South African Medical Journal : Suid-Afrikaanse Tydskrif vir Geneeskunde

Cape Town, 30 July 1960 Volume 34 No. 31 Deel 34 Kaapstad, 30 Julie 1960

SEVERE EPISTAXIS DUE TO A LEAKING EXTRACRANIAL ANEURYSM OF THE INTERNAL CAROTID ARTERY

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It is taught in the ENT Department of this hospital that a child with epistaxis severe enough to warrant admission is not suffering from a purely local cause. Usually such a child will be found to be a case of one of the exanthemata, particularly the prodrome of measles or typhoid or more rarely acute rheumatic fever, acute or chronic nephritis, or one of the blood dyscrasias. The case described below was to some extent exceptional.

R.G., a Coloured male aged 3 years, was admitted on 12 April 1957 with a history of having fallen on his nose 1 week previously with a resulting severe epistaxis. On the morning of admission he had a further sharp epistaxis.

On admission he was not bleeding but looked pale, with a haemoglobin reading of 7 g.%. He was given 250 c.c. of blood and next morning he looked well, with a haemoglobin of 12 g.%.

On examination no general cause for the bleeding was found. He was apyrexial and there was no apparent enlargement of the lymph glands or spleen. A blood smear showed mild hypochromia, 13,500 white blood cells per c.mm. with a normal differential count, no abnormal white cells, and plentiful platelets. The urine was normal. Nothing abnormal was found on anterior rhinoscopy.

On 15 April he had a second severe epistaxis, which ceased spontaneously before the resident house surgeon could get to the child, but 400 c.c. of blood, a third of his estimated blood volume, was needed to resuscitate him. At this stage a diagnosis of secondary haemorrhage due to a fractured nose was still tenuously held.

On 22 April a further sharp epistaxis occurred; the child was shocked and again 400 c.c. of blood was given.

X-ray showed a nasopharyngeal shadow, possibly adenoids or a nasopharyngeal angiofibroma.

On 23 April an examination under anaesthesia showed a red granular protrusion 3 mm. in diameter, surrounded by a pulsatile mass 2·5 cm. long, in the posterolateral nasopharyngeal wall on the right. On careful palpation behind the angle of the jaw more prominent pulsation was felt on the right then on the left. A diagnosis of leaking aneurysm, probably of the right internal carotid, was made.

Blood was kept available, with a cut-down tray, at the bedside. The Wassermann reaction was subsequently found to be negative.

On 29 April Dr. F. van Niekerk, of the Department of Neurosurgery, carried out a right carotid angiogram, which showed an aneurysm of the internal carotid at the base of the skull (Figs. 1 and 2). This was followed immediately by ligation and division of the internal carotid artery just above its origin.

Beyond a post-operative Horner's syndrome on the right side, which has since disappeared, the child showed no neurological signs at all, and when last seen in August 1959 he was mentally and physically normal and active. He had had no further epistaxis and no mass remained in the nasopharynx.

DISCUSSION

On looking back at this case, the diagnosis appears simple, for epistaxis so rapidly exsanguinating could only be due to rupture of a major vessel, aneurysm or angioma. The commonest cause is rupture into the nasopharynx of an aneurysm of the internal carotid; others are rupture of an intracranial internal carotid aneurysm through the sphenoid sinus,[1] and possibly rupture of vessels in a juvenile naso-

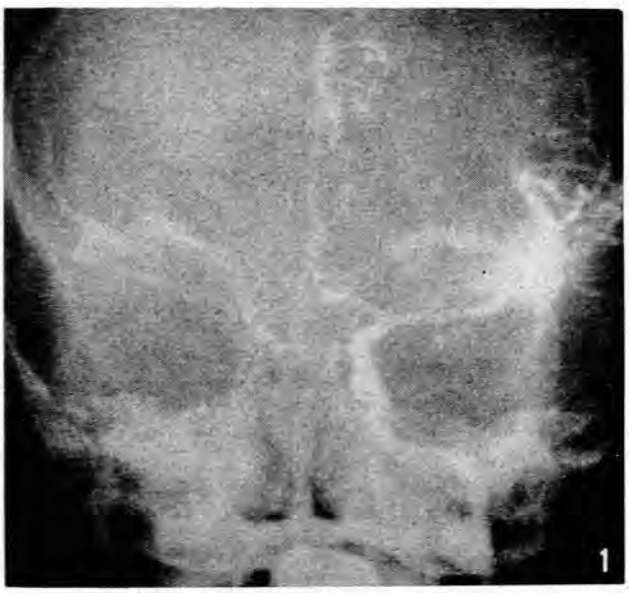

Fig. 1. Right carotid angiogram, anteroposterior.

