# A preliminary study of the structure and function of enlarged parotid glands in chronic relapsing pancreatitis by sialography and biopsy methods

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EDITORIAL SYNOPSIS Enlargement of the parotid glands is associated with calcific pancreatitis in one area of India. Surprisingly, the enlarged parotids showed evidence of hypertrophy and hypersecretion.

In the course of gathering material for a thesis, it became evident that bilateral parotid enlargement was associated with chronic relapsing pancreatitis in a large percentage of cases. Information on this combination was meagre in the literature available to us, although Zuidema (1959), Shaper (1960), Wegmann and Sollberger (1960), and Pitchumoni (1965) have noted the association. There is, however, no reference to the changes in structure and function that the parotid glands undergo, except that Wegmann and Sollberger (1960) noted a high amylase content.

A study of 23 patients with enlarged parotid glands and chronic relapsing pancreatitis was undertaken, and the results are the substance of this report. The patients were from families whose other members did not show either parotid enlargement or chronic relapsing pancreatitis. Nearly all the patients examined had high fasting blood sugar levels and a near normal total protein content although the albumin:globulin ratio was not normal (Alappatt, 1965). The study was restricted to the structure and function of the parotid gland by biopsy and sialography and the quantity and amylase content of the saliva.

## MATERIAL

Diabetic patients attending the Medical College Hospital, Trivandrum, who had chronic relapsing pancreatitis and showed coincidental parotid enlargement were selected. They were first screened by routine clinical examination to exclude known local and general causes of parotid enlargement and pancreatitis was confirmed by blood sugar estimations and plain radiographs of the abdomen. All the 23 had high fasting blood sugar levels and pancreatic calculi. Those suitable were selected and their

blood estimated for protein and haemoglobin content. Patients with general ill-health, chronic skin infection, and others in whom biopsy and sialography might have proved risky were excluded.

For the purposes of this study a parotid gland was considered enlarged when the normal concavity between the ramus of the mandible and the anterior margin of the sternomastoid was obliterated and the gland was palpable (Wolfe, Summerskill, and Davidson, 1957).

## METHODS OF STUDY

All the patients selected for this study were treated with insulin to bring the diabetes under control over a period of one week before the tests were undertaken. Function of the parotid gland was assessed in respect of secretory activity and amylase content only. The parotid duct was catheterized and the amount of saliva secreted to a standard stimulus (chewing chocolate for five minutes) was measured and its amylase content estimated by the achromic end-point method (Levinson and MacFate, 1956).

Ten normal healthy blood donors were studied and an average taken as the standard for comparison in this study.

Bilateral sialography was carried out in all patients, according to the standard method described by Pyrah and Allison (1931). As a control a sialogram was taken of a normal healthy adult.

Material for biopsy in 14 cases was obtained by open surgery under aseptic conditions and there were no complications. A section of a parotid gland from post-mortem material was taken for comparison.

# OBSERVATIONS

In general it was observed that the parotid glands showed only increased secretory activity and no change in amylase content, which ranged from 20 to 80 units per millilitre, and in the controls from 10 to 100 units per millilitre. But the rate of flow of saliva after stimulation was found to be significantly high. The mean secretion from one parotid gland was 3 ml. each five minutes with a standard deviation of 0.75, but in the controls it was only 0.85 ml. each five minutes with a standard deviation of 0.15.

Sialography demonstrated no uniform deviation. No parotid gland showed calculi. The appearances were normal in seven. Sixteen cases presented the so-called 'leafless tree' appearance (Du Plessis, 1956), the main duct and the tributaries being normal and well defined. The finer ducts appeared to be more widely separated when compared with the normal parotid gland in which they were found closely packed (Fig. 6). The finest ducts were not visualized. Six cases showed a varying degree of dilatation of the main duct in addition to the leafless tree appearance, a dilatation which might be a reflection of hyperactivity.

Biopsy material in general showed signs of hyperfunction. The histological changes noted were that the acini appeared larger than normal and the individual cells appeared swollen. This change was confirmed by comparing the number of nuclei in the normal and the hypertrophied gland per high-power field. In the hypertrophied gland there were fewer nuclei per field (Fig. 2). A varying degree of roundcell infiltration around the intra- and interlobular ducts was found in 13 cases (Fig. 4), possibly due to low-grade infection. (Culture of the saliva was not, however, done in any case.) The ducts appeared normal, as in only three cases was there an occasional duct showing intraluminal epithelial proliferation. The interlobular fibrous tissue was not increased. One case alone showed marked fibrosis with atrophy of the acini. This case had marked round cell infiltration and considerable dilatation of the main duct (Fig. 5).

### DISCUSSION

The object of this study was restricted to finding out the nature of change that brings about parotid gland enlargement in cases of chronic relapsing pancreatitis, which is prevalent in Kerala (Geevarghese, Pillai, Joseph, and Pitchumoni, 1962). We have confirmed Pitchumoni's observation that the two diseases are associated and attribute the parotid enlargement to functional hypertrophy. We have as yet made no contribution to the reason for this association.

#### **SUMMARY**

A study of enlargement of the parotid glands in (x 475). Note the large acini.



FIG. 1. Enlarged parotid glands in a patient.

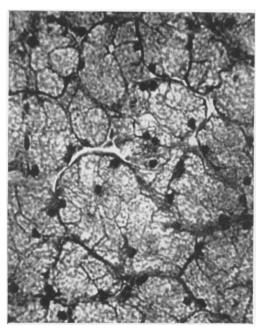


FIG. 2. Photomicrograph of enlarged parotid gland  $(\times 475)$ . Note the large acini.

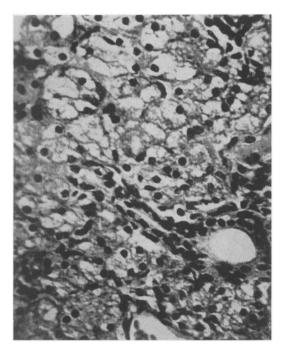


FIG. 3. Photomicrograph of normal parotid gland (  $\times$  475) showing the normal acini for comparison.

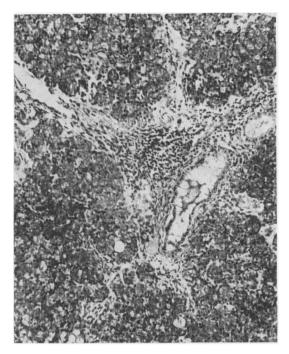


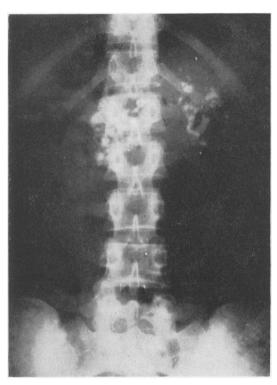
FIG. 4. Photomicrograph of an enlarged parotid gland  $(\times 110)$  showing the periductal round cell infiltration.



Fig. 5. Fig. 5.

- FIG. 5. Sialogram showing dilatation of parotid duct and 'leafless tree' appearance.
- FIG. 6. Normal sialogram for comparison.





Plain film of the abdomen showing pancreatic calculi.

23 patients with chronic relapsing pancreatitis was undertaken to ascertain the nature of the parotid enlargement, which was found to be in the nature of a functional hypertrophy.

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