

Successful Surgical Treatment for Dysphagia Secondary to Descending Necrotizing Mediastinitis

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ABSTRACT

Descending necrotizing mediastinitis (DNM) is a life-threatening disease with a high fatality rate that occurs as a complication of deep neck abscess. DNM complicated by severe dysphagia during the postoperative period has been previously reported, but there have been no published cases of surgical treatment for severe dysphagia secondary to DNM. A 63-year-old man was diagnosed with a deep neck abscess followed by DNM. The patient had dysphagia after incision and drainage of the abscess and drainage for DNM with video-assisted thoracic surgery (VATS). A comprehensive long-term physiotherapy program with a speech and language therapist did not reduce his dysphagia. Thus, the patient underwent laryngeal elevation and cricopharyngeal myotomy, which enabled oral intake. Surgery should be considered for prolonged severe restriction of laryngeal elevation and esophageal introitus opening that is unresponsive to physiotherapy with a speech and language therapist.

Key words cricopharyngeal myotomy; deep neck abscess; descending necrotizing mediastinitis; dysphagia; laryngeal elevation; surgical treatment

Descending necrotizing mediastinitis (DNM) is caused by downward spread of deep neck infections.^{1–7} DNM arises as a complication of odontogenic, pharyngeal, or other cervicofacial infection, and may result in sepsis.^{1, 6, 8} Despite modern antibiotic treatment, advances in diagnostic and surgical techniques, and improvements in anesthetic and intensive care protocols, DNM remains a life-threatening condition with reported mortality rates of up to 40%.^{3, 5, 9}

DNM requires prompt, aggressive, and multidisciplinary medical and surgical therapy. Fundamentally,

the primary treatment for DNM includes intravenous broad-spectrum antibiotics, airway management, surgical restoration of the pharyngeal or dental focus, and adequate drainage of the neck and the mediastinum.⁷

DNM complicated by severe dysphagia during the postoperative period has been previously reported. Glen et al reported that the swallowing function improves with a comprehensive long-term physiotherapy program provided by a speech and language therapist and percutaneous endoscopic gastrostomy (PEG) feeding.¹⁰ However, to the best of our knowledge, no cases of surgical treatment for severe dysphagia secondary to DNM have been previously reported.

We report a case of a male patient who presented with dysphagia secondary to DNM with improvement after surgical treatment. We also describe the mechanisms underlying dysphagia and the indications for surgical treatment of dysphagia secondary to DNM.

PATIENT REPORT

A 63-year-old man was admitted to our department with high fever, neck pain, and neck swelling. He had been experiencing pain in his left lower jaw for 4 days. Neck swelling, erythema and pain during palpation were present, with an elevated body temperature (39.3°C) (Fig. 1). Transnasal endoscopic examination showed edema of the arytenoid fold and swelling of the lateral pharyngeal wall. Laboratory testing at admission revealed elevated inflammatory parameters (CRP 19.02 mg/dL, WBC 8,800 / μ L, leukocytes 7,568 / μ L) and a low platelet count (55,000 / μ L). Neck ultrasonography showed emphysema throughout the neck. Computed tomography (CT) demonstrated emphysema in the retropharyngeal, paratracheal, paraesophageal, perilaryngeal, submandibular, and both carotid spaces, and in the upper mediastinum (Fig. 2). The patient was diagnosed with deep neck abscess secondary to infection by anaerobic bacteria.

Urgent incision and drainage of the retropharyngeal, carotid, paratracheal, paraesophageal, perilaryngeal, and submandibular spaces was performed. Abscess was found in both carotid spaces and the paratracheal and perilaryngeal spaces. Tracheostomy was performed for laryngeal and pharyngeal edema.

