The Journal of Laryngology and Otology
February 1999, Vol. **113,** pp. 127–134

Juvenile angiofibroma: the lessons of 20 years of modern imaging

GLYN LLOYD, D.M., F.R.C.R., DAVID HOWARD, B.Sc., F.R.C.S., PETER PHELPS, M.D., F.R.C.S., F.R.C.R., ANTHONY CHEESMAN, B.Sc., F.R.C.S.

Abstract

Seventy-two patients with juvenile angiofibroma have been investigated by computerized tomography (CT) and/or magnetic resonance imaging (MRI) over a period of 20 years. The evidence from these studies indicates that angiofibroma takes origin in the pterygo-palatine fossa at the aperture of the pterygoid (vidian) canal. An important extension of the tumour is posteriorly along the pterygoid canal with invasion of the cancellous bone of the pterygoid base, and greater wing of the sphenoid (60 per cent of patients). Distinctive features of angiofibroma are the high recurrence rate, and the rapidity with which many tumours recur. It is postulated that the principal determinant of recurrence is a high tumour growth rate at the time of surgery coupled with incomplete surgical excision. The inability to remove the tumour *in toto* is principally due to deep invasion of the sphenoid, as described above. In this series 93 per cent of recurrences occurred with this type of tumour extension. A contributory cause in these patients is the use of pre-operative embolization. The treatment implications of these findings are examined.

Key words: Angiofibroma; Tomography scanners, X-ray computed; Magnetic resonance imaging

Introduction

Juvenile angiofibroma is a rare benign, but unencapsulated and highly vascular tumour occurring in adolescent males. The juvenile nasopharyngeal angiofibroma was so-called because it was formerly thought to arise in the nasopharynx. Why these tumours occur almost exclusively in a restricted age group and restricted site at the base of the skull is unknown, or whether they should be considered as neoplasms or hamartomas. According to Acuna (1956) the condition was known to, and treated by Hippocrates but the first authentic case treated by surgery was reported by Liston (1841) and verified as angiofibroma by Myhre and Michaels (1987) from histological sections made from the original operative specimen. Chelius (1847) noted the fibrous nature of the lesion and its occurrence at about the time of puberty, and Gosselin (1873), emphasized the occurrence of nasopharyngeal fibrous polyps exclusively in young males, and noted that while some lesions tend to regress as the patient becomes adult, others required surgical removal. The term juvenile nasopharyngeal fibroma was introduced by Chauveau (1906), and Friedberg (1940) suggested the name angiofibroma. Following these descriptions a large volume of literature has accumulated about this relatively rare condition.

Since the decade of the 1970s angiofibroma has been much better demonstrated pre-operatively both in site and extent by the introduction of CT scanning and MRI. The latter investigation is especially important for identifying tumour recurrence, a conspicuous feature of the natural history of angiofibroma. The purpose of this review is to illustrate the application of these techniques, their contribution to our understanding of angiofibroma and their influence on clinical management.

Materials and methods

There were 72 patients with histologically verified angiofibroma. These were seen in the 20-year period starting in March 1978 when biplane CT became available. All were males between the ages of six and 26 years with an average age of 15.9 years. There were 28 known recurrences with multiple recurrence in 10. All patients were investigated by CT or MRI or both (30 per cent). In general, the bone changes were best shown by CT and soft tissue extent by MRI. All tumours demonstrated by the latter method showed the presence of signal voids to a greater or lesser extent in the tumour mass due to its vascular nature; and for the same reason strong enhancement was invariably shown on MR after intravenous gadolinium.

From the The Royal National Throat, Nose and Ear Hospital, Gray's Inn Road, London, UK.
Accepted for publication: 16 November 1998.

TABLE I
DISTRIBUTION OF 72 JUVENILE ANGIOFIBROMATA AT THE SKULL BASE

1	A soft tissue mass in the nose or nasopharynx.	100%
2	Mass in the pterygo-palatine fossa.	100%
3	Erosion of the bone of the posterior margin of sphenopalatine foramen extending to the base of the medial pterygoid plate.	100%
4	Enlargement or erosion of the vidian canal.	96%
5	Extension to the sphenoid sinus.	83%
6	Enlargement of the pterygo-maxillary fissure and extension to the infratemporal fossa.	64%
7	Invasion of the sphenoid bone.	60%
8	Pressure erosion of the sphenoid.	40%
9	Orbit invaded.	27%
10	Extension to the middle fossa of the skull.	17%

The distribution of these tumours at the skull base is listed in Table I.

Discussion

Site of origin

In this series the tumour was consistently present in 100 per cent of patients in the pterygo-palatine fossa and the nasal cavity, but was not always shown at other sites at the skull base: for example the nasopharynx or infratemporal fossa (Table I). In addition, all patients with satisfactory pre-operative coronal CT scans showed an erosion of the posterior margin of the sphenopalatine foramen and the bone

