

Surgical treatment of angiofibromas of the nasopharynx - 34 cases

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SUMMARY

Between 1966 and 1987 we have treated 34 angiofibromas. The patients were between 9 to 28 years of age (average 16,5 years). The tumour extension was determined formerly by tomography and angiography, but these two methods have been replaced by angioscanning, which give the best results. Nineteen patients were classified stage I, fourteen stage II and one stage III. The surgical approach was transmaxillary in most cases. After tumour resection, two local recurrences were seen and a second operation was necessary to achieve a definitive cure in these patients.

INTRODUCTION

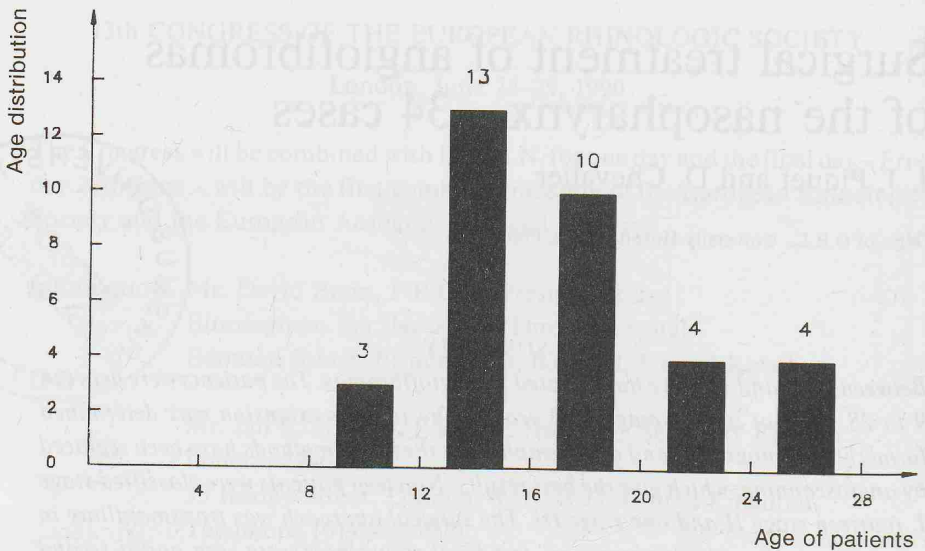
The treatment of a nasopharyngeal angiofibroma still remains a controversial subject, because of the many modalities. Nevertheless, the introduction of less aggressive methods of investigation makes it easier to determine the extension of the tumour. It is generally agreed that surgery is the first choice when the tumour is easy to extirpate, while over the extended forms discussion continues.

CLINICAL PRESENTATION

Between 1966 and 1987 we treated 34 angiofibromas. All of these patients were investigated and operated upon by the same surgeon, resulting in a homogenous series.

The incidence of this disease seems to be increasing, because we treated 14 cases between 1966 and 1976 and 20 cases between 1977 and 1986. We think that this relative augmentation in frequency of occurrence is due to more precise and easier diagnosis with CT-scan.

All of our patients were males, aged between 9 and 28 years. The mean age was 16 years and 6 months, but eight of our 34 patients (27%) were older than 20 years (Figure 1). The relatively late diagnosis would seem due to the absence of symp-



toms, i.e. lack of pain, only minor epistaxis. Among the most frequently observed clinical signs was nose obstruction 28/34 (82%). This symptom precedes epistaxis, which occurred in only 25/34 (73%) of the patients. Severe haemorrhages are exceptional. We encountered a few cases of abundant bleeding as the only symptom. This seldom led to the correct diagnosis. We think that the disappearance of severe bleeding is due to the frequent use of antibiotics that eliminates infection of the tumour.

Hearing loss due to otitis media with effusion was found four times (11%). In two cases the angiofibroma was discovered as a result of meningeal problems, e.g. purulent meningitis and a meningeal irritation syndrome.

GRADE OF TUMOUR EXTENSION

Arising in contact with the pterygoid hamulus, in the upper lateral part of the cavum, the angiofibroma will expand into the regions of least resistance, which are the cavum and the nasal fossa. Deep penetration can occur into the inferior temporal fossa and the orbita. Finally it can extend upperwards and backwards into the sphenoid sinus, the cavernous sinus and the floor of the temporal lobe. These latter extensions are particularly dangerous, because of vascular problems during surgery.