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Abbreviations: CT, computed tomography; DNM, descending necrotizing mediastinitis; FOSS, Functional outcome swallowing scale; PEG, percutaneous endoscopic gastrostomy; VATS, video-assisted thoracic surgery; VF, videofluoroscopy

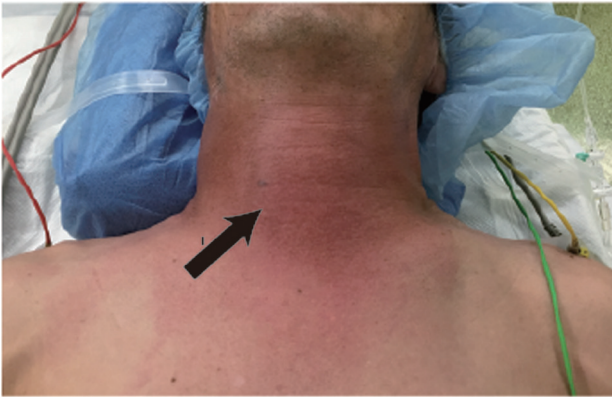


Fig. 1. Preoperative clinical appearance of the neck at presentation. Severe erythema was observed throughout the neck (black arrow).

We installed multiple large drains and left the cervical incision open to increase tissue exposure to oxygen. Intravenous piperacillin/tazobactam (12 g/day) was administered. Wound packing and irrigation with an antiseptic solution were performed multiple times per day.

On postoperative day 1, he underwent chest CT to evaluate for infection, which revealed a multi-segmented low-density area in the mediastinum. Based on the laboratory data and radiologic findings, he was diagnosed with DNM and immediately underwent mediastinal drainage with video-assisted thoracic surgery (VATS). Weakening of the mediastinal pleura due to abscess formation was observed. A pleural effusion was founded in the right thoracic cavity. Incision of the mediastinal pleura and drainage of the mediastinal abscess were performed. Finally, a thoracostomy tube was inserted into the mediastinum and thoracic cavity. Postoperatively, the cavity was rinsed through the thoracostomy tube twice daily, which helped achieve reduction of discharge. Microbiologic cultures of sections

from the neck revealed the presence of *Streptococcus anginosus* and *Fusobacterium*. We switched piperacillin/tazobactam to meropenem (3.0 g/day) based on susceptibility testing of the cultured microbes.

The patient's clinical condition improved and laboratory parameters of inflammation normalized, so antibiotic administration was completed on hospital day 21. Follow-up CT identified no recurrent abscesses. The drains in the neck and the tracheal cannula were removed on postoperative day 24.

Even after DNM was treated, the patient experienced difficulty in swallowing, despite being neurologically intact. Videofluoroscopy (VF) revealed poor laryngeal elevation, inadequate upper esophageal sphincter opening, residue in the pharyngeal cavity (valleculae and pyriform sinuses), aspiration, and poor pharyngeal constrictor function (Fig. 3a). A nasogastric tube was placed for nutrition with nonoral intake which is stage 5 on the Functional Outcome Swallowing Scale (FOSS). (For reference, the stages of FOSS are as follows: stage 0 = normal function and asymptomatic; stage 1 = normal function but with episodic or daily symptoms of dysphagia; stage 2 = compensated abnormal function manifested by significant dietary modifications or prolonged mealtime (without weight loss or aspiration); stage 3 = decompensated abnormal function with weight loss of 10% or less of body weight over 6 months due to dysphagia, or daily cough, gagging, or aspiration during meals; stage 4 = severely decompensated abnormal function with weight loss of more than 10% of body weight over 6 months due to dysphagia, or severe aspiration with bronchopulmonary complications, nonoral feeding recommended for most of nutrition, and stage 5 = nonoral feeding for all nutrition. Consequently, no laryngo-pharyngeal paralysis, ocular motility disorder or facial sensory-motor disturbance was observed and CT showed no abnormality in the brain stem and cerebellum. The patient underwent a

