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# Catastrophic Caudad Spread of a Peritonsillar Abscess: A Case Report

*Gerard R. Manecke, Jr., M.D.,* Saad Marghoob, M.D.,† Kathleen C. Finzel, M.D.,‡ David C. Madoff, M.D.,§ Irene H. Quijano, M.D.,‖ Paul J. Poppers, M.D.#*



PERITONSILLAR abscess is a known cause of serious airway complications, and, in this context, is discussed in anesthesiology texts.[1,2] Other medical literature contains reports of peritonsillar abscesses that spread along the deep fascial planes of the neck and chest. We report a rare case in which caudad spread of a peritonsillar abscess lead to life-threatening conditions in addition to airway compromise.

## Case Report

A previously healthy 29-yr-old man was admitted to an outlying hospital with a 4-day history of fever, dysphagia, and dysphonia. Examination at that hospital revealed bilateral tonsillar hypertrophy, right-sided peritonsillar abscess, and probable retropharyngeal abscess. The patient was transferred to our hospital. During the 2-h transport, he experienced difficulty breathing. At arrival to the emergency room, he was diaphoretic, tachypneic (respiratory rate 28 breaths/min), and in moderate respiratory distress. Heart rate was 128 beats/min, blood pressure was 144/88 mmHg. Oxygen saturation as measured by pulse oximetry ($Sp_{O_2}$) was 88%, despite 100% oxygen inhalation. Results of external examination of the airway were normal. Fiber-optic nasopharyngoscopy was performed by an otolaryngologist and confirmed the presence of a peritonsillar abscess. The uvula was deviated to the left, but the glottis and vocal cords were normal. Intravenous antibiotics were administered (ceftriaxone and clindamycin). A chest radiograph showed bilateral lung infiltrates and a widened mediastinum. After 12 ml pus was aspirated from the right tonsillar area, a nasopharyngeal airway was placed in the left naris. These maneuvers resulted in modest improvement in breathing pattern, but $Sp_{O_2}$ remained at 88–90%. Arterial blood gas revealed the following: *p*H: 7.45; partial pressure of carbon dioxide ($P_{CO_2}$): 33 mmHg; partial pressure of oxygen ($P_{O_2}$): 58 mmHg; bicarbonate: 23 mEq/l. The otolaryngologist requested that we secure the airway with an endotracheal tube, preferring to obtain a computerized tomography of the neck before surgical intervention. With the otolaryngologist present and a tracheostomy kit at hand, an awake fiber-optic nasotracheal intubation was performed after topical anesthesia with 4% lidocaine spray and phenylephrine was applied. The intubation was difficult and time consuming, requiring approximately 40 min and five attempts by physicians experienced in fiber-optic intubation. During this period, the patient became more uncomfortable, and edema and erythema of the anterior neck were noted. Although preparations for tracheotomy were being made, intubation was successful on the fifth attempt. After successful intubation, the oxygen saturation improved to 97%. Computerized tomography of the neck and chest was then performed. The scan of the neck showed air density and swelling in the left and anterior parapharyngeal spaces, with tracheal deviation (fig. 1). The scan of the chest showed air density and swelling in the anterior and posterior mediastinum, large bilateral pleural effusions, a large pericardial effusion, and air density in the pericardial space (fig. 2). A thoracic surgeon was consulted, and plans were made to bring the patient to the operating room emergently for bilateral chest tube thoracostomy, right thoracotomy, pericardial window, neck exploration, and tracheostomy. During transport to the operating room, hemodynamic status deteriorated such that, at arrival, heart rate was 150 beats/min and blood pressure was 60/40 mmHg. Phenylephrine and dopamine infusions were begun. While the chest tubes were inserted, bilateral femoral venous large-bore intravenous catheters and a right radial arterial catheter were inserted. When the right thoracotomy with pericardial window was performed, approximately 100 ml pus was drained from the pericardial space, and approximately 300 ml was removed from each pleural space. This resulted in a dramatic improvement in the patients hemodynamic condition. Large amounts of pus were also found in all fascial planes of the neck. At the conclusion of the surgery, the patient no longer required hemodynamic support. His heart rate was 94 beats/min and blood pressure was 130/80 mmHg. $Sp_{O_2}$ was 99% during mechanical ventilation with 50% oxygen and 5 cm positive end-expiratory pressure.