The different extensions were very difficult to explore by means of classical radiography, thus angiography became inevitable. This method of examination

demands a well trained team. Even in the best circumstances the risks remain. In our series, it was only used in tumours with major extension, i.e. 21 cases (62%), and it was complicated by transient unilateral brachiooplegia.

Since 1978, the extension has been demonstrated by scanning using iodine injection. This technique offers many advantages (Hill, 1985; Hoover, 1983). It is non-invasive and can be repeated when desired. Pooling of the contrast confirms the diagnosis of an angiofibroma.

The tumour extension is perfectly delineated in all the danger zones, e.g. inferior temporal fossa, cavernous sinus, meninges, apex of temporal bone (Table 1).

Table 1. Extension of angiofibromas.

cavum	34 (100%)
inferior temporal fossa	23 (67%)
sphenoid	10 (29%)
ethmoid	10 (29%)
penetration meninges	2 (6%)
apex petrosal bone	1 (3%)

Extension into the inferior temporal fossa, in particular, was notably frequent. Pre-operatively it was found in 67% of cases, but at surgery, the percentage rose to 90%. The intracranial extension is one of the most discussed points during the evolution of the angiofibroma. Its frequency has been reported as being 10 to 20% of cases (Bremer, 1986). This would seem to us to be too high, because in our series there was no intracranial invasion and the dura was exposed in only 6% of cases. Jones (1986) has recently made a complete study of this problem. He states that normally the tumour remains extradural. In a survey of the literature between 1967 and 1986 Jones found only one case of 84 in which dural penetration occurred. This study confirms our opinion that intracranial invasion is very exceptional. If we use Session's classification (1981), our series seems very homogeneous, with very few areas in which extension occurred (Table 2).

Table 2. Classification according to Session.

I = 19
IIA = 8
IIB = 6*
IIC = 0
III = 1

* One patient with penetration of the dura.

TREATMENT

Pre-operative treatment

There are certain forms of medication that reduce the volume of bleeding from the tumour. We have no experience with normal hormonal or chemical therapy

(Goepfert, 1985). We used embolisation in four cases before 1976, but this method seemed dangerous, so we abandoned it. It may have some value in cases where the tumour is very voluminous or recurrent (Natvig, 1984).

Surgery

The objective is total removal of the tumour with all its prolongations together with primary haemostasis of the internal maxillary artery, so that significant bleeding can be avoided. It has to include systematic inspection of the inferior temporal fossa which is very often invaded by the tumour.

Several approaches are to be discussed: The palatal approach, recently advised by Economou (1988), seems to us rather restricted. It does not allow either the internal maxillary artery or the inferior temporal fossa to be reached. Thus we have never used it.

The transfacial approach is advised by Harrison (1987). It gives a direct view of the maxillary sinus and the nasal fossa, but it necessitates excision of the anterior bony wall which can leave aesthetical sequelae (Cannoni, 1985; Hadjean, 1985). The creation of a bony panel is described, which avoids this disadvantage because the panel is replaced at the end of the procedure.

The external route, nevertheless, has the disadvantages of leaving a cutaneous scar and the possibility of stenosis of the lacrimal duct. In our opinion it should be reserved for very voluminous tumours.

The transmaxillary approach that we advised leaves no cutaneous scar because it involves a gingival incision (Piquet, 1985). A firm periosteal flap is created up to the inferior orbital nerve, then the anterior wall of the maxillary sinus is removed as in the operation described by Denker. The gouge followed by the burr are used to perform an extensive osseous resection.

The maxillary sinus is opened and the medial as well as the posterior walls are resected. The internal maxillary artery is clamped.

The tumour is then progressively detached and its pterygoidal insertion pole is loosened. The tumour is then removed by gentle traction.

The use of the microscope in focus 300 permits careful inspection of the cavity. It enabled the detection in two cases, of a prolongation situated behind the pterygoidal hamulus, that could be removed, thus avoiding a recurrence.

The transmaxillary approach was used in 31 patients, in two cases we used the external route, because in a young child the gingival approach is too narrow.

