 was 0.036 cases per 100,000 total population, with a 52% mortality rate for patients 60 years of age and older [9]. The exotoxin, known as tetanospasnin, is produced by the bacillus *C. tetani* in a contaminated wound, usually following a puncture injury. The injury itself may be trivial, and in 10–20% of cases

no history of an injury or wound may be found [8]. The toxin, once produced, is distributed in the bloodstream and is taken up by the peripheral endings mainly of motor nerves and, to a lesser extent, sensory and autonomic nerves. It is then retrogradely transported to cell bodies and eventually crosses into the presynaptic terminals of inhibitory interneurons. The tetanus toxin has been shown to block transmission at inhibitory synapses by presynaptic interference with both spontaneous and stimulus-evoked neurotransmitter release. At the spinal cord level, the toxin is thought to block the normal feedback inhibition of the Renshaw cells, which are small inhibitory interneurons that use glycine as a neurotransmitter [10].

The initial symptom of tetanus is dysphagia in as many as 30% of all cases [2]. Nyhan and colleagues stated: "The hypertonicity of the masseters which produces trismus is often accompanied by increased tone and incoordination of the swallowing mechanism" [11, p. 27]. The presence of dysphagia suggests a moderate expression of tetanus [1, 11, 12]. Patients with moderate or severe tetanus usually remain dysphagic for 2–4 weeks [11].

The present case report substantiates earlier findings of pharyngeal dysphagia with aspiration [5], and documents the long-term outcome of dysphagia in tetanus. Our patient showed profound dysphagia acutely; moderate dysphagia at the end of a 26-day hospitalization; and mild persistent dysphagia at 15 months after onset. In our patient, dysphagia was not only the initial symptom but also a long-lasting sequela of tetanus.

Tetanus is usually regarded as a reversible illness, if the patient survives. Earlier studies suggested that patients who recover from tetanus do not present with any residual deficits [12, 13]. However, recovery may require nerve terminal sprouting and formation of new synapses, which processes may be slow or impaired in an elderly individual. Also, brainstem lesions that could result in residual impairment in survivors have been reported in patients dying from tetanus [8]. We suggest that subjective and clinical assessments of complete recovery from tetanus may miss subtle residual abnormalities of swallowing function, as demonstrated in our patient by a modified barium swallow study.

We conclude that the modified barium swallow study is valuable for understanding the acute and chronic effects of tetanus. As in all types of neurogenic dysphagia, the information obtained from this procedure is useful in determining individual feeding management programs. As a result of the multidisciplinary care described in this report, our patient ultimately resumed use of a regular diet despite residual dysphagia.

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