# Correspondence
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## Tuberculosis of the parotid gland

Although tuberculosis is common in India, tuberculous infection of the parotid gland is uncommon.[1] Kant and associates[2] in 1977 reported 35 cases, and Yaniv and Avedillo[3] added 2 more cases in 1985. We recently had a case of parotid tuberculosis simulating a parotid neoplasm.

### Case report

A 9-year-old boy presented with gradually progressive swelling over 1 year in the left parotid region. Physical examination revealed no other abnormality. Examination of the parotid area revealed a firm, nontender swelling that lifted up the ear lobe. The mass was fixed to the underlying structures, but the facial nerve was spared. He had a family history of tuberculosis. Hematologic and biochemical investigations revealed only a raised erythrocyte sedimentation rate (56 mm/h). Chest x-ray films appeared normal. The Mantoux test gave borderline results. The preoperative diagnosis was a parotid tumour. The boy underwent a superficial parotidectomy. There was evidence of calcification of the mass with central caseation. Histologic examination of the operative specimen disclosed tuberculous parotitis.

### Discussion

Tuberculosis of the salivary glands is rare. Granulomatous lesions may be present as localized nodules and can make the diagnosis difficult. The source of the infection in parotid tuberculosis is controversial. Van Stubenrauch[4] postulated extension of infection along Stenson's duct from the oropharynx and Bockhorn[5] postulated a vascular mode of spread from any primary focus in the body or through wounded oral mucosa. According to Berman and Fein[6] spread by lymphatic vessels, particularly from infected tonsils and the external auditory canal, plays an important role. Carmody[7] formulated a canalicular mode of spread from infected molar teeth. With respect to the types of mycobacteria involved, bovine strains have been considered an important etiologic agent, with or without pulmonary involvement.[3,6] The causative role of atypical mycobacteria in infection is remote. If the diagnosis is made preoperatively, parotid tuberculosis can be treated with antitubercular drugs only. Otherwise parotidectomy must be performed, followed by a full course of antitubercular therapy. With the increasing incidence of tuberculosis along with immunodeficiency syndromes, this association may be an important one to bear in mind when cases of parotid tuberculosis are encountered.

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## Renal autotransplantation

In response to Taguchi's editorial (*Can J Surg* 1996; 39: 93), concerning the paper on renal autotransplantation (*Can J Surg* 1996; 39: 121–125), his scepticism is understandable and mirrors my own when first asked to consider autotransplantation for loin pain–hematuria syndrome. Experience with renal autotransplantation is limited; up to 1993, only 40 patients had reportedly been treated with this procedure for intractable pain. Pain is, of course, a subjective complaint, but the patients considered for surgery had failed other options and were disabled by their pain.

Until I was presented with my first case in 1990, I, too, had never knowingly seen a case of loin pain–hematuria syndrome nor had such a case been discussed at university rounds. However, loin pain–hematuria syndrome obviously exists, although it represents only a very small fraction in individuals presenting with flank pain. Autotransplantation is used as a last resort and only after symptoms have been present for some years. Approximately 30% of patients in one series[1] had spontaneous resolution of their