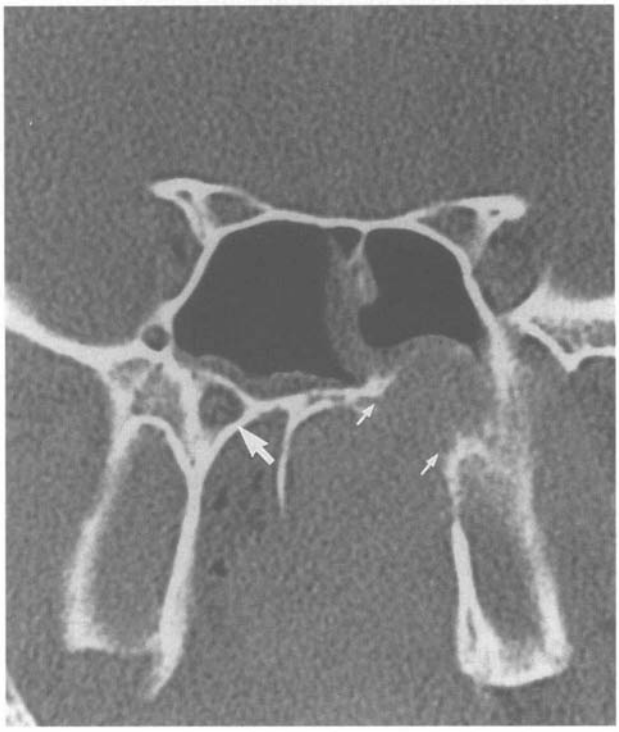


FIG. 1

Coronal CT at the level of the pterygoids. On the normal side (right) the recess in the pterygo-palatine fossa at the anterior aperture of the pterygoid canal is demonstrated (large arrow). This is the likely site of origin of angiofibroma. On the left an angiofibroma has expanded this area and eroded the bone of the medial wall of the recess (small arrows), formed by the sphenoidal process of the palatine bone and the medial pterygoid lamina.

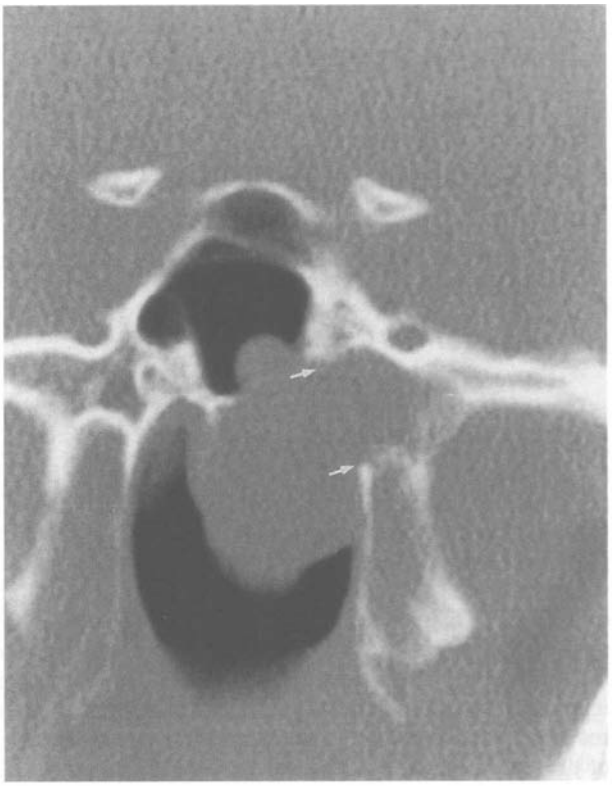


FIG. 2

Coronal CT showing the characteristic changes of angiofibroma. Soft tissue mass encroaching on the air space with erosion of bone behind the sphenopalatine foramen (arrows) and early invasion of the sphenoid sinus.

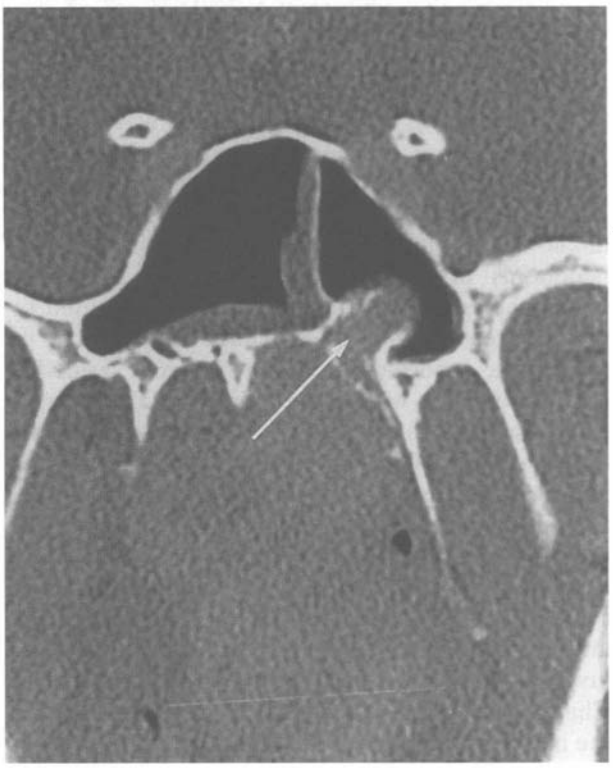


FIG. 3

Same patient as Figure 1. Coronal CT at the level of the sphenoid sinus showing enlargement of the pterygoid (vidian) canal by angiofibroma (arrow).

https://doi.org/10.1017/S0022215100143373 Published online by Cambridge University Press

