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An unexpected cause of upper airway obstruction

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Abstract
A three-year-old boy with a swelling on the right side of his neck was suspected of having a parapharyngeal abscess after clinical examination and CT scan (computed tomography scan) of this region. Later it became clear, that the swelling was caused by an aneurysm of the internal carotid artery. This case report describes the pitfalls and difficulties encountered in the diagnostic course and treatment planning.

Key words: Neck; Aneurysm; Carotid artery, internal

Introduction

A peritonsillar abscess and also a parapharyngeal abscess, are possible complications of bacterial tonsillitis. They commonly occur in young adults. If children present with signs of these forms of abscess, they must be investigated, because these abscesses rarely occur in children.

Case report

A three-year-old boy developed fever with signs of an upper airway infection and subsequently swelling of the right side of his neck. There was no history of trauma. In the first instance the diagnosis was a peritonsillar infiltrate on the right accompanied by a torticollis caused by cervical lymphadenitis. Treatment with antibiotics was commenced. Two days later protrusion of the tongue developed, the diagnosis of a peritonsillar abscess was made and it was decided to perform a tonsillectomy. During the operation no pus was aspirated. After the operation the swelling increased in size and an upper airway obstruction developed, so it was thought inappropriate to extubate the patient.

A CT scan was performed (Figure 1) which demonstrated a large tumour in the infratemporal region on the right measuring 3 × 5 × 6 cm containing a large vascular structure. The tumour almost totally obstructed the pharynx and was diagnosed as a parapharyngeal abscess containing a swollen jugular vein.

The child was then transferred to paediatric intensive care with a request to drain the abscess. He had a large, firm swelling on the right side of his neck, which was slightly mobile but pulsating. On and around the mass hyperplastic lymphnodes were palpable. The CT scan suggested the possibility that there was an aneurysm of the internal carotid artery. A DSA (digital subtraction angiography) was performed which showed an aneurysm of the internal carotid artery on the right side (Figure 2). Ultrasound imaging of the mass was reported as consistent with an abscess cavity filled with debris or an organized haematoma.

Laboratory investigations showed an ESR of 18 mm/h and a leucocytosis of 13 with a left shift. Coagulation was normal as were electrolytes, kidney and liver function tests. One day after admission, selective angiography was performed and it showed a 1.5 cm saccular aneurysm of the internal carotid artery just below the skull base. There was a complete Circle of Willis (Figure 3).

After extensive interdisciplinary consultation it was decided to pursue an expectant policy in the first instance and to follow the process radiologically. The plan was to explore the mass later when it had settled, in the hope that it would be possible then to excise the aneurysm without having to sacrifice the internal carotid artery. This expectant policy was chosen because it was uncertain, whether control of the extracranial distal end of the carotid artery could be obtained during the operation, since it reached the base of the skull. MRI (magnetic resonance imaging) two days after admission was difficult to interpret because of the tube and ventilation, but there was some evidence of a

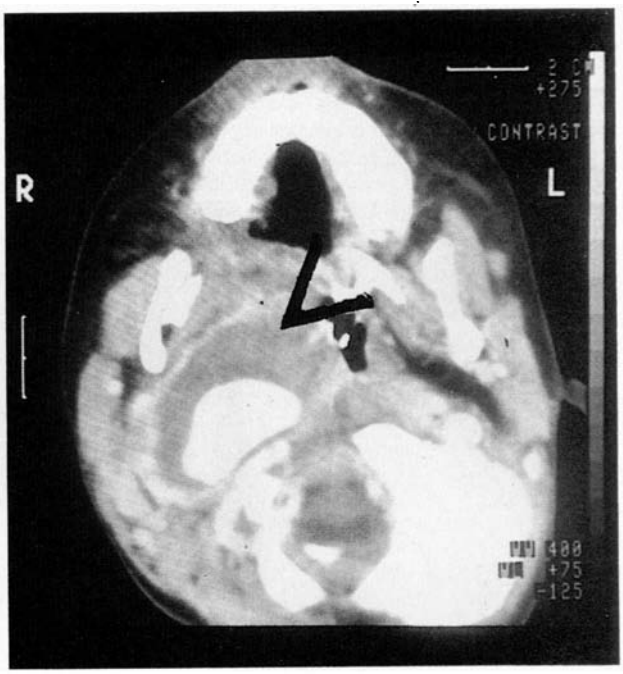

Fig. 1

CT scan with contrast: the arrow indicates the cavity filled with blood, which in the first instance was attributed to a swollen jugular vein.