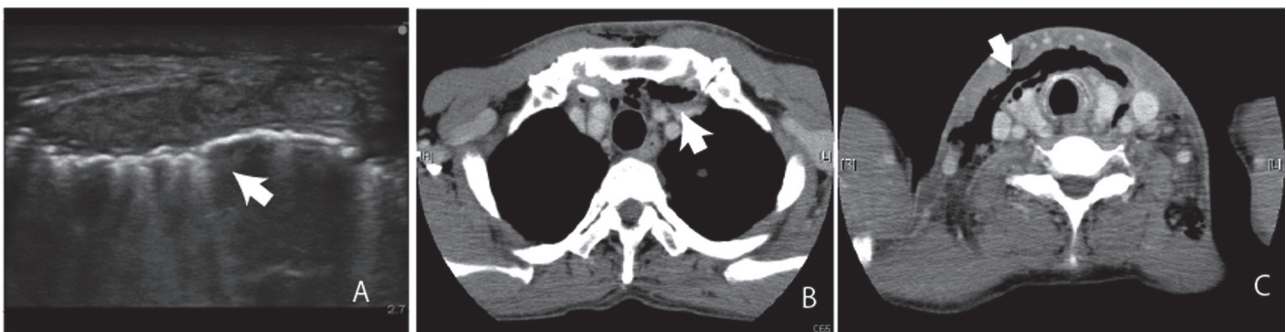


Fig. 2. Preoperative computed tomography (CT) and ultrasound images. **A:** Ultrasonography showed an anechoic area just below the hyperechoic line (white arrow). **B:** CT of the upper mediastinum showed air patterns in the retrosternal region (white arrow). **C:** CT of the neck showed air patterns in the paratracheal space, perilaryngeal space, and both carotid spaces (white arrow).

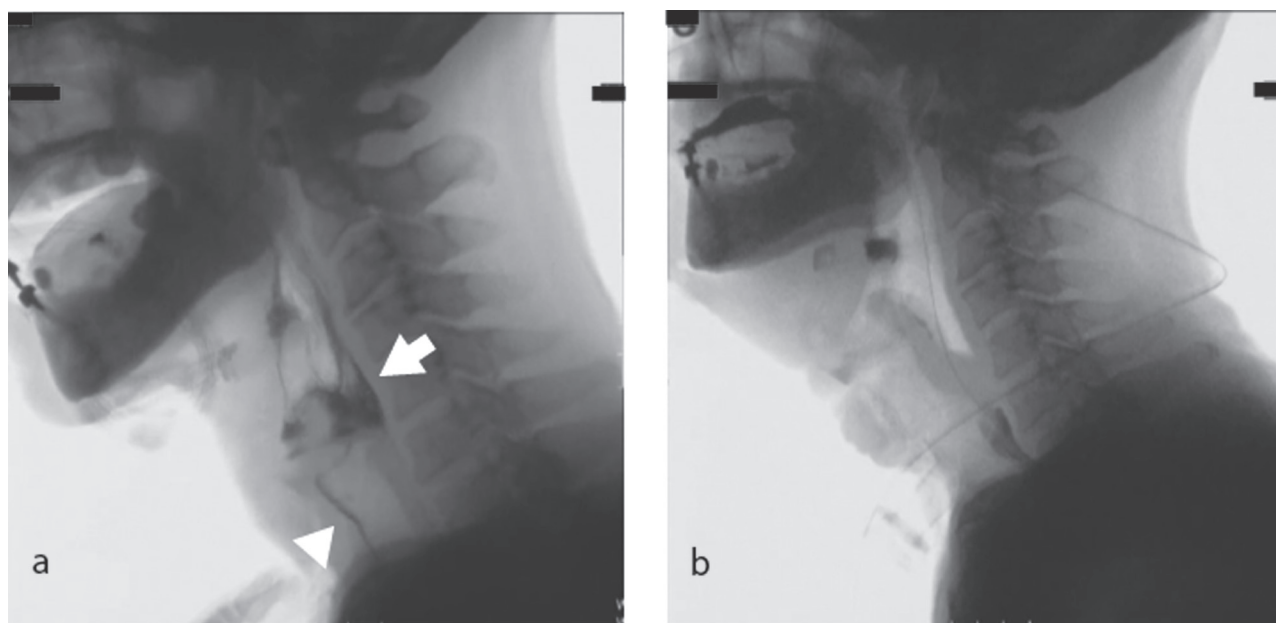


Fig. 3. Videofluorography. **A:** Preoperative videofluorography showed poor laryngeal elevation, inadequate upper esophageal sphincter opening, residue in the pharyngeal cavity (valleculae and pyriform sinuses) (white arrow), aspiration (white triangle), and poor pharyngeal constrictor function. **B:** Postoperative videofluorography showed improved upper esophageal sphincter opening, less residue in the pharynx, and no aspiration.

comprehensive long-term physiotherapy program such as oral rehabilitation with a speech and language therapist. However, no head raising exercise like the Shaker exercise was performed because the neck was still not completely healed. Despite the physiological program, no visible improvement in swallowing function was observed.