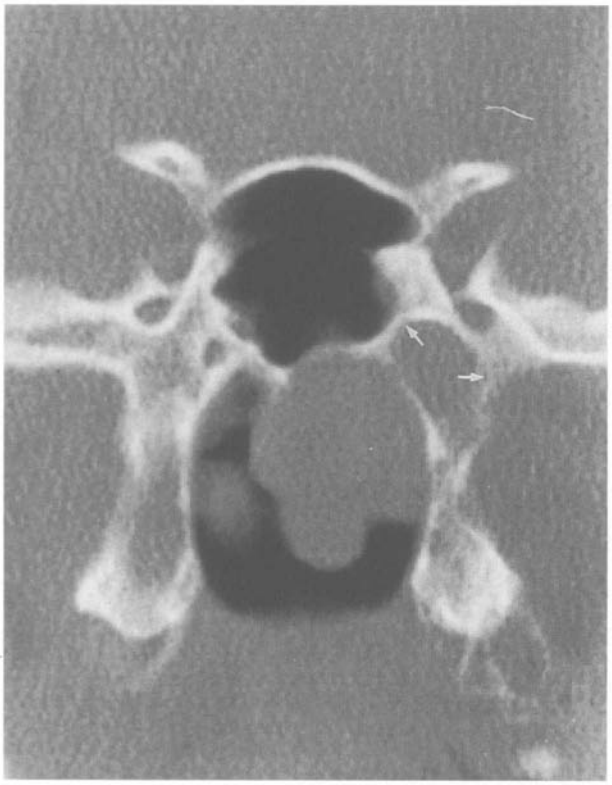


FIG. 4

Coronal CT section immediately anterior to Figure 2, showing expansion of the pterygoid canal (arrows), and the soft tissue mass of the angiofibroma in the posterior nasal air space.

behind, formed by the sphenoidal process of the palatine bone and medial pterygoid lamina (Figures 1 and 2). A further clue to the probable site of origin was the presence of bone changes in the forward part of the pterygoid (vidian) canal and pterygoid lamina. In 56 patients with satisfactory CT studies of the canal it was shown to be enlarged or eroded in 54 (96 per cent) (Figures 3, 4 and 5). The presumption is that angiofibroma arises in the pterygo-palatine fossa in the recess behind the sphenopalatine ganglion, at the exit aperture of the pterygoid canal (Figures 1 and 6). By the time the tumour has enlarged enough to give rise to symptoms, it will have expanded medially into the nasal cavity via the sphenopalatine foramen, and by erosion of the palatine bone forming the medial boundary of the recess. At this stage the tumour presents diagnostic features on CT (Vide infra).

Diagnosis

The diagnosis of angiofibroma is made clinically by the history of a young or adolescent male with nasal obstruction, epistaxis or both, and the presence of a soft tissue mass in the nose or nasopharynx; expansion of the tumour may lead to facial deformity and proptosis if the orbit is invaded. Imaging is required for confirmation, but when there is any doubt about the clinical diagnosis the radiologist may be needed to identify the angiofibroma pre-operatively. With such a vascular tumour severe bleeding may accompany biopsy and for this reason surgeons

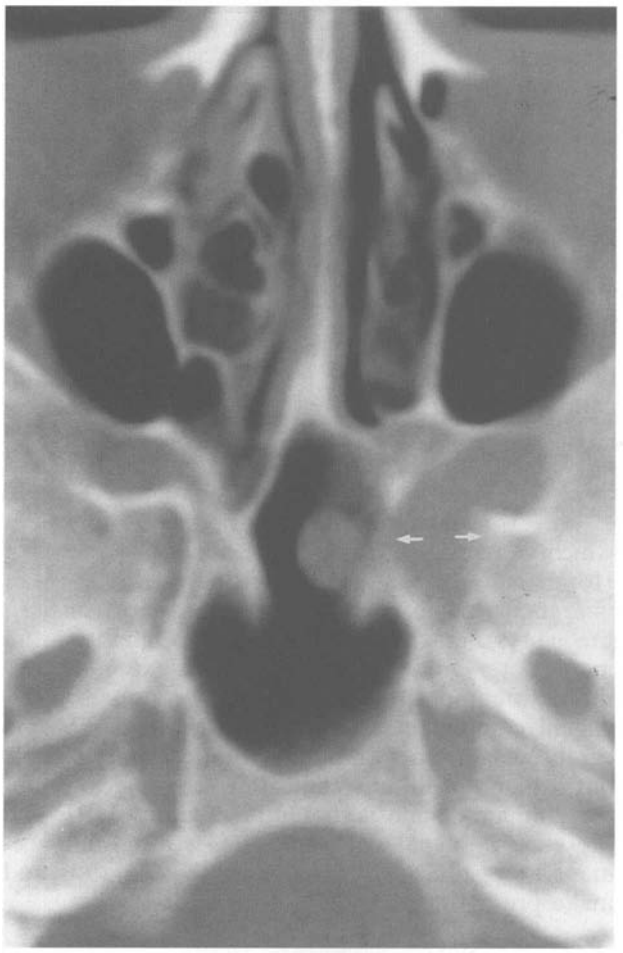


FIG. 5

Same patient as Figure 4. Axial CT also shows expansion of the pterygo-palatine recess and pterygoid canal (arrows).

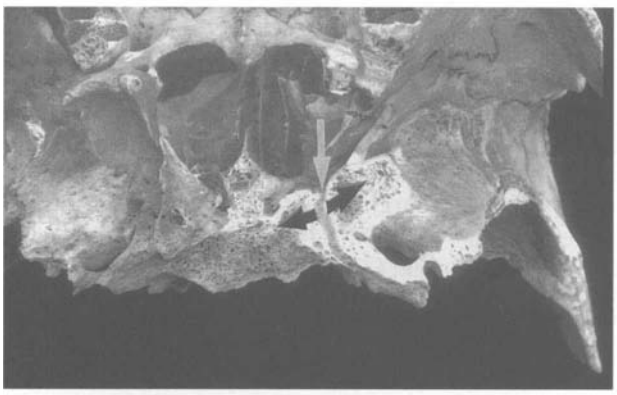


FIG. 6

Axial section of the left sphenoid bone made on a dried skull at the level of the pterygoid canal. The white arrow indicates the most probable site of origin of angiofibroma. The black arrows indicate the route of invasion of the cancellous bone of the pterygoid base. Medial to the pterygoid canal the vaginal process of the sphenoid may be destroyed by external pressure erosion or by direct invasion, but the important direction of spread of angiofibroma is laterally with invasion and expansion of the body and greater wing of the sphenoid (see Figure 8).

https://doi.org/10.1017/S0022215100143373 Published online by Cambridge University Press

are reluctant to undertake biopsy of a nasopharyngeal mass in an adolescent male patient and prefer to rely upon imaging methods to decide whether the mass is likely to be an angiofibroma or a non-vascular lesion such as an antro-choanal polyp. Diagnosis on CT is based upon the site of origin of the lesion as described above and the two constant features: namely a mass in the nose and pterygo-palatine fossa and erosion of bone behind the spheno-palatine foramen at the root of the pterygoid plate. This erosion is readily demonstrated by coronal CT (Figures 1 and 2) and is diagnostic of angiofibroma. Polyps or tumours not taking origin from this site are unlikely to present this change.