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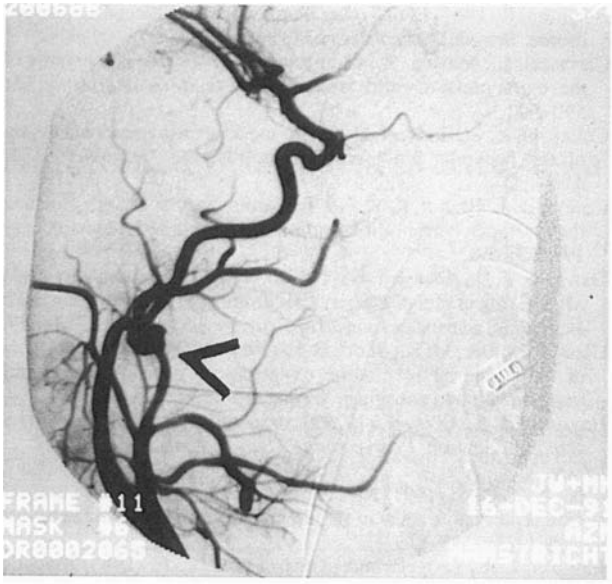

FIG. 2

Digital subtraction angiography showing a saccular aneurysm of the internal artery (arrowed).

fresh thrombus. According to the radiologist this was more consistent with a thrombosing vascular cavity than a mycotic aneurysm, because in the latter case one would expect a lobulated and better defined wall. Ten days after admission an attempt to extubate the patient was not successful and paralysis of the right vocal fold was noted, probably as a result of neuropraxia of the vagus nerve running through the mass.

Two weeks after admission, bleeding from the oropharynx developed. This was controlled intraorally and the neck was explored as an emergency. Very close to the skull base the internal carotid artery was shown to be connected to a large aneurysm. Bleeding necessitated ligation of the internal carotid artery both distally and proximally to the mass. The part of the carotid artery which was resected, appeared normal, but was sent for histology. The cavity of the aneurysm appeared to be filled with clots but no pus and no true wall of the aneurysm was found. During the operation a tracheostomy was performed. No signs of inflammation were found in the artery wall.

Post-operatively Horner's syndrome on the right was noted, but no other signs of neurological deficit were found. A CT scan of the brain showed no infarction. Two weeks after this operation the patient was decanulated successfully and no serious problems arose. At the time of discharge some speech and swallowing problems remained and speech therapy was commenced. Two months later the mobility of the right vocal fold was normal and Horner's syndrome had almost disappeared.

It should be noted that this patient had received speech therapy before this incident because of speech problems: he was talking very little and what he was saying was neither clear nor loud enough. He also had swallowing problems mainly confined to liquids.

Discussion

This case report illustrates that an aneurysm of the internal carotid artery can mimic a peritonsillar or parapharyngeal abscess (Rensburg, 1964). The precise cause of the aneurysm in our case remains uncertain. The criteria used for the diagnosis are discussed below.

(1) *False aneurysm of the internal carotid artery caused by a parapharyngeal abscess.* A false aneurysm (Colley and Clark, 1980) originates from a rupture of the wall of an artery, caused by trauma or infection. In such an aneurysm the wall is composed of connective tissue formed by neighbouring organs and tissue and

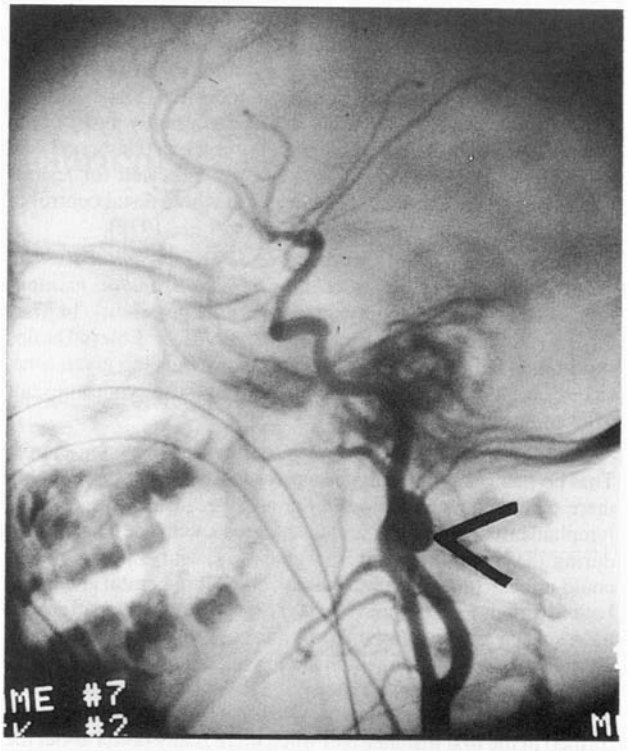

FIG. 3

Angiography showing the aneurysm against the skull base (arrowed).