At 6 months after initial surgery, his swallowing had not improved. Surgical treatment was performed for the purpose of improving swallowing function and allowing oral intake. Laryngeal elevation and transcervical cricopharyngectomy were selected as surgical procedures because he had poor laryngeal elevation and an inadequate upper esophageal sphincter opening.

Surgery was performed under general anesthesia. The tissue was extremely hemorrhagic and there was a severe adhesion from the peritracheostoma to the anterior of the larynx, making it difficult to elevate the cervical flap (Fig. 4a). Also, there was marked scarring of the perilaryngeal and infrahyoid muscles, including the sternohyoid, thyrohyoid, and omohyoid muscles and adjacent tissues. We resected the infrahyoid muscles, resulting in mobility of the larynx. The thyroid cartilage was advanced approximately 2 cm using sutures placed into the lower inside portion of the mandibular bone (Fig. 4b). Finally, the cricopharyngeal muscle was exposed using an intraluminal balloon catheter as a guide. The

cricopharyngeal muscle was weak and thin, so we carefully resected it as much as possible from the left side to prevent the pharyngeal wall from breaking (Fig. 4c). We used intermittent intraoperative neural monitoring of the recurrent laryngeal nerve to prevent recurrent laryngeal nerve palsy because scar tissue from the previous neck surgery was expected. It was easy to identify the recurrent laryngeal and superior laryngeal nerves and maintain good hemostasis. Histopathological examination of the cricopharyngeal muscle showed disuse atrophy and scar formation of the muscle fibers but not myopathy. The affected portion of the muscle was 20 μm in diameter, narrower than the normal muscle (50–60 μm). Muscle fibers did not exhibit either necrosis or regeneration, however there was an inflammatory cell infiltration (Fig. 5).

Ten days after laryngeal elevation and transcervical cricopharyngectomy, VF revealed the development of laryngeal elevation and upper esophageal sphincter opening (Fig. 3b). Adequate oral intake was possible (FOSS: 1).

DISCUSSION

Dysphagia is known to be a late complication in DNM, after the infection is under control. Sander et al reported that 2 out of the 45 cases diagnosed with DNM had dysphagia.⁵ Furthermore, Glen et al reported that

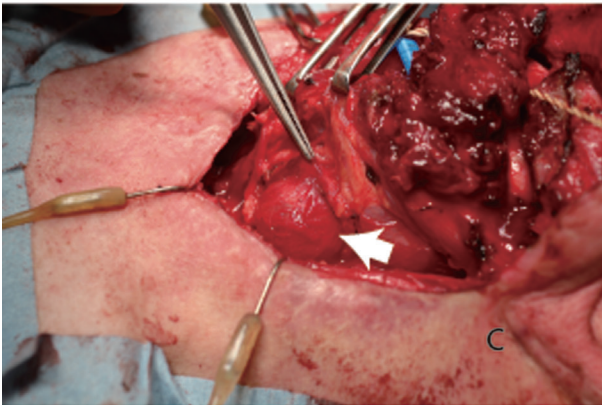
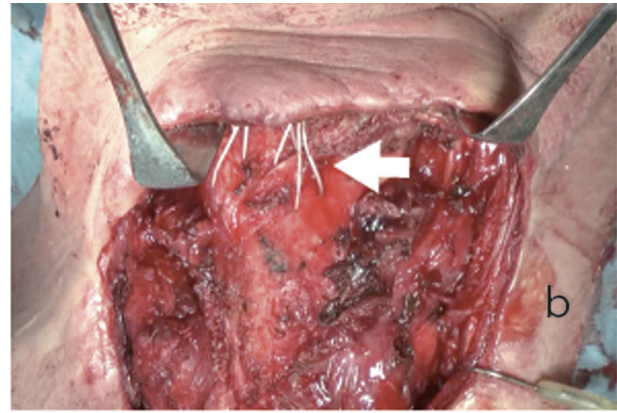


Fig. 4. Intraoperative findings of laryngeal elevation and transcervical cricopharyngectomy. **A:** There was a severe adhesion from the peritracheostoma to the anterior of the larynx. **B:** The thyroid cartilage was advanced approximately 2 cm using sutures (FiberWire®) placed into the lower inside portion of the mandibular bone (white arrow). **C:** The cricopharyngeal muscle was exposed using an intraluminal balloon catheter as a guide (white arrow). The cricopharyngeal muscle was weak and thin, so we carefully resected it as much as possible from the left side to prevent the pharyngeal wall from breaking.

severe dysphagia following DNM developed despite a comprehensive long-term physiotherapy program for 6 months.¹⁰ To the best of our knowledge, a patient with severe dysphagia who underwent surgical treatment such as laryngeal suspension and cricopharyngeal myotomy has not been reported previously.