To be positive these distinguishing signs of angiofibroma depend upon invasion of the nasal cavity by the tumour (100 per cent in this series). However, there are examples in the literature in which there was no mass in the nose or nasopharynx. Hora and Weller (1961) described an extra-nasopharyngeal angiofibroma with apparent origin from the pterygo-maxillary space and attachment to the medial pterygoid plate. The tumour extended into the infratemporal fossa but there was no nasopharyngeal component. With this caveat angiofibroma can be diagnosed with almost total certainty by CT, but if there is still any clinical or radiological doubt the ultimate diagnosis should be made by MRI. The presence of signal voids and very strong enhancement after gadolinium will confirm the diagnosis, and make pre-operative biopsy unnecessary.

Extension of angiofibroma

To show the pre-operative extension of angiofibroma both CT and MRI are employed – the former to show bone changes and the latter to show the soft tissue extent. Of the two, CT is the more important pre-operatively. Angiofibroma can be successfully managed by CT only, prior to initial surgery. The complexity of the bony structures at the skull base invaded by angiofibroma, demands the best bone imaging available and this can only be provided by CT. From its site of origin in the pterygo-palatine fossa the expanding tumour grows medially into the area of least resistance – the nasal cavity and nasopharynx, enlarging the spheno-palatine foramen and eroding the bone behind it. Forward enlargement indents the postero-superior border of the maxillary antrum, giving rise to the so called 'antral sign' described by Holman and Miller (1965). This consists of an anterior bowing of the posterior antral wall seen on lateral plain X-ray or axial CT. The tumour can also gain access to the infratemporal fossa by lateral extension via the pterygo-maxillary fissure and invade the apex of the orbit through the inferior orbital fissure. From here, lying outside the rectus muscle cone, it may extend to the middle fossa of the skull through the superior orbital fissure.

Growth of tumour posteriorly occurs along the line of the pterygoid canal eroding or invading the base of the pterygoid process. Two varieties of this extension can be recognized: in the first there is a simple pressure erosion of the pterygoid base and

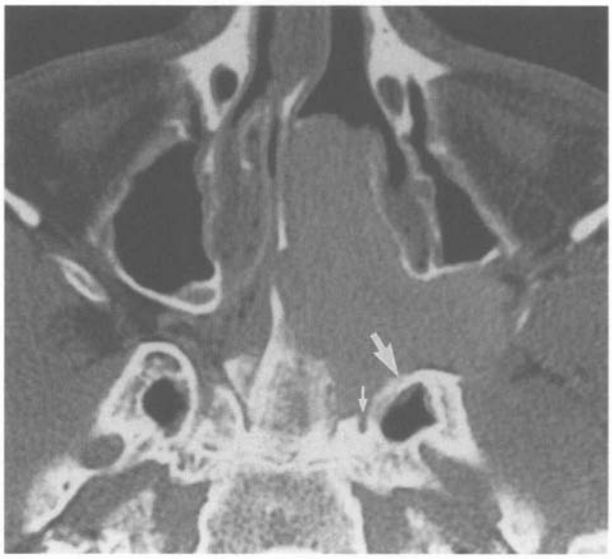


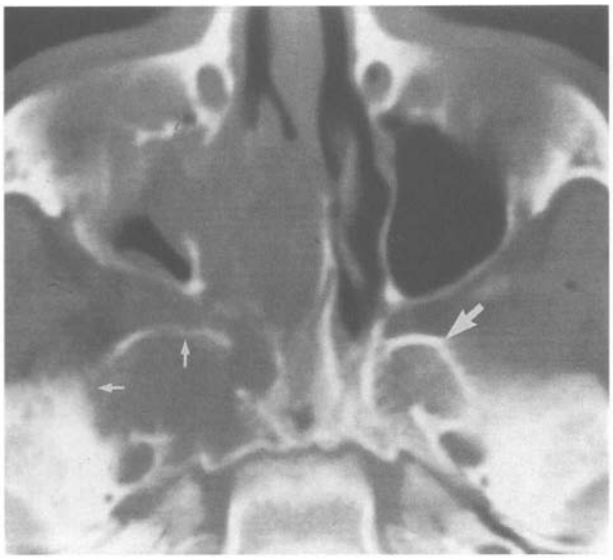
FIG. 7

Axial CT scan of the skull base showing angiofibroma in the nose and pterygo-palatine fossa. There is displacement and pressure erosion of the pterygoid process (large arrow) without invasion of the sphenoid or expansion. The pterygoid canal is eroded but not expanded (small arrow).

