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Case Report

Non-fatal extensive cerebral venous thrombosis as a complication of adenotonsillectomy

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ABSTRACT

Adenotonsillectomy, a common ambulatory surgical procedure performed in the pediatric population, may at times lead to serious postoperative complications.

The case of a 10-year-old with extensive cerebral venous thrombosis (CVT) following routine adenotonsillectomy is presented and the likely risk factors are discussed. Recent literature regarding CVT in children will be reviewed.

To our knowledge, there are no previous reports in the Otolaryngology literature of extensive CVT as a complication of adenotonsillectomy. This clinical entity is more common than previously thought. Awareness and a high index of suspicion and initiation of timely management can reduce the risk of potentially fatal outcomes.

1. Introduction

Adenotonsillectomy is one of the most common ambulatory surgical procedures performed in the pediatric population [1]. Sleep disordered breathing, obstructive sleep apnea and recurrent throat infection are the most common indications, all of which can significantly impact a child's quality of life [1]. Despite its routine nature, throat pain, postoperative nausea and vomiting, delayed feeding and postoperative hemorrhage are some of the more commonly encountered adverse outcomes [2–4]. However, there are rare but serious and possibly life threatening complications that may arise. Awareness of all potential complications and appropriate recognition by the responsible surgeon will help to ensure proper patient management and the avoidance of possibly disastrous patient outcomes.

Following is a case report of a 10-year-old child that underwent routine uncomplicated adenotonsillectomy for obstructive sleep apnea, who presented on postoperative day 14 with worsening headache and persistent emesis and was found to have extensive cerebral venous thrombosis (CVT) on CT imaging. This is a very rare yet serious complication. This case is reviewed as well as the possible contributing risk factors resulting in this adverse outcome. An overview of the presentation, management and outcome of cerebral venous thrombosis in children as pertaining to this case will also be discussed. Appropriate informed consent was obtained from the patient and his parents prior to the report of this case.

2. Case report

LB was a 10-year-old boy when he presented to the Pediatric Otolaryngology Clinic at BC Children's Hospital with sleep disordered breathing with associated morning headaches and daytime sleepiness, nasal congestion, snoring and mouth breathing. He was obese, with a weight of 49.9 kg (98% percentile) and his blood pressure was in the upper limits of normal (110/80). Oropharyngeal examination showed bilateral grade 3 tonsillar hypertrophy. Fiberoptic nasal endoscopy revealed moderately enlarged adenoids. He was referred to Pediatric Respirology for a polysomnogram, which demonstrated severe obstructive sleep apnea with an Apnea Hypopnea index (AHI) of 92.5 with associated oxygen desaturations as low as 58%.

Past medical history included birth by C-section secondary to failure to progress and possible chorioamnionitis. There were no complications from birth and he was discharged home without delay. He was otherwise healthy and had met all his age-appropriate developmental milestones. Family history was significant for thalassemia minor in his maternal uncle but there was no family history of deep venous thrombosis, pulmonary embolisms or autoimmune conditions. He had previously used fluticasone

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nasal spray for more than a year without significant improvement in his chronic symptom of nasal obstruction and congestion.

The patient underwent a routine adenotonsillectomy at the Children's Hospital, with the use of bipolar cautery for the tonsils and monopolar suction cautery for the adenoids. Monopolar cautery was not used during tonsillectomy. Topical 0.5% bupivacaine on soaked patties was placed on the tonsil beds for 2 min at the end of the procedure. The child had a planned overnight stay in the Pediatric Intensive Care Unit for monitoring. Overnight post-operatively, he had no desaturations. He had an excellent appetite and minimal discomfort on postoperative day (POD) 1. He was found to have a low-grade fever and was discharged home on a ten-day course of amoxicillin and clavulanic acid.

Following discharge however, his oral intake decreased and he developed intermittent frontal headaches with nausea and vomiting starting on POD 3. He presented to a local Community Emergency Department (ED) on POD 6 and again on POD 7 due to persistence of his symptoms as well as neck discomfort and was admitted for intravenous fluid hydration. He presented on POD 14 to BC Children's Hospital with worsening headache radiating to his occiput and persistent emesis. He had no noted weakness, sensory loss, visual changes, ataxia, seizure episodes, or changes in his mental status.

On admission, he was alert and oriented, and had an unremarkable cardiac, respiratory, abdominal and dermatological exam. Oropharyngeal examination revealed expected postoperative changes with small amounts of granulation tissue in the tonsillar fossae. No neck edema or tenderness was demonstrated. On neurological examination, he had normal gait, cranial nerve function, tone, power and reflexes in all four extremities and there was no sign of meningismus. Mild papilledema, left greater than right, was noted on ophthalmologic exam. His visual acuity was 20/20 bilaterally with normal color vision.

