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Main Articles

Conservative management of sporadic unilateral acoustic neuromas

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Abstract

Our objective was to review retrospectively patients with a unilateral acoustic neuroma managed by observation. One hundred patients with tumours (<24 mm) were followed a median 25.5 months. Thirty-six acoustic neuromas grew with four growth patterns. No factors were associated with growth. Eighty per cent of growing tumours grew in the first year. Eleven patients proceeded to surgery. Twenty-two patients were eligible for hearing preservation surgery; five of the 15 available for analysis subsequently lost eligibility.

In conclusion, selected patients can be safely observed with serial imaging and follow up. Size increase in the first year may predict future growth. Delaying surgery until required by symptoms or tumour growth does not result in more morbidity for the patient. Some may lose the opportunity for hearing preservation surgery but operating on all would result in more sustaining a loss of hearing in the first few years after diagnosis.

Key words: Neuroma; Acoustic; Diagnostic Imaging; Disease Management; Surgical Procedures, Operative

Introduction

Acoustic neuromas (AN), more properly known as vestibular schwannomas,[1] are benign tumours arising from the VIIIth cranial nerve in the internal auditory canal and the cerebello-pontine (CP) angle. Prior to the development of modern imaging techniques they presented late with major symptoms, loss of hearing, and usually proceeded to operative removal. The advent of magnetic resonance imaging (MRI) has resulted in the diagnosis of small tumours with minimal symptoms and functional hearing. Operative removal of such small tumours preserves the hearing in only 45–68 per cent.[2,3] The growth rate of AN is extremely variable. MRI has thus led to a clinical dilemma - observation versus early surgical removal.

This study reviews a series of AN treated conservatively.

Materials and method

All patients with AN who were managed conservatively from 1992 to 2003 inclusive were identified from one author's (PF) private prospective records. All patients had been fully informed of their treatment options and the decision for observation was a joint consensus between the patient and the surgeon. Conservative management was considered if the tumour was of a small size or there were unfavourable patient factors (old age, ill-health, mild symptoms, or only one hearing ear). Patients excluded from this study were those with neurofibromatosis type 2, recurrent tumours and 19 patients awaiting follow-up imaging.

Records were examined retrospectively. The patient's gender, age at the initial diagnostic scan, symptoms on presentation, length of follow up (time from the initial scan to the last scan), initial and subsequent PTA (pure tone average-pure tone thresholds in decibels for the frequencies 0.5, 1, 2, and 4 kHz) and speech discrimination scores were recorded. Details of scans were gathered from the radiologist's reports and the examining doctor's (PF) notes. Recorded were the type of scan (MR or

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TABLE I

DETAILS OF THE STUDY POPULATION

Number	n = 100
Gender	
Male	46
Female	54
Median age and range (years)*	61 (31-86)
Median follow up and range (months)†	25.5 (5-150)
Mean PTA (dB) on presentation	47
Side of tumour	
Right	50%
Left	50%
Site of tumour	
IC	52
CPA	48

*Average age 60 years. †Average follow up 33 months. PTA: pure tone average of the frequencies 0.5, 1, 2, and 4 kHz (no PTA in 21 patients; 16 with no tumour growth and five with tumour growth). IC: intracanalicular; CPA: cerebello-pontine angle and intracanalicular (no tumours were solely confined to the CPA).

computed tomography [CT]), tumour type (solid or cystic), side (right or left), site (intracanalicular only, in the CP angle only, or in the CP angle with an intracanalicular component) and size (largest measured diameter and if the tumour was in the CP angle, the intracanalicular component was excluded). Tumour growth, regression or stability from scan to scan was recorded. The growth rate (mm/year) was calculated by size increase divided by the period of growth in months then multiplied by 12. Outcome at the end of the study period in terms of continued observation, surgical or radiotherapy treatment, and the loss of the opportunity for hearing preservation surgery was recorded. The criteria for hearing preservation surgery were set at a PTA <30 dB, speech discrimination score >70 per cent, and tumour size <15 mm in the CP angle.