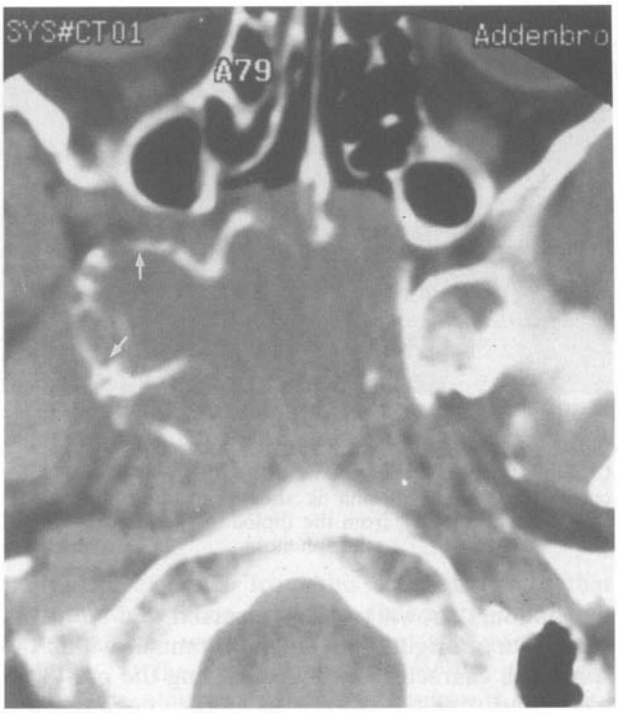
the vaginal process of the sphenoid, often with backward and lateral displacement of the pterygoids (Figure 7), but without invasion of the pterygoid base or body of the sphenoid; the tumour is largely exophytic within the nasal cavity. In the second variety there is deep extension into the cancellous bone at the base of the pterygoid process often with expansion and invasion of the diploë of the greater wing of the sphenoid and in some patients invasion of the middle fossa (Figures 8a, b, c, d and Figure 9). This provides a second route of intracranial extension to that via the orbit (see above).

Recurrence of angiofibroma

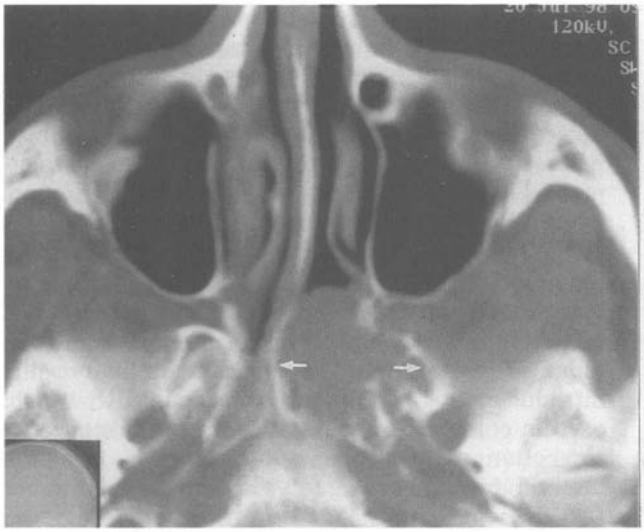
Recurrence is a distinctive feature of angiofibroma. In this series 39.5 per cent of tumours recurred with a multiple recurrence rate of 13.9 per cent and a time to recurrence varying from four months to three years. Forty-six and a half per cent of recurrences presented within 12 months of initial surgery. Gullane *et al.* (1992) recorded a 36 per cent recurrence rate for tumours treated by primary surgery and 57 per cent for those receiving initial radiotherapy, while Harma (1959) from a group of 49 patients with this condition found a recurrence rate of 46 per cent with multiple recurrences in 28 per cent. It was notable that half of the recurrences occurred within eight months of the completion of the preceding treatment (surgery or radiotherapy) and 90 per cent within two years. Other authors (Laffargue, 1947) have observed that some tumours do not recur at all, while others operated on by the same surgeon, by the same method and under similar conditions recur with astonishing rapidity. The combination of these findings would indicate that the principal determinant of recurrence is tumour growth rate coupled with incomplete surgical exci-



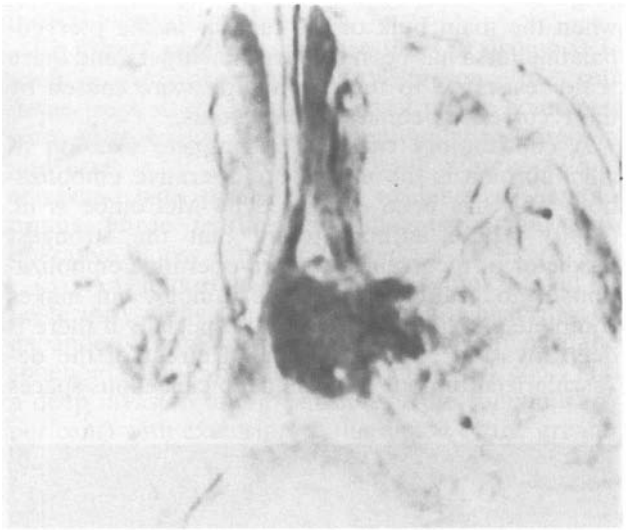
(a)



(b)



(c)



(d)

FIG. 8

Three examples of sphenoid invasion by angiofibroma: a) Axial CT of the skull base showing expansion of the pterygoid base and greater wing of the sphenoid (small white arrows), anterior to the foramen ovale. The normal appearance is shown on the opposite side (arrow). b) Axial CT: expansion of the base of the pterygoid process by angiofibroma. c) Third example of angiofibroma invading and expanding the base of the pterygoid process on axial CT (arrows). d) Corresponding axial section to 8(c) shown by subtraction MRI. The soft tissue extent of the angiofibroma is demonstrated in the pterygoid base.

sion. When initial surgery happens to coincide with early maximum growth rate, recurrence is almost inevitable, if there is anything less than total removal at initial surgery.

