

Laryngeal Candidiasis: An Uncommon Cause of Postoperative Stridor

O Kocyigit, S Celebi, H Gonen, A Turkoz, R Turkoz, G Arslan

Citation

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Abstract

We report a case of laryngeal candidiasis that caused postextubation stridor following cardiac surgery. A 3.5-year-old boy presented with cough. Anomalous supracardiac-type total pulmonary venous return, an atrial septal defect, and pulmonary hypertension were identified by echocardiogram. Surgery for correction of the anomaly was done, and the patient developed a stridor and respiratory distress. Standard therapy for postextubation stridor was ineffective. Laryngoscopic examination demonstrated the existence of ulcerated white lesions involving the larynx. Fungal culture of the lesions grew *Candida albicans*. The patient was treated with systemic antifungal medications and had complete resolution of her symptoms. This case demonstrates the importance of the differential diagnosis of postextubation stridor as it requires specific therapy.

INTRODUCTION

C. albicans is normally a commensal organism in the upper airway and gastrointestinal tract in humans. It can become pathogenic in response to treatment of patients with antibiotics, steroids, chemotherapy, or radiotherapy. Laryngeal candidiasis generally develops secondary to pulmonary or pharyngeal infection,¹ and the most common symptoms are hoarseness, dysphagia, and inspiratory stridor.² Here we present a case of stridor secondary to postoperative laryngeal candidiasis in a child treated for anomalous total pulmonary venous return.

CASE REPORT

A 3.5-year-old boy was admitted to our hospital with a cough. His medical history was notable for lung infections that had been treated with antibiotics. Results of a physical examination showed that the oropharynx was clear, the palatopharyngeal plica was mildly hyperemic, a grade 2/6 systolic murmur was present at the left second and third intercostal levels, and the liver was enlarged. The patient was cyanotic, and the saturation of peripheral oxygen value (SpO₂) was 81%. His body weight was less than the 10th percentile. Sinusal tachycardia, left atrial deviation, and right ventricular hypertrophy were present on electrocardiogram. A chest radiograph showed a large mediastinum and parenchymal vascularity. An echocardiogram showed anomalous supracardiac-type total pulmonary venous return, an atrial septal defect, and pulmonary hypertension.

Preoperative laboratory assay results were unremarkable.

The patient was sedated with oral midazolam 0.5 mg/kg and ketamine 5 mg/kg. Anesthesia was induced with fentanyl 10 µg/kg, vecuronium 0.12 mg/kg, and thiopental sodium 3 mg/kg via a 24-gauge cannula. The direct laryngoscopy score was grade I, and an endotracheal intubation was done with a 5.5-mm internal diameter, uncuffed, endotracheal tube. At the time of intubation, it was noted that the laryngeal mucosa was edematous and mildly hyperemic. Anesthesia was maintained with a fentanyl infusion of 10 µg/kg/h, and the patient breathed a mixture of 50% O₂ in air. A radial arterial line and an internal jugular venous catheter were inserted. Aortic cross-clamp and cardiopulmonary bypass were done at 72 minutes and 110 minutes, respectively, during surgery for total pulmonary venous return connection.

The patient was transferred to the cardiovascular surgery intensive care unit and ventilated in pressure-control mode. Hemodynamic stability was maintained with 5 µg/kg/h dopamine infusion. He was treated with cefazolin sodium 200 mg 3 times per day and amikacin 50 mg twice per day intravenously. Synchronized, intermittent, mandatory-ventilation mode was applied at the 14th postoperative hour, and the patient was extubated at the 19th postoperative hour. After extubation, blood gas values were pH: 7.43, Pco₂: 32 mm Hg, Po₂: 197 mm Hg, HCO₃: 22.6 mM, Base excess: -1.3 mM, and lactate: 0.7 mM. The arterial blood pressure