There are three potential pathways for the spread of a neck infection into the mediastinum: (1) the pretracheal route to the anterior mediastinum, (2) the lateral pharyngeal route to the middle mediastinum, and (3) the retropharyngeal-retrovisceral route to the posterior mediastinum.¹¹ In this patient, the pretracheal route was the main route for the spread of infection to the mediastinum. Therefore, chronic inflammatory changes within the fascial spaces were thought to have caused fibrosis and scarring of the laryngeal and hypopharyngeal muscles. This results in inadequate laryngeal elevation, poor opening of the esophagus, and impairment of the pharyngeal phase of swallowing. Intraoperatively, extensive scarring around the larynx, pharynx, and trachea were observed. The removal of scar tissue around the larynx and hypopharynx improved laryngeal elevation, and cricopharyngeal myotomy improved the opening of the esophagus, which restored swallowing function. This case suggests that surgery should be

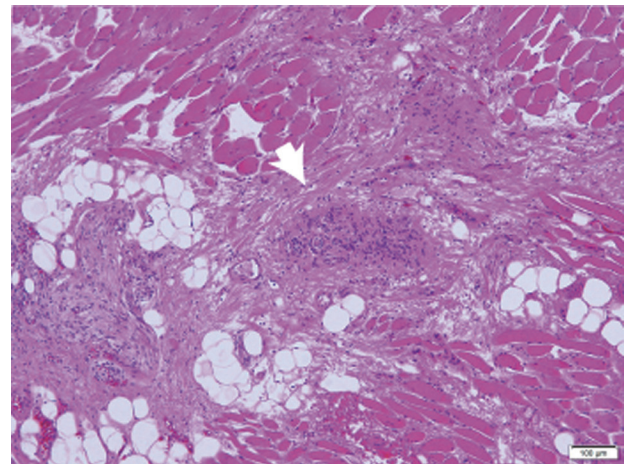


Fig. 5. Histopathological examination of the cricopharyngeal muscle showed disuse atrophy and scar formation of the muscle fibers but not myopathy (white arrow). The affected portion of the muscle was 20 μ m in diameter, narrower than the normal muscle (50–60 μ m). An inflammatory cell infiltration was observed.

considered for patients with prolonged severe restriction of laryngeal elevation and esophageal introitus opening who are unresponsive to a physiotherapy program with a speech and language therapist.

Long-term absence of oral intake causes impairment of swallowing function as well as progression of scar formation.¹² In this patient, it took 3 months to start oral intake. Long-term lack of oral intake induced scar progression and resulted in dysphagia. Although it goes without saying that infection control is important, oral intake should be started based on an evaluation of swallowing as soon as possible after infection is under control.

Challenges in re-operation after drainage for DNM include scar formation and impaired blood flow. Thus, in this patient, we introduced intraoperative neural monitoring to prevent recurrent laryngeal nerve injury and preserve swallowing function.¹³ It was useful not only in detecting the position of the recurrent laryngeal nerve, but also to preserving perineural blood flow; no recurrent laryngeal nerve paralysis occurred during re-operation. The use of intraoperative neural monitoring of the recurrent laryngeal nerve might be considered if there is a foreseeable risk of extensive scarring, for example, with re-operation.

This patient had dysphagia after incision and drainage of a deep neck abscess as well as drainage with VATS for DNM. A comprehensive long-term physiotherapy program with a speech and language therapist did not reduce dysphagia. Thus, the patient underwent laryngeal elevation and cricopharyngeal myotomy, which made oral intake possible. Surgery should be considered for patients with prolonged severe restriction of laryngeal elevation and esophageal introitus opening who are unresponsive to a physiotherapy program with a speech and language therapist.

The authors declare no conflict of interest.

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