Total removal of angiofibroma is made particularly difficult if there is deep invasion of the skull base, so that the disposition of the tumour, as shown on pre-operative imaging, has an influence on the recurrence rate. Invasion and expansion of the cancellous bone at the base of the pterygoid process, as described above, is closely associated with a high recurrence rate. Ninety-three per cent of recurrences were found in this group, and multiple recurrences were only associated with this type of tumour extension. In contrast only seven per cent of recurrences occurred in the group which eroded, but did not invade the sphenoid.

Additional evidence of the association of deep invasion of the sphenoid and tumour recurrence is provided by follow-up CT and subtraction MR: all

https://doi.org/10.1017/S0022215100143373 Published online by Cambridge University Press

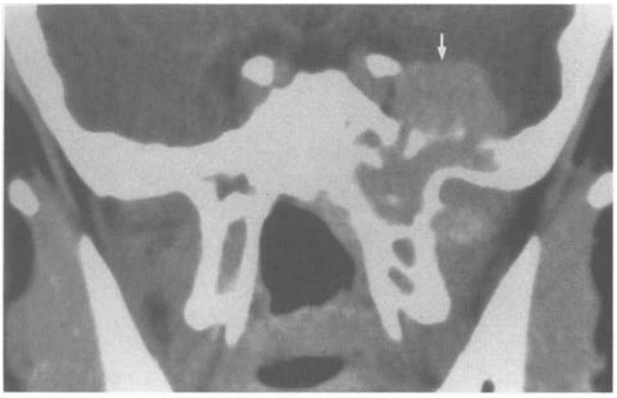


FIG. 9

Coronal CT section at the level of the pterygoids. After contrast the angiofibroma is shown invading the middle cranial fossa (arrow), from the diploë of the greater wing of the sphenoid.

the tumours shown to have recurred, did so at the initial site of origin of the angiofibroma or within the sphenoid, characteristically expanding the pterygoid base and greater wing. In some patients this expansion of the pterygoid process produces a forward displacement of the pterygoid laminae (Figures 10 and 11) on the post-operative scans, when the main bulk of the tumour in the pterygo-palatine fossa has been removed at surgery and there is no resistance to the forward pressure caused by the recurrence behind the pterygoids.

A contributory cause of incomplete excision of angiofibroma is the use of pre-operative embolization. This has been explained by McCombe *et al.* (1990). These authors found that the strongest predictor of recurrence was pre-operative embolization. Embolization shrinks the tumour, but makes complete excision more difficult especially if there is deep invasion of the sphenoid. Retreat of the devascularized tumour into the deep cancellous spaces

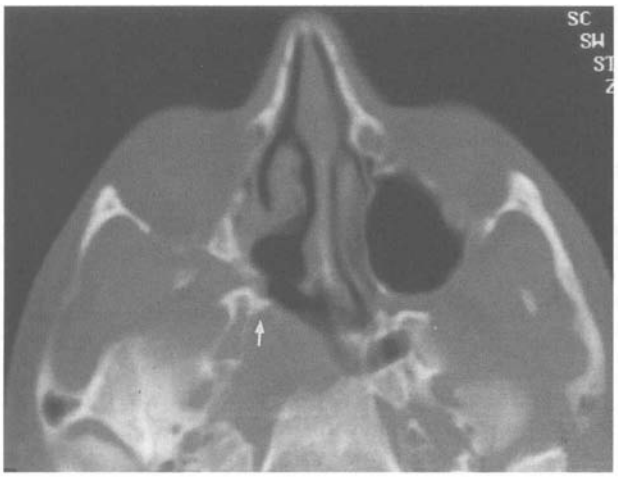


FIG. 10

Recurrence of angiofibroma. Axial CT of the skull base showing forward displacement of the pterygoid laminae (arrow): Previous surgery has removed the tumour from the pterygo-palatine fossa, and there is no resistance to the pressure of the recurrence behind the pterygoids.

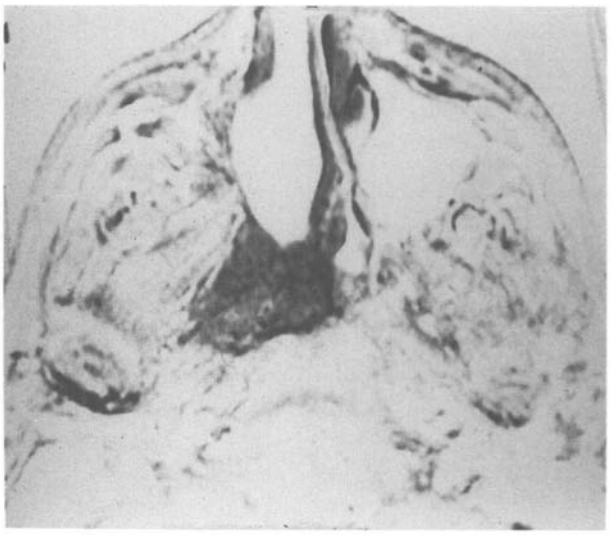


FIG. 11

Subtraction gadolinium-enhanced MRI. Same patient as Figure 10. The corresponding axial section shows the soft tissue extent of the recurrence.

of the sphenoid allows a large and rapid recurrence once the tumour is re-vascularized in the immediate post-operative period.