was 95/55 mm Hg, and the heart rate was 98 bpm. Oxygen was administered via a nasal cannula at a rate of 3 L/h. Chest tubes were removed at the 22nd postoperative hour. Stridor was noted at the 30th hour after extubation. Oral feeding was halted, and the treatment was changed to methylprednisolone 10 mg 4 times per day, racemic epinephrine 0.2%, and salbutamol 6 times per day, and dexamethasone 8 times per day with a nebulizer. During this time, no fever was observed, heart rate and blood pressure were in the normal ranges, SpO₂ was greater than 95%, and laboratory values were in the normal ranges, with the exception of C-reactive protein, which was 64.5 mg/dL. At the 35th hour after extubation, C-reactive protein had increased to 130 mg/dL. At the 48th hour after extubation, because standard therapy for postextubation stridor was ineffective and the clinical condition of the patient progressively deteriorated, an immediate laryngoscopy followed by a nasal fiberoptic examination of the larynx was performed by an otolaryngologist in the intensive care unit under local anesthesia and sedation. Direct laryngoscopy did not reveal severe obstruction caused by laryngeal edema. Nasal fiberoptic examination demonstrated ulcerated white lesions involving the larynx and the airway size was within the normal range. Because the lesions made us think candidiasis was present, as the corticosteroids were being tapered off antifungal therapy was begun at once (therefore 1 dose of fluconazole 6 mg/kg/day and 1 dose of amphotericin B 0.75 mg/kg/day were added to the treatment). Fungal culture of the lesions demonstrated presence of *C. albicans* 2 days later. The patient recovered quickly in response to treatment, and C-reactive protein decreased to 16.4 mg/dL. On the fifth day in the hospital, a narrow tapeworm, believed to be *Ascaris lumbricoides*, was identified in the feces. Therefore, 1 dose of levamisole 2.5 mg/kg was added to the treatment regimen. Cefazolin sodium and amikacin treatments were terminated at the end of the week, and fluconazole was continued until the 15th day after extubation. Before the patient was transferred from the intensive care unit to the clinic repeat laryngoscopy demonstrated near complete resolution of finding. The patient was discharged from the hospital on the 10th day with only mild hoarseness and stable hemodynamics. At the end of the first postoperative month, he recovered without hoarseness, and results of a physical examination were normal.

DISCUSSION

Stridor is caused by air flow through a narrowed respiratory tract and is the most common symptom of respiratory tract obstruction. In children, acute stridor is usually caused by

viral croup,³ the presence of a foreign body, thermal or chemical burns of the hypopharynx, angioneurotic edema, retropharyngeal abscess, or trauma.⁴

Stridor during the early postextubation period may be caused by a large intubation tube, trauma during laryngoscopy or intubation, excessive neck manipulation during surgery, hard movement of the tube, blood, mucus, the suction catheter, surgical debris or foreign bodies in the mouth, extubation cough, and preoperative or postoperative infection.^{5,6} At the 30th hour after extubation, we initiated conventional treatment in the belief that the cause of stridor in the present case was one of the above. Twenty-four hours after the initial treatment, we consulted an otolaryngologist because of standard therapy for postextubation stridor was ineffective and clinical patient progressively deteriorated.

Laryngeal candidiasis is usually seen secondary to pulmonary or pharyngeal infection, and it can be superficial or can infiltrate the epithelium. In the present case, microbiologic examination of the white lesions observed at bronchoscopy showed fungal cells, and the patient recovered dramatically in response to antifungal treatment. The medical history included lung infections that were treated, and physical examination showed no evidence of infection, with the exception that the laryngeal mucosa was hyperemic and edematous. Postoperative fiberoptic examination demonstrated to have an ulcerated white lesions involving the larynx, but the trachea and vocal cords were in normal anatomic positions. To our knowledge, this is the first report of invasive laryngeal candidiasis resulting in stridor in a child who has undergone surgery for pulmonary venous return reconstruction.

Sakakura and associates evaluated causes of chronic stridor in 55 children and reported that the most frequent causes were laryngomalacia (36.4%), subglottic stenosis (30.9%), and vocal cord paralysis (29.1%).⁴ Thirty-one of the patients showed comorbidity, the most frequent being atrial septal defect, patent ductus arteriosus, ventricular septal defect, pulmonary hypertension, and gastrointestinal tract disease, including duodenal atresia and rectocloacal fistula, along with other entities such as intracranial hemorrhage and mental retardation. Metin and associates reported a case of isolated mucosal laryngeal candidiasis in a 70-year-old man caused by long-term antibiotic use and cigarette smoking in addition to dyspnea.⁷

This case demonstrates an unusual cause of stridor in a child. Primary causes of stridor include undefined *Ascaris*

infection in rare cases, open heart surgery, malnutrition, stress due to surgery or anesthesia, antibiotic therapy, and steroid inhalation therapy. Secondary stridor can be caused by invasive candidiasis. In the differential diagnosis of the patients with progressive deterioration of clinical conditions ineffective therapy of postextubation stridor should be considered as fungal reasons.

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Author Information

Ozgen Ilgaz Kocyigit, MD

Department of Anesthesiology, Baskent University Istanbul Teaching and Medical Research Center, Istanbul, Turkey

Serdar Celebi, MD

Department of Anesthesiology, Baskent University Istanbul Teaching and Medical Research Center, Istanbul, Turkey

Hulya Gonen, MD

Department of Anesthesiology, Baskent University Istanbul Teaching and Medical Research Center, Istanbul, Turkey

Ayda Turkoz, MD

Department of Anesthesiology, Baskent University Istanbul Teaching and Medical Research Center, Istanbul, Turkey

Riza Turkoz, MD

Department of Cardiovascular Surgery, Baskent University Istanbul Teaching and Medical Research Center, Istanbul, Turkey

Gulnaz Arslan, MD

Department of Anesthesiology, Baskent University Istanbul Teaching and Medical Research Center, Istanbul, Turkey