* Associate Professor of Anesthesiology.

† Assistant Professor of Anesthesiology.

‡ Assistant Professor of Radiology.

§ Resident in Radiology.

‖ Resident in Anesthesiology.

# Distinguished Professor and Chairman of Anesthesiology.

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Address reprint requests to Dr. Manecke: Department of Anesthesiology, Health Sciences Center, SUNY at Stony Brook, Stony Brook, New York 11794-8480. Address electronic mail to: GManecke@epo.som.sunysb.edu

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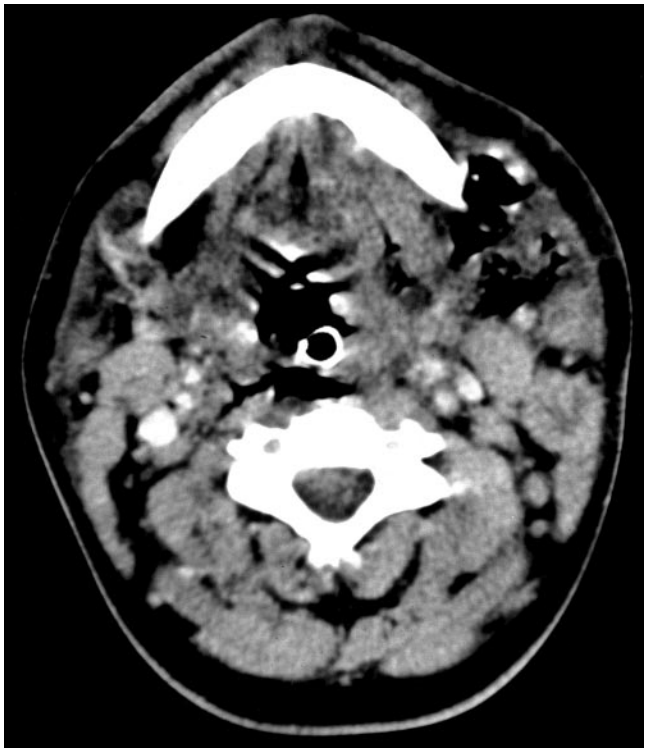

**Fig. 1. Computerized tomography (CT) scan of the neck showing soft tissue swelling, gas in the submandibular space, and tracheal deviation.**

Unfortunately, the patient underwent a long, protracted convalescence. On the second postoperative day, he had an episode of cardiac arrest, which appeared to be precipitated by a vagal reaction to suctioning of the tracheostomy. This resulted in a stroke that caused him to be ventilator dependent. Postoperative course was also complicated by recurrent pneumonia and sepsis. He is receiving rehabilitation in a long-term care facility.

## Discussion

The airway problems resulting from peritonsillar and retropharyngeal abscesses are well-documented in the anesthesiology literature.[3] In this patient, however, emergency airway management was merely the first step.

Various life-threatening complications of oropharyngeal infections have been reported, primarily in the otolaryngology, emergency medicine, and thoracic surgery literature. These include carotid artery erosion,[4,5] jugular venous thrombophlebitis (Lemierre Syndrome),[6] mediastinitis,[7] empyema,[8] meningitis,[9] esophageal perforation, pericarditis,[10] and septicemia.[11] The deep fascial planes of the neck divide the midline tissue into the prevertebral space (posterior), the retropharyngeal space, and the "danger space," which lies between them. The danger space is so called because it is contiguous with the deep cervical and mediastinal tissues. Oropharyngeal infections, particularly those involving necrotizing organisms, can spread to the retropharyngeal space and the deep fascial planes of the neck. The danger space provides a conduit for invasion into the mediastinum, often leading to pleural and pericardial involvement. The presence of gas-producing bacteria, as in this case, can cause the appearance of multiple air-density areas during computed tomography of the head and neck (figs. 1 and 2).