Spontaneous regression of angiofibroma has been documented primarily in patients with residual or recurrent disease (Gullane *et al.*, 1992). One authentic example of spontaneous regression has however been demonstrated in an 11-year-old boy, whose parents refused to allow any form of treatment (Weprin and Siemers, 1991). Twelve years later CT showed complete regression and no evidence of residual tumour. No comprehensive long-term follow-up has yet been undertaken on the patients in this series to show the progress of involution after the cessation of treatment. Assessment of tumour size was inaccurate before the introduction of gadolinium-enhanced MRI, which, used in combination with fat suppression sequences or better photographic subtraction, is now the method of choice (Figures 8d and 11). However two patients, prior to the availability of these techniques, did show evidence of tumour regression on CT. One has been reported previously (Stansbie and Phelps, 1986). This was a single recurrence that occurred 16 months after initial surgery, and showed some reduction in tumour size on follow-up CT. More recently a long-term follow-up has been obtained on a patient who in 1985 had a double tumour recurrence and was treated by embolization, surgery and radiotherapy. On the initial CT there was a deeply invasive tumour expanding the body and greater wing of the sphenoid. In the following year post-treatment MRI without contrast demonstrated a residual tumour of some size, but a CT scan nine years later showed no evidence of tumour and almost total bone reformation in the sphenoid.

Since the introduction of subtraction MR (Lloyd and Barker, 1991) this technique has been used for follow-up of patients with angiofibroma, mostly to confirm a clinically suspected recurrence, but also to

https://doi.org/10.1017/S0022215100143373 Published online by Cambridge University Press

monitor the behaviour of the tumour following further treatment. Cessation of tumour growth after incomplete surgical removal has been clearly demonstrated, and one patient has shown minimal tumour shrinkage, but the results to date suggest that involution is a slow process and no dramatic decrease in tumour size has yet been observed.

Treatment

The mid-facial degloving operation, first published by Casson *et al.* (1974) and described by Howard and Lund (1992) has been employed in these patients for the last 10 years and is now the procedure of first choice. The approach is particularly appropriate for juvenile angiofibroma, allowing excellent exposure of the lesion, whilst avoiding an external incision. It is possible to gain excellent access to both nasal cavities, maxillary antra and thence ethmoids, sphenoids, pterygo-palatine fossa, nasopharynx and infratemporal area. Thus the posterior wall of the sphenoid sinus, pterygoid plates with attached muscles and posterior wall of nasopharynx become the posterior limits of the resection. The superior limit is formed by the cribriform plate, the roof of the ethmoids and laterally by the coronoid process of the mandible.

Ideally the degloving procedure would best be applied when tumour growth rate is slowing so that any regrowth of tumour remnant would be minimal and subclinical, but this delay is impossible at at time of rapid tumour growth when clinical and parental pressure for surgery is intense. In these circumstances, reduction of the recurrence rate is best achieved by total removal of the angiofibroma or, when this is not possible by reducing the size of any tumour remnant to a minimum. The first step in this process is to obtain a good bone CT of the sphenoid and pterygoid base. If there is simple erosion and displacement, recurrence is unlikely since degloving allows total removal of the tumour. On the other hand when there is deep invasion of the pterygoid base, with extension to the greater wing of the sphenoid, recurrence is very likely if any sizable tumour remnant is left. In this group embolization prior to surgery is likely to promote recurrence (see above) and is contraindicated, but it should be noted, this veto does not apply when there is no CT evidence of sphenoid invasion as in the former group.

For the patients with invasion of the sphenoid (60 per cent) a more rigorous surgical approach is needed. It is normal at the end of surgical excision to inspect the cavity previously involved by the angiofibroma by means of an operating microscope and to remove all obvious tumour from the skull base. In addition, it may be that recurrence rates will be markedly reduced if the base of the pterygoid process is carefully removed along with the vaginal process of the sphenoid and if necessary the diploë of the greater wing of the sphenoid.

Treatment of angiofibroma with radiation as a primary or secondary therapy has been used for many years. In recent years Cummings *et al.* (1984) from Toronto have been the most enthusiastic proponents of primary radiotherapy reporting symptomatic control in 80 per cent of 55 patients, although 50 per cent still had clinically visible tumours 12 months after treatment.

Economou *et al.* (1988) were less successful recording better symptomatic control with primary surgery, and concluding that radiotherapy should be reserved for patients with intracranial involvement. Clearly, the main problem is the risk of radiation-induced head and neck malignancy, which in the Toronto series was 3.6 per cent. This is unacceptable when the alternative of primary surgery offers little morbidity and nil mortality as in the series under review. Radiotherapy should therefore be reserved for patients showing multiple recurrencies, the object being to contain any tumour remnant after repeat surgery by slowing the growth rate.

Conclusions

(1) The evidence from this series indicates that angiofibroma takes origin in the pterygo-palatine fossa in the recess behind the spheno-palatine ganglion, at the anterior aperture of the pterygoid canal. By expansion the tumour breaks into the nose and nasopharynx via the sphenopalatine foramen and by erosion of the bone behind it, formed by the sphenoidal process of the palatine bone and the medial pterygoid plate.

(2) Based on these changes the CT diagnosis is made on coronal sections by the presence of a soft tissue mass in the posterior nasal cavity combined with enlargement of the spheno-palatine foramen and erosion of its posterior bony margin. When combined with positive MRI evidence of angiofibroma, these features are diagnostic, and pre-operative biopsy is not warranted.

(3) An important extension of the tumour is posteriorly along the pterygoid canal which may be expanded. There are two types of involvement of the sphenoid: simple pressure erosion (40 per cent) and a deep invasion and expansion of the sphenoid (60 per cent) with extension to the diploë of the greater wing and in some patients invasion of the middle fossa.