Data were entered into a software program for statistical analysis (InStat for MacIntosh 1993 GraphPad Software, version 2, July 1993). Fisher's exact test for categorical variables and the Student's t-test for continuous variables were employed with $p < 0.05$ being accepted as significant.

Results

The characteristics of the 100 patients studied are shown in Table I.

The presenting symptoms are shown in Table II with some patients having multiple symptoms.

Tumour growth occurred in 36 patients. Their growth rates are shown in Figure 1.

TABLE II

PRESENTING SYMPTOMS

Symptom	Number
Hearing loss	73
Tinnitus	30
Disequilibrium	25
Fullness in ear	2
Otalgia	2
Vertigo	2
No details	2

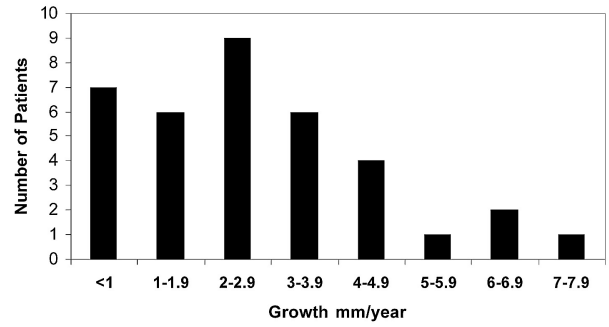

FIG. 1

Growth rate per annum in vestibular schwannomas that showed growth. Total = 36 patients. Average tumour growth 2.68 mm/year.

There were 52 intracanalicular tumours; 27 measuring 0–5 mm, 24 measuring 5–10 mm, and one measuring 11 mm. There were 48 tumours into the CP angle; measuring the CP angle component revealed four tumours at 0–5 mm, 17 at 6–10 mm, 17 at 11–15 mm, eight at 16–22 mm and in two there was no initial measurement. None of the tumours was cystic. Eighty-nine of the 100 patients had MR as the imaging modality as opposed to CT scanning.

Growth versus no growth

The characteristics of those exhibiting growth and no growth are seen in Table III. No significant differences were identified between these two groups. There was no significant difference in initial symptoms between those patients with tumours that grew or did not grow. AN growth rate did not correlate to either the patient's age (r = 0.21, p = 0.24) or the initial tumour size (for intracanalicular tumours r = 0.34, p = 0.19; for CP angle tumours r = 0.12, p = 0.64).

Tumour behaviour

There were six discernable patterns of tumour behaviour (Table IV) over the time span of this study. Most AN displayed no growth (62 per cent). Two showed tumour regression. Nineteen displayed steady growth. There were three further groups of patients where growth alternated with tumour stability. In 10 patients (Growth/Stable) no further growth was seen eight to 58 months (median 31) after the initial scan. In four patients (Stable/Growth) growth occurred eight to 60 months (median 35) after the initial scan, and growth was seen in three patients (Stable/Growth/Stable) after 15, 19 and 19 months of no growth.

In 29 of the 36 patients (80 per cent) in which the tumours grew, this growth was noted in the first year (assuming that seven patients with constant growth who had their second scan after 12 months would have shown growth on a scan within the first year). Of these, 19 continued to grow and 10 subsequently stabilized. Seven of the 36 patients (20 per cent) with tumour growth had a latent period of eight to 60 months before growth occurred.

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TABLE III
TUMOUR GROWTH VERSUS NO TUMOUR GROWTH IN 100 PATIENTS

	Growth		No growth		*P* value
Number	36		64*		
Gender					
Male	16	44%	30	47%	0.84
Female	20	56%	34	53%	
Median age and range (years)	61	31–86	60	31–84	0.88
Median follow up and range (months)	36	5–106	22	5–150	0.04
Mean PTA (dB) on presentation	39		48		0.58
Side of tumour					
Right	17	47%	33	52%	0.83
Left	19	53%	31	48%	
Site of tumour					
IC	17	47%	35	55%	0.53
CPA	19	53%	29	45%	

*Includes two patients with tumour regression. PTA: pure tone average of the frequencies 0.5, 1, 2, and 4 kHz; IC: intracanalicular; CPA: cerebello-pontine angle and intracanalicular.