not by arterial tissue. Therefore there is a likely chance of rupture because of lack of elastic tissue (Carrascal *et al.*, 1978). Spontaneous resolution is not very likely and an operation is necessary. In our case a false aneurysm could have been caused by a parapharyngeal abscess, which caused erosion of the internal carotid artery and subsequent bleeding into the abscess cavity. It commonly occurs in the carotid artery close to the bifurcation. Signs can occur gradually as a result of compression of neighbouring structures e.g. the vagus nerve, but can also occur suddenly in the form of a rupture. In the case of a parapharyngeal localization of the abscess, the bleeding is commonly into the oropharynx but sometimes through the parotid gland to the external ear canal.

(2) *Mycotic aneurysm as a result of infection of the internal carotid artery by a parapharyngeal abscess.* Definitions of a mycotic aneurysm are variable but usually an aneurysm of infectious aetiology is described (Johnson *et al.*, 1983). When it is bacterial contamination of an existing aneurysm we speak of 'infected aneurysm'. A mycotic aneurysm is rare but with a high incidence of morbidity and mortality. The first case was described by Osler (1885) concerning a patient suffering from bacterial endocarditis who developed multiple aneurysms. This misleading term was introduced by Osler (1885) because the lumen of the artery resembled a fungal mycelium. Later on it was demonstrated to be a bacterial infection. The infection can be caused by an intravascular source (like a bacteraemia) or an extravascular source, (like an infection in neighbouring tissue) or iatrogenic. Vessels affected by atherosclerosis are predisposed to septic emboli. This disease occurred more frequently before the antibiotic era and was most often caused by bacterial endocarditis occasionally by syphilis or tuberculosis. These days it is encountered in peripheral vessels as a result of intravascular drug abuse, or of *Salmonella spp.* infections (Jebara *et al.*, 1991; Dawson *et al.*, 1992). In the case of a mycotic aneurysm the carotid artery is seldom affected (Ledgerwood and Lucas, 1974). The signs of a mycotic aneurysm consist of a painful, pulsating and expansive swelling and fever. It is agreed that treatment with a high dose of antibiotics intravenously is necessary (O'Connor

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et al., 1972; Slade-Howell, 1977). However the wall of the aneurysm is fairly resistant to antibiotics, hence a resection of the aneurysm is sometimes necessary. Primary repair or repair by means of a graft is usually not very successful (Slade-Howell, 1977; Purdue *et al.*, 1981). This means that usually a part of the artery together with the aneurysm is resected (Haywood and Ellis, 1979). An expectant policy is only indicated for lesions difficult to reach e.g. the base of the skull where distal control of the bleeding can be difficult (O'Connor *et al.*, 1972).

(3) *Bleeding of a congenital (caused by a congenital disease) or acquired aneurysm (caused by infection and/or manipulation).* A congenital aneurysm was another possibility. In Marfan's syndrome (Cotton and Brandt, 1976) or Ehlers-Danlos syndrome (Beighton, 1968) aneurysms can develop given time. In our case however there were no signs of these syndromes nor of fibromuscular dysplasia.

In the case described several questions remain unanswered. That no pus was found suggests there was no abscess. Probably there had been an infection in the area because of the cervical lymphadenitis, high fever and leucocytosis with a left shift. That during the operation no clear wall of an aneurysm was found, could indicate that there was no mycotic or congenital aneurysm but only a false aneurysm. The fact however, that the patient did have speech and swallowing problems before this period, could point to an existing congenital aneurysm and existing paresis of the vagus nerve. Parapharyngeal abcesses rarely occur in children but signs should be investigated. A CT scan should be performed in the first instance and when there is any doubt about the vascular origin of the swelling, selective angiography. It is important also to be informed about the completeness of the Circle of Willis in case it is necessary to sacrifice the internal carotid artery. In children however, neurological deficits after ligation of the internal carotid artery very rarely occur. This is shown in studies of the developmental outcome in children after extracorporal membrane oxygenation (ECMO), which allows temporary cardiopulmonary support for high-risk children who have severe respiratory failure (Krummel *et al.*, 1984; Glass *et al.*, 1989; Adolph *et al.*, 1990). During this technique the right common carotid artery and jugular vein are ligated. It was shown in these studies there was significant neuromotor abnormality or developmental delay after one year in two to 10 per cent of the children, which could be related to hypoxia and acidosis before ECMO, to severe intracranial haemorrhage or to chronic lung disease. Ligation was not associated with a lateralizing lesion.

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