A CT scan with contrast of the head and neck was performed which showed extensive cerebral venous sinus thrombosis involving the length of the sagittal sinus through the right transverse sinus, sigmoid sinus and jugular bulb down to the right internal jugular vein to the level of C3, approximately 4 cm below the skull base (see Figs. 1 and 2). Although the left jugular vein and sigmoid sinus were patent, thrombus was also seen in the left transverse sinus, straight sinus to the vein of Galen and in both internal cerebral veins. There was no evidence of focal cerebral edema or hemorrhage, and the third and lateral ventricles were of normal size, shape and position. There were no signs of thrombophlebitis.

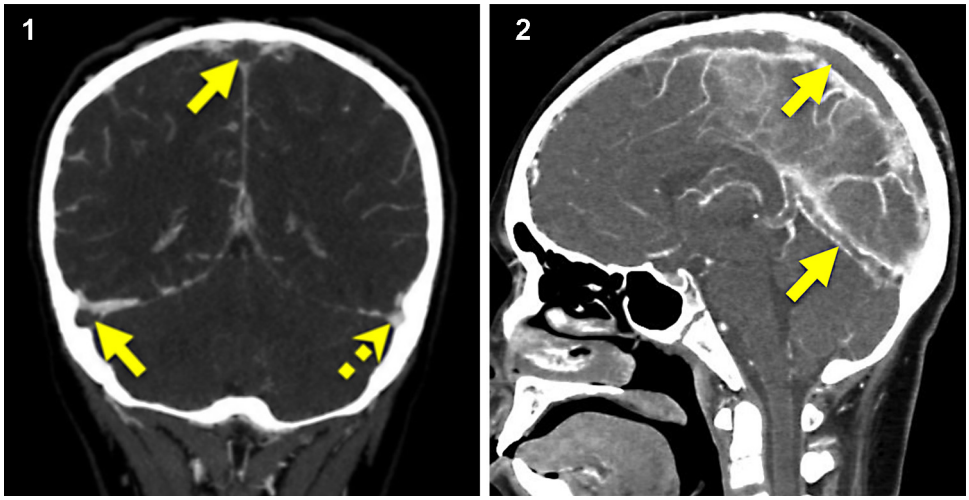
His blood work showed a white count of 16.9 (normal range (NR) 3.9–10.2), hemoglobin of 135 (NR 118–146), platelets of 440 (NR 180–440). PT was 11.8 (NR 9.6–12), INR 1.09 (NR 0.87–1.11), APTT 22.5 (NR 22.5–28.1), dilute Russell's viper venom time 1.03 (NR 0.76–1.15) and fibrinogen level 4.06 (N 1.68–4.4). His anti-cardiolipin antibody test was negative, and his antithrombin III and homocysteine levels were normal. Protein C activity was slightly elevated at 1.4 but was reported as not clinically significant by hematopathology. Protein S activity level was normal and testing for Factor V leiden and prothrombin (Factor II) mutations were negative. A blood film showed non-specific poikilocytosis and microcytic anemia in favor of iron deficiency.

The Pediatric Neurology and Pediatric Hematology services were consulted. A lumbar puncture was performed which revealed increased intracranial pressures (opening pressure of 42 cm H_2O). The CSF gram stain and culture were negative. The patient was started on prophylactic anti-seizure medication (levetiracetam 500 mg p.o. BID). He was started on acetazolamide 325 mg p.o. TID for elevated intracranial pressure. Enoxaparin (Low Molecular Weight Heparin) was initiated for his extensive cerebral venous thrombosis, which was titrated to a therapeutic goal (anti-Xa level of 0.5–1). He was discharged home on POD 19 as he had improved symptomatically and was able to tolerate oral hydration with a plan to continue anticoagulation for a 3-month duration. Follow-up was arranged with the Neurology and Otolaryngology team at BC Children's Hospital for reassessment and re-imaging to determine whether an additional 3 months of therapy would be required.

On follow-up he was noted to have decreased headache frequency and severity. He had no associated nausea or emesis since discharge and his papilledema had improved. His repeat imaging at 3 months revealed resolution of his cerebral venous thrombosis (see Figs. 3 and 4) and his repeat polysomnography demonstrated a dramatic improvement in his sleep apnea, with an AHI of 7.4 compared to his pre-operative AHI of 92.5.

3. Discussion

Cerebral venous thrombosis is a serious yet very rare complication following adenotonsillectomy. There are only a few case reports in the literature that have reported on this complication, with the most recent being a case of superior sagittal thrombosis and cortical infarction reported by Reilly et al. in 2006, and a case of jugular thrombosis post tonsillectomy



Figs. 1 and 2. CT scan at the time of presentation. CT scan of the head and neck with contrast (coronal and sagittal views) demonstrating extensive cerebral venous sinus thrombosis involving the sagittal sinus, the right transverse sinus and the straight sinus (block arrows) with a patent left transverse sinus (dotted arrow).