Surgery

Surgery was performed on 11 patients. The reason for surgery was tumour growth without clinical progression of symptoms (six patients), growth of the AN and worsening symptoms (four patients), and worsening vertigo with no growth of a 5 mm intracanalicular AN. All cases had total tumour resection except a 75-year-old man, with poor cardiac function, who had subtotal removal of a tumour indenting the brainstem. All cases were operated on by the author (PF), 10 via a translabyrinthine approach and one had a middle fossa removal with preservation of the hearing. All patients had preservation of full facial nerve function and only one patient suffered some persistent post-operative disequilibrium. There was no other change in neurological status. There were no patient deaths during the study period and no patients had radiotherapy treatment.

Hearing preservation

There were no initial audiograms available for 21 of the 100 patients. Of the rest, 22 patients (28 per cent of 79) met the criteria for hearing preservation surgery on their initial tests (median age 48 years, range 28–73 years). No follow-up audiometry was available in seven patients, leaving 15 for analysis. Of these, five (33 per cent) patients lost the opportunity for hearing preservation surgery. In four patients hearing deteriorated outside the criteria parameters (no tumour growth in one and growth in three but size remained <15 mm). The remaining patient had good hearing still but the tumour grew to 24 mm after a stable period and was removed via a translabyrinthine approach. The seven patients without follow-up audiograms still had tumours <15 mm on follow up.

Discussion

The advent of MRI and strict investigation criteria (every patient with a unilateral sensorineural hearing loss has an acoustic tumour until proved otherwise) has led to the discovery of many acoustic neuromas that in the past may never have been diagnosed at all (presumably the group in which no growth takes place) or are diagnosed late (the growth group). Physicians are therefore presented with a new dilemma which is how best to manage these small tumours. It is reasonable to presume that large tumours began as small ones and all studies show that some tumours will progress. The aim of studies like ours is to try and identify which tumours will progress and which will not.

Series to date have varied as to the proportion of tumours that grow up to 81 per cent.[4–23] This compares with 36 per cent in this study. Growth patterns can change over time, as was found in the updated follow up of 127 tumours previously examined which found that growth dynamics had changed in 13 tumours, with 10 growing following a period of no growth or regression and three showing various positive growth patterns.[10,14] We identified six patterns of tumour behaviour, as described in other series.[6,10,13,15] Tumour regression was seen in two per cent in this study and has been reported in three to 15 per cent of some series.[5–8,10,14–17] In this series tumour growth averaged 2.68 mm/year. Others have reported similar[17] or slightly lower values.[5,8]

This series found, as in other series, that occurrence of growth did not correlate to presenting symptoms, age, gender, or side of tumour.[5–23] The growth rate did not correlate to the patients' age or the initial tumour size as has been found by others.[5] However positive correlations of these factors to growth were found by one study[11] and a negative correlation between tumour size and growth rate in another.[8] Two other studies, as well as this, found no difference in the mean pure tone audiogram (PTA) at presentation between those patients whose tumours did and did not grow.[13,18] In this study the initial location of the AN, whether intracanalicular

TABLE IV
PATTERNS OF TUMOUR BEHAVIOUR (100 PATIENTS)

Growth time course	Number
No Growth	62
Growth	19
Growth/Stable	10
Stable/Growth	4
Stable /Growth/Stable	3
Regression	2

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or into the CP angle, made no significant difference in the proportion of AN showing growth, which accords with the results of two other studies.[19,20] Most studies have grouped both together.

A promising predictor of growth may be size increase in the first year of follow up. In this study, 80 per cent of those that grew did so within the first year, which corroborates the findings of other studies.[13,17,20,21]

Hearing deterioration without AN growth occurred in one of our patients and has been described by others.[13] Some studies have examined the loss of hearing versus the growth of the tumour over time with growing tumours exhibiting a tendency toward greater hearing loss than those without growth,[13] and a significant correlation between tumours growing more than 10 mm and a change in the PTA.[15]

- **This is a retrospective review of 100 patients with vestibular schwannomas managed by observation and regular scanning**
- **The growth pattern of such tumours was variable but the majority exhibited no growth. The authors suggest that size increase in the first year of follow up may predict future growth**
- **The rate of growth in those tumours that did show growth was variable and there appeared to be no predictors of tumour growth**