Postoperative care was carried out for five years. We never used systematic angiography. We confirmed ourselves to clinical examination, with, in some cases, a CT-scan one year after surgery.

We diagnosed two local recurrences (6%), which have been reoperated successfully.

Radiotherapy

Proposed by Cummings (1984) in Canada, it would seem of interest in very voluminous tumours with an extension in particular to the apex of the temporal bone or the cavernous sinus. We used it in one case. The patient had a very large extension in this region. Irradiation with a dose of 45 Gray was used. It achieved very marked regression of the tumour over a period of six months. Unfortunately the tumour started growing again and we had to operate in the ninth month. The transmaxillary approach was used successfully.

DISCUSSION

Voluminous angiofibromas with extension to the cavernous sinus and the apex of the temporal bone still pose great problems with regard to treatment. There are two possibilities:

1. Excision by the transfacial approach with resection of the tumour as completely as possible, keeping the possibility of radiotherapy in mind, in the event of deep recurrences.
2. Primary radiotherapy with view to an eventual surgical resection when a recurrence occurs.

The choice between these two alternatives is difficult because we have to take into account the age of the patient. Bearing in mind the dangers of surgery within the irradiated areas, we feel that primary surgery is to be advocated, possibly followed by radiotherapy should removal of the tumour prove incomplete or a recurrence occurs. The angio-scanners facilitates follow-up and enables early detection of a recurrence which can then be irradiated under favourable conditions.

In conclusion, we continue to perform surgical removal of angiofibromas. The other forms of therapy, particularly radiotherapy, seem only to be indicated in cases of recurrences whose surgical removal presents difficulties.

REFERENCES

1. Bremer JW, Neel HB, De Santo LW. Angiofibroma treatment trends in 150 patients during 40 years. *Laryngoscope* 1986; 96: 1321-1329.
2. Cannoni M, Pech A, Zanaret M. Les volets fronto-naso-maxillaires. *J Fr Oto-Rhino-Laryngol* 1985; 34: 409-415.
3. Cummings BJ, Blend R, Keane T, Fitzpatrick P. Primary radiation therapy for juvenile nasopharyngeal angiofibroma. *Laryngoscope* 1984; 94: 1599-1605.
4. Economou TS, Abemayor E, Ward PH. Juvenile angiofibroma. *Laryngoscope* 1988; 98: 170-175.
5. Goepfert H, Cangir A, Lee YY. Chemotherapy for aggressive juvenile nasopharyngeal angiofibroma. *Arch Otolaryngol* 1985; 111: 285-289.
6. Hadjean E, Klap O, Thurel A, Sicot H, Tran Ba Huy P. Le volet transfacial dans la chirurgie des tumeurs des tiers moyens et postérieurs de la face et de la base du crâne. *Annls Oto-Lar (Paris)* 1985; 102: 481-485.

7. Harrison DF. The natural history, pathogenesis and treatment of juvenile angiofibroma. *Arch Otolaryngol* 1987; 113: 936-942.
8. Hill JH, Mafee MF, Lygizos NA. Dynamic computed tomography. Its use in assessment of vascular malformations and angiofibroma. *Arch Otolaryngol* 1985; 111: 62-65.
9. Hoover LA, Hanafee WN. Differential diagnosis of tumours by computed tomography scanning. *Arch Otolaryngol* 1983; 109: 43-47.
10. Jones GC, De Santo LW, Bremer JW, Neel HB. Juvenile angiofibromas. Behaviour and treatment of extensive and residual tumours. *Arch Otolaryngol* 1986; 112: 1191-1193.
11. Natvig K, Scalpe IO. Pre-operative embolization of juvenile nasopharyngeal angiofibromas with gelfoam. *J Lar Otol* 1984; 98: 829-833.
12. Piquet JJ, Vaneecloo FM, Moreau P. Fibrome naso-pharyngien à propos de 29 cas. *Acta Oto-Rhino-Laryng Belg* 1985; 39: 994-1000.
13. Session RB, Bryan RN, Naclerio RM, Alford BR. Radiographic staging of juvenile angiofibroma. *Head Neck Surg* 1981; T3: 279-283.

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