Risk factors for the development of peritonsillar abscess or retropharyngeal abscess include immune deficiency, recent dental work, chronic or recurrent tonsillitis, alcohol abuse, and cocaine abuse. Our patient had recurrent tonsillitis, but none of the other conditions. The mortality rate for these patients has been reported to be as high as 36%. Infections are usually polymicrobial, including gram-positive cocci, gram-negative rods, and anaerobes.[12] Indeed, cultures in our patient grew various aerobic and anaerobic organisms, including $\beta$-hemolytic *Streptococcus, Peptostreptococcus,* and bacterioids. Best results are obtained when aggressive antibiotic and surgical therapy are instituted quickly.[7] Surgery often involves procedures similar to those performed here: aggressive neck exploration, tracheostomy, tube thoracostomy, thoracotomy, and pericardial window. Hemodynamic instability may result from cardiac tamponade

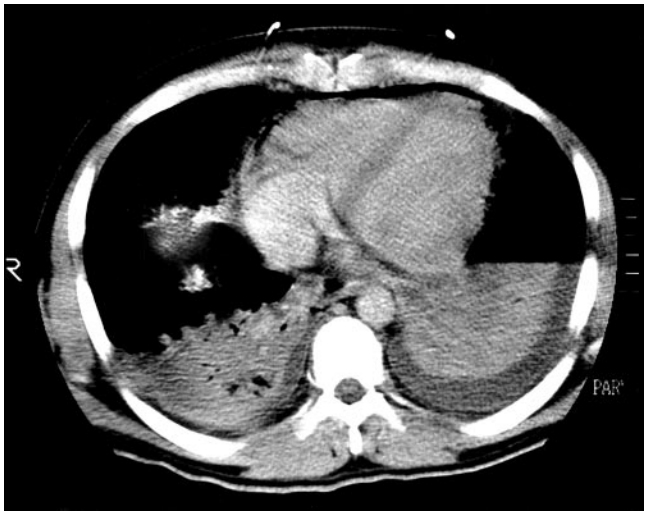

**Fig. 2. Computerized tomography (CT) scan of the chest showing bilateral pleural effusions, pulmonary consolidation, pericardial effusion, and gas in the pericardial space.**

and septicemia, requiring the prompt institution of hemodynamic support, invasive monitoring, and surgical intervention.

This case illustrates three points. First, the presence of abnormal chest radiograph findings (widened mediastinum, blunted costophrenic angles, pulmonary infiltrates) in a patient with peritonsillar abscess should lead the physician to suspect caudad spread of the process. Second, repeated airway manipulations may lead to swelling and airway deterioration. Limits of time and number of attempts limit should be imposed, followed by tracheostomy if necessary. In this patient, intubation was difficult because of moderate anatomic distortion of the airway and abundant secretions. The secretions tended to obscure the view through the fiber-optic bronchoscope and interfered with our ability to adequately anesthetize the glottis with the "spray as you go" technique (dilution of the local anesthetic). On each unsuccessful fiber-optic attempt, the glottis could be visualized, but introducing the scope past the glottis was prevented by glottic closure (inadequate anesthesia). For obvious reasons, we were unwilling to perform nerve blocks of the upper airway, and it was only after multiple "sprays" with 4% lidocaine that we were able to anesthetize the glottis. We do not believe that airway manipulation caused the empyema and cardiac tamponade; these processes were present before our involvement. We do believe, however, that the intubation attempts contributed to the rapid development of edema and erythema of the neck by disrupting the cervical fascial planes. In the future treatment of such patients, we are likely to use 20–30 min as a time limit with a maximum of 3 or 4 intubation attempts, depending on patient condition. Third, this case serves as a reminder that the airway management of those with pharyngeal infections, however critical and difficult, may represent only the first step in the total treatment of such patients.

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