This study, as do others,[13] shows that there is no change in the neurological status of patients who were at first not treated and then come to surgery, with the possible exception of five of our patients who lost serviceable hearing. Hearing preservation criteria in acoustic neuroma surgery is a contentious issue. Not only do the eligibility criteria for hearing preservation surgery vary widely but claims of hearing preservation in the past have often not been substantiated with audiometric data. Further, inter-aural differences after surgery are rarely referred to. Hearing may be preserved in the immediate post-operative period but subsequently deteriorate. A study found hearing was preserved in nine of 20 patients undergoing retrosigmoid tumour removal. Two patients lost hearing within a few weeks of surgery while the other seven had stable hearing one to two years later.[24] Radiotherapy too, to the best of our knowledge, has not reported long-term hearing preservation rates. If operating on patients with good hearing only preserves that hearing in 50 per cent and if all patients meeting the criteria for hearing preservation surgery were operated on, then this would have resulted in more sustaining a hearing loss over the follow-up interval in this study. da Cruz[2] and Lassaletta[26] show how changing the criteria for surgery selection and for reporting the post-operative hearing result can change the hearing preservation rates. It is the opinion of these authors that stricter criteria for useful hearing, comparisons with the other ear and a long-term follow up, will show that a very few patients who come to treatment will have useful hearing.

In the end, the aim of acoustic tumour sugery is to remove the tumour without brainstem or other neurological sequelae and to preserve the facial nerve. If hearing preservation can be attained, all to the good but it will always remain the least important of the aims in acoustic tumour surgery and should not be allowed to distract the surgeon from the pursuit of the other goals.

Limitations of this study include the fact that this was a retrospective study examining a select group of those presenting with acoustic neuroma. The imaging included the use of CT scan as well as MRI. Determination of scan-to-scan growth was done by direct comparison of the current scan to previous scans in an un-blinded fashion. A study found that computer analysis of tumour size and growth rate in the coronal and axial planes was statistically the same as the radiologist's description of the tumour size and growth.[15] The growth pattern was determined by scan-to-scan growth. This will give a snapshot of growth patterns only over the course of the study, and as noted above, the future growth pattern can change. Also varying the length of time between scans may alter the perceived pattern of tumour growth. For example, a tumour that has a growth spurt then regresses would be seen as stable if scanned before the spurt and after the regression. The follow up is short for a benign slow-growing tumour, so an early second scan may not reveal the growth that a later scan does. We aim for scans at yearly intervals, as rapid growth spurts can occur, and then may increase the length of time between scans if the situation is stable. The group with no growth had overall shorter follow up than the group that displayed growth. It may be that further follow up will reveal more tumours growing over time.

The longest follow up of a patient in this series was 12.5 years, and in other series follow ups of patients to 16 and 17 years have been recorded.[4,15] Given that all studies have shown that growth rates can vary, a phenomenon confirmed in this study, the natural history of acoustic tumour will only be determined by a study that is of some 20 or 30 years' duration. The current study continues. This study and others have shown that provided follow up is diligent, timely surgical removal can be carried out, when indicated, without a worsening of the neurological status.

Conclusions

(1) Conservative treatment of acoustic neuroma by observation is yet to be clearly defined.
(2) Acoustic neuroma behaviour and pattern of growth is variable and there are no predictors of tumour growth at the present time.
(3) Selected patients (small tumours, old age, ill-health, minimal symptoms, only or better hearing ear, and refusal of surgery) can be observed with minimal morbidity providing that there is serial imaging and clinical follow up to detect growth of the tumour and symptomatic progression.

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(4) If the issue of hearing preservation surgery is laid aside, then delaying surgery until required by progression of symptoms or increase in tumour size does not result in more morbidity for the patient. Some may lose the opportunity for hearing preservation surgery but operating on all would result in more sustaining a loss of hearing in the first few years after diagnosis.

(5) Longer follow up of patients is required to further elucidate the natural history of acoustic neuroma.

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