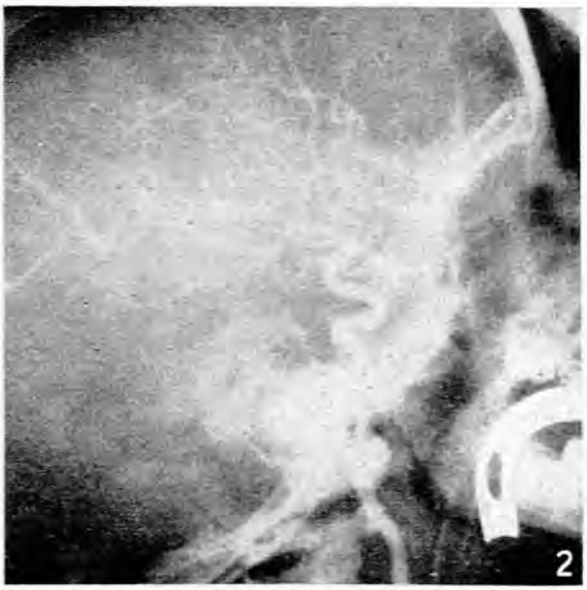

Fig. 2. Right carotis angiogram, lateral.

pharyngeal fibro-angioma. However, at the time, it did not seem so patently obvious and we felt ourselves lucky in demonstrating the aneurysm without provoking further haemorrhage.

Young,[2] in 1941, described 2 cases and reviewed the previous literature. Apart from war injuries the majority of the cases described were children and followed a typical course. One child developed an acute upper-respiratory-tract infection, diagnosed as scarlatina, and a parapharyngeal abscess followed, with subsequent septic erosion of the carotid sheath. An aneurysm or pseudo-aneurysm formed, which later ruptured into the external auditory meatus or pharynx. Of 28 cases, 11 survived, and in 7 of these ligation of the internal carotid had been necessary.

Silcox and Updegrove[3] reported an arteriosclerotic aneurysm in an adult and reviewed 85 cases of extracranial aneurysm of the internal carotid, of whom all but 2 were under 15 years of age.

Pierini and Agra[4] described a case of aneurysm of syphilitic origin of the petrous portion of the carotid, with probable rupture into the eustachian tube and epistaxis.

The aetiology of the case reported here remains in doubt; there was no history of parapharyngeal abscess, there was no cause for mycotic aneurysm, the Wassermann reaction was negative, and the patient was too young for atheroma. There was a history of trauma, but this may have been conjectural in view of the epistaxis. The suggestion that the child fell with a sharp foreign body in the mouth which penetrated the pharynx was negated by the absence of any scar and by the fact that the aneurysm was above the soft palate. Possibly the aneurysm was congenital.

The commonest treatment has been proximal ligation of the internal carotid artery. The obvious danger is ischaemic cerebral damage. However, McCall *et al.*[5], quoting de Fourmestraux (1906), state that the mortality rate for ligation of the carotid in aneurysm is 13·3% and in haemorrhage 54%. A low mortality in aneurysms, they state, is confirmed by Dandy's figure of 5% (1935) and that of Matas of 4% (1940). Schorstein[6] explains this by showing that in elective ligation of the internal carotid for intracranial aneurysm in young persons the morbidity is low, while in cases where the operation is performed when the patient is shocked, with a slow cerebral circulation, the mortality is extremely high.

The artery is divided between ligatures to avoid pulsation of the proximal segment which may dislodge any thrombus that has formed distal to the ligature.[4]

To try and avoid neurological sequelae, Matas applied pressure over the skin to the carotid and observed the results. We were not prepared to attempt this, in case pressure high enough to occlude the internal carotid caused further haemorrhage, and Dandy's exposure of the artery under local anaesthesia and compression[7] was not considered practicable in a 3-year-old child.

Ligation of the common carotid, allowing retrograde flow to the internal carotid from the external carotid, was felt to be uncertain of cure and yet not without danger.

Resection and graft would hardly have been possible, even in the hands of the most experienced vascular surgeon, owing to the high level of the upper anastomosis.

CONCLUSIONS

1. Leaking aneurysm of the internal carotid artery as a cause of epistaxis, though uncommon, should be considered in sudden gross epistaxis.

2. The treatment is replacement of blood lost, and ligation of the internal carotid artery when the blood pressure is normal again.

3. The possible neurological sequelae may be foreseen and perhaps avoided by prior compression of the internal carotid for several minutes; while an EEG may be used as a monitor.[5]

SUMMARY

A case is described of rupture of a congenital aneurysm of the internal carotid artery at the base of the skull in a 3-year-old child.

Ligation of the internal carotid artery was performed, with subsequent cure and no neurological sequelae.

The condition is discussed and conclusions drawn.

I wish to thank Dr. J. G. Burger, the Superintendent of Groote Schuur Hospital and Dr. D. J. Roux, head of the Department of Otorhinolaryngology for permission to publish this case. Dr. H. Maisels, then the House Surgeon, performed prompt and life-saving transfusion of the child on several occasions.

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