

Leading article

Management of tumour spillage during parotid surgery for pleomorphic adenoma

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Accepted 18 May 2013

Available online 27 June 2013

Abstract

Over the decades parotid surgery for benign tumours has developed into a reproducible, conservative operation with low morbidity. Despite the advances tumour spillage can still occur, and its management remains controversial. Since no universal consensus exists the aim of this article is to review the approach to tumour spillage and derive a protocol for its management based on existing evidence.

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Keywords: Tumour spillage; Parotid surgery; Pleomorphic adenoma

Introduction

The average annual age-adjusted incidence of salivary gland tumours is around 5/100,000 in the United Kingdom.¹ Around 80% of tumours in the parotid gland are benign, the most common of which are salivary adenomas. Although the excision of parotid lesions was attributed to Bertrandi in the early part of the 19th century, the intimate relation between the facial nerve and parotid tumours was not understood until the 1850s, with Codreanu, a Romanian native performing the first total parotidectomy with preservation of the facial nerve in 1892. The modern era of parotid surgery began in the United States with the work of Beahrs and Adson who described the relevant anatomy and surgical techniques in 1958. They stressed which landmarks to use to avoid injuring

the nerve and advocated complete removal of the superficial portion of the gland for non-invasive lesions.

The clinical problem

Pleomorphic adenomas exhibit wide cytomorphological and architectural diversity. Tumours consist of epithelial, myoepithelial, and stromal (mesenchymal) components that may vary quantitatively from one tumour to the other. The epithelial component consists of epithelial and myoepithelial cells with divergent growth patterns which include trabecular, tubular, solid, cystic, and papillary architecture. The stromal component, which is a product of modified myoepithelial cells, may appear mucoid, myxoid, hyaline, chondroid, myxochondroid, or even osseous. Tumours with high cystic or mucoid content are particularly friable.

Adenomas are often irregularly shaped with a bosselated surface, and they display variable cystic changes. Typically,

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surrounding the tumour is a fibrous capsule of inconstant thickness, which may be focally deficient particularly in more mucoid tumours. Microscopic satellite tumour nodules, pseudopodia, and focal penetration may be seen beyond the capsule.² Since most patients with pleomorphic adenomas have operations, microscopic spillage, particularly of friable tumours, can occur. If such spillage is recognised at the time of operation it raises several questions: whether the spillage increases the risk of subsequent recurrence; whether the intraoperative plan should be changed to include additional techniques; whether any postoperative adjuvant treatment should be considered; and whether recurrent pleomorphic adenomas can be salvaged.

Does intrasurgical rupture of pleomorphic salivary adenomas result in an increased rate of tumour recurrence?

The rate of tumour recurrence after operation varies considerably in different clinical settings and depends on the surgical technique used. Although it is traditionally thought that intraoperative rupture of the capsule is associated with increased recurrence, the impact of tumour spillage on the subsequent clinical course has been questioned. A number of follow-up studies have been undertaken to ascertain whether intraoperative rupture, tumour spillage, or any histopathological feature might have an impact on recurrence.

Witt compared matched pairs of 60 pathological specimens of parotid salivary adenoma (20 each after total parotidectomy, partial superficial parotidectomy, and extracapsular dissection) for capsular exposure and the degree of cellularity of the tumour.³ Statistical analysis of the respective rates of capsular rupture and recurrence showed that focal capsular exposure occurred in virtually all operations regardless of type, and dissection in the plane of the facial nerve led to a positive margin in 25% of cases. Capsular rupture did not result in a significantly high rate of recurrence and did not vary among the operations. Hypocellular tumours did not have a higher incidence of capsular rupture or recurrence.

In a retrospective review of cases with intraoperative tumour spill over a 19-year period (mean follow-up 7.4 years), Buchman et al. also showed that spillage at the time of operation was not predictive of local recurrence.⁴ In a clinical observation study, Natvig and Sjøberg reviewed the medical records of 346 patients who had operations in the Department of Otolaryngology in the National Hospital, Norway between 1965 and 1981 with special reference to the relation between the surgical margins and the tumour capsule.⁵ The average time of observation was 18 years (range 11–25) and no patients had postoperative irradiation. A total of 6/238 (2.5%) patients had recurrence between 7 and 18 years postoperatively (mean 11.8). Rupture of the capsule with microscopic spillage of tumour cells occurred in 26 patients (11%), 2 of which (8%) developed recurrent tumours. Surgical dissection close to the capsule took place in 87 cases (36%)

and there was one recurrence (1%). In the remaining 121 patients (51%) dissections were carried out without visualisation of the tumour capsule; 3 (2.5%) of them developed recurrent tumour. The 8% recurrence rate after rupture of the capsule was not statistically different from the 2% for the other patients. The authors also examined recurrence according to the microscopic status of surgical margins. In 10% of patients there were microscopic positive margins, tumour cells had penetrated the capsule in 20%, and in 70% the margins were negative. There was no difference in recurrence between patients in these histological groups.

In a similar retrospective study at the Huddinge University Hospital, Henriksson et al. reviewed the medical records of 255 patients operated on for parotid salivary adenoma over a 19-year period.⁶ Of the 28 patients who had macroscopic rupture of the capsule during operation, 2 (7.1%) had recurrence at a later stage. This was not statistically higher than the 4.1% recurrence rate for patients in whom no rupture was seen. Pseudopodia and microscopic finger-like formations of tumour tissue extending beyond the main tumour were the most significant risk factors for local recurrence (5/9 tumours that subsequently recurred) although statistical analysis did not show whether this finding alone was predictive of recurrence above those without pseudopodia.

Should additional intraoperative procedures be done after macroscopic tumour spillage?

There is no evidence on which to base recommendations about the advantages or otherwise of the use of additional intraoperative techniques in the event of tumour spillage. Some authorities recommend copious irrigation to minimise recurrence while others suggest that it spreads viable cells, which can increase the likelihood of potential recurrence being multifocal. We also know of no reported evidence to support the suggestion that one potential irrigating solution has advantages – for example, isotonic compared with hypertonic solutions.

Should postoperative adjuvant procedures be advised after tumour spillage?

Although not commonly used for benign disease, radiotherapy has been suggested as a way of reducing recurrence after incomplete removal of parotid salivary adenoma or intraoperative tumour spillage. Although there are early reports of radium needle implants at the time of operation most authors describe beam-directed external radiotherapy using a 3-field technique or wedge pair. Barton et al. published their findings of a retrospective analysis of 187 patients who had adjuvant radiotherapy after tumour spillage at the Christie Hospital between 1951 and 1984.⁷ The median age was 46 years and nearly half of the patients (87/187) were aged between 40 and 60. A total of 115 patients had radiotherapy immediately

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