(4) It is postulated that recurrence of angiofibroma is principally determined by tumour growth rate at the time of surgery, combined with incomplete surgical excision, which is greatly dependent on the type of tumour extension. Ninety-three per cent of recurrences were found in patients in whom the tumour had invaded and expanded the sphenoid, whereas seven per cent occurred in those showing only pressure erosion without invasion. Deep invasion of the sphenoid leaves a large post-operative tumour remnant and the tumour recurs at its initial site of origin or within an expanded sphenoid. Characteristically, the latter produces a forward displacement of the pterygoid laminae on follow-up CT, after the main bulk of the tumour has been removed from the pterygo-palatine fossa at surgery.

(5) A contributory cause of recurrence is the use of pre-operative embolization. Embolization shrinks the tumour but makes total removal more difficult especially if there is deep invasion of the sphenoid.

Patients who show this on pre-operative CT are at high risk of recurrence and embolization is contra-indicated.

(6) Long term regression of angiofibroma has been recorded by CT in an untreated patient (Weprin and Siemers, 1991), but most instances of tumour involution have been documented with residual or recurrent disease. Two patients in this series showed involution on CT – one treated by surgery and radiotherapy and one by surgery alone. Cessation of tumour growth has also been clearly demonstrated using subtraction MRI, and one patient has shown minimal tumour shrinkage, but the results to date suggest that involution is a long-term process and no dramatic decrease in tumour size has yet been recorded.

(7) The above considerations influence the manner in which the imaging techniques are used. Essential pre-operative imaging is by CT bone studies to make the initial diagnosis and to show the type of sphenoid involvement. Post-operative imaging for recurrence is again initially by CT to demonstrate characteristic bone changes, in particular the forward displacement of the pterygoids and the presence of a nasal soft tissue mass. Subtraction MRI is then used to show the exact extent of the recurrence, to allow size assessment after radiotherapy or to monitor natural tumour involution.

(8) For angiofibroma the degloving operation is the surgical procedure of choice and for patients, who do not show sphenoid invasion on pre-operative CT, the tumour can be removed *in toto* without recurrence. In contrast the group of patients showing sphenoid invasion on pre-operative CT are at high risk of recurrence. For these, more radical surgery is needed to eliminate or reduce in size any tumour remnant within the sphenoid.

Radiotherapy as a primary treatment is to be avoided, so that there is no risk of radiation-induced malignancy. It should be reserved for patients with multiple recurrences, the object being to contain any tumour remnant after surgery by slowing the growth rate.

Acknowledgement

We would like to thank Mr Andrew Gardner and his staff for the illustrations.

References

Acuna, R. T. (1956) The nasopharyngeal fibroma and its treatment. *Archives of Otolaryngology* **64:** 451–455.

Casson, P. R., Bonnano, P. C., Converse, J. M. (1974) The mid-face degloving procedure. *Plastic and Reconstructive Journal* **53:** 102–113.

Chelius, M. J. (1847) *A System of Surgery*. Vol 2, Renshaw, London, p726.

Chauveau, C. (1906) *Histoire des Maladies du Pharynx*. J.B. Ballière et Fils, Paris p562.

Cummings, B. J., Blend, R., Keane, T. (1984) Primary radiation therapy for juvenile nasopharyngeal angiofibroma. *Laryngoscope* **94:** 1599–1605.

Economou, T. S., Abemayor, E., Ward, P. H. (1988) Juvenile nasopharyngeal angiofibroma. An update on the UCLA experience, 1960-1968. *Laryngoscope* **98:** 170–175.

Friedberg, S. A. (1940) Nasopharyngeal fibroma. *Archives of Otolaryngology* **31:** 313–326.

Gosselin, L. (1873) Fibrome ou polype fibreux nasophayngien. *Clinique Chirugicable De Hôpital de la Charite (Paris)* **1:** 92–116.

Gullane, P. J., Davidson, J., O'Dwyer, T., Forte, V. (1992) Juvenile angiofibroma: a review of the literature and a case series report. *Laryngoscope* **102:** 928–933.

Harma, R. A. (1959) Nasopharyngeal angiofibroma. *Acta Otolaryngologica* **(Suppl 146):** 7–74.

Holman, C. B., Miller, W. E. (1965) Juvenile nasopharyngeal angiofibroma. *American Journal of Roentgenology* **94:** 292–298.

Hora, J. F., Weller, W. A. (1961) Extranasopharyngeal juvenile angiofibroma. *Annals of Otology Rhinology and Laryngology* **70:** 164–170.

Howard, D. J., Lund, V. J. (1992) The midfacial degloving approach to sinonasal disease. *Journal of Laryngology and Otology* **106:** 1059–1062.

Laffargue, B. (1947) Considerations sur les fibrose naopharyngiens étude anatomico-clinique. *Revue de Laryngologie* **68:** 436-465; 514–539.

Liston, R. (1841) University College Hospital. Tumour of the upper jaw bone: Excision and recovery. *Lancet* **1:** 67–68.

Lloyd, G. A. S., Barker, P. B. (1991) Subtraction magnetic resonance for tumours of the skull base and sinuses: a new imaging technique. *Journal of Laryngology and Otology* **105:** 628—631.

McCombe, A., Lund, V. J., Howard, D. J. (1990) Recurrence in juvenile angiofibroma. *Rhinology* **28:** 1–6.

Myhre, M., Michaels, L. (1987) Nasopharyngeal angiofibroma treated in 1841 by maxillectomy. *Journal of Otolaryngology* **16:** 390–392.

Stansbie, J. M., Phelps, P. D. (1986) Involution of residual angiofibroma. *Journal of Laryngology and Otology* **100:** 599–603.

Weprin, L. S., Siemers, P. T. (1991) Spontaneous regression of juvenile nasopharyngeal angiofibroma. *Archives of Otolaryngology - Head and Neck Surgery* **117:** 796–799.

Address for correspondence:
Dr Glyn Lloyd,
107 Harley Street,
London W1N 7DG.