

cirrhosis results in asymptomatic enlargement of the parotid glands in 30–80% of these patients (Regezi, Sciubba, and Jordan 2003). In such cases, parotid enlargement has been attributed to protein deficiency. In diabetes mellitus, the mechanism of acinar hypertrophy associated with this condition is unknown. Due to the numerous causes of sialosis, as well as a large number of diagnoses that can clinically resemble sialosis, the patient's history is paramount in such cases so as to properly initiate the diagnostic process. In addition, the treatment for these disorders differs significantly.

CLINICAL MANIFESTATIONS OF SIALOSIS

Sialosis is characterized by chronic, afebrile salivary enlargement. The enlargement is described by patients as slowly evolving and recurrent. A thorough history will most frequently divulge symptoms associated with comorbid disease such as diabetes mellitus, achalasia, alcoholism, or others.

DIAGNOSIS OF SIALOSIS WITH SALIVARY GLAND BIOPSY

The role of salivary gland biopsy in a patient suspected of having sialosis is to rule out Sjogren's

syndrome, sarcoidosis, and lymphoma. Sialosis is a disease limited to the major salivary glands such that an incisional biopsy of parotid enlargement is indicated, rather than an incisional biopsy of the lip as might be considered in Sjogren's syndrome or sarcoidosis. As such, a minor salivary gland biopsy is of no value in making a diagnosis of sialosis. While histopathologic confirmation of this process is valuable, it is certainly possible to make a clinical diagnosis of sialosis based on historical findings (Mandel, Vakkas, and Saqi 2005). In addition, once a histopathologic diagnosis of sialosis has been established, the underlying cause of this disorder must be ascertained, if not already known preoperatively. Prompt treatment of the underlying disease process must then occur.

Histopathology of Sialosis

The parotid swelling of sialosis is due to acinar enlargement (Figure 6.16). The diameter of the acinar cell tends to increase by two to three times that of normal. The nuclei tend to be basally situated, and the cytoplasm tends to be packed with granules. There is no correlation between the specific clinical type of sialosis and the histologic appearance. Inflammatory cells tend to be absent. The long-standing nature of the underlying disease may ultimately lead to acinar atrophy and replacement with fat (Werning 1991).

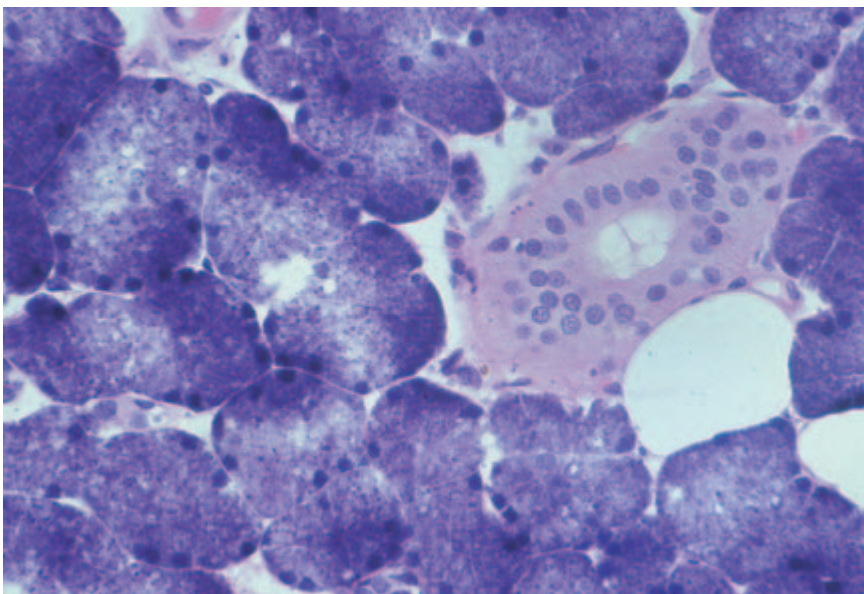


Figure 6.16. The histopathology of the incisional parotid biopsy performed on the patient in Figure 6.14. Acinar hypertrophy is noted. The physical, radiographic, and histologic information confirms a diagnosis of achalasia. He was treated accordingly.