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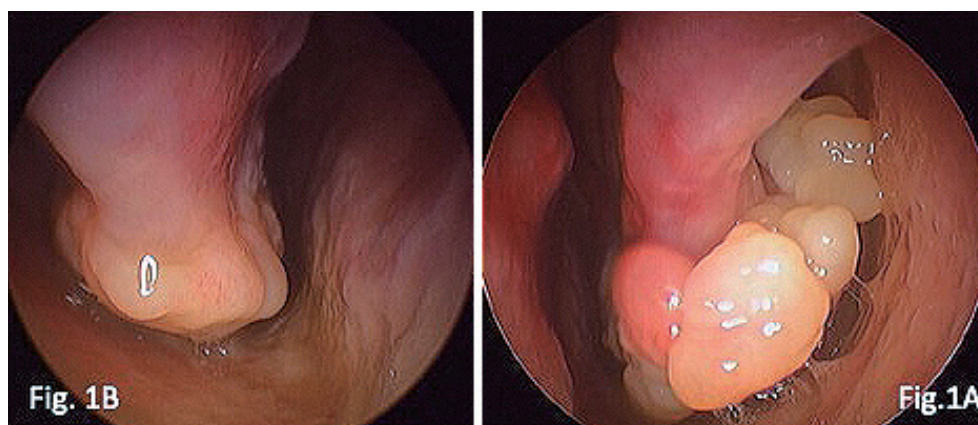


Figure 1 Both endonasal findings

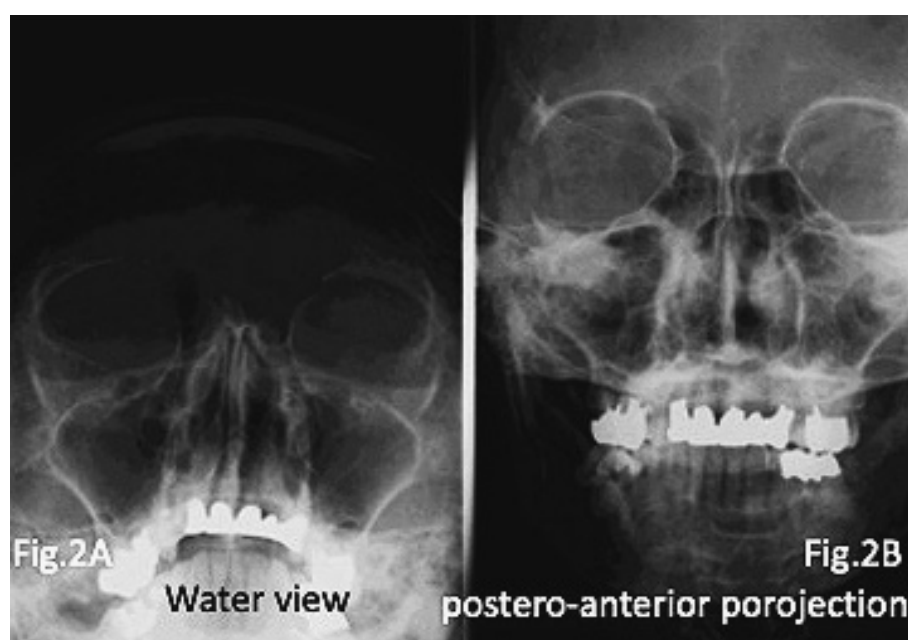


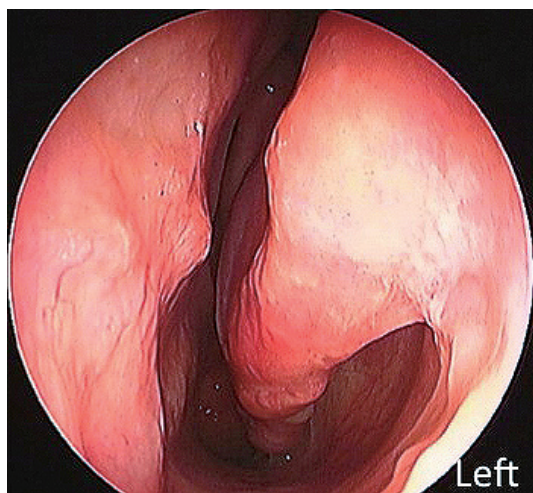
Figure 2 Paranasal X ray

surgery found no clear evidence of recurrence of the polyps (Fig. 3). Histological studies showed, in the lamina propria, hyperplasia of blood vessels with thickened walls, interstitial edema, lymphocytes and plasma cells. There were no findings of neoplasms. Although the histology was similar to that of polyps, there was little infiltration by inflammatory cells, including eosinophils (Fig. 4).

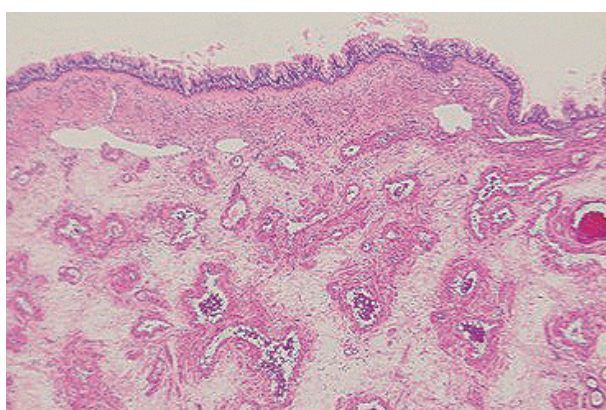
### Discussion

Reports of multiple pedunculated polyps in the inferior turbinate are very rare<sup>1)</sup>. Histologically, these polyps are considered to be fibrous lesions covered by

pseudostratified cylindrical epithelium surrounding a hemangioma, with little infiltration of inflammatory cells. They are similar to the mucosal epithelium of ordinary nasal polyps, but they differ by showing less infiltration by inflammatory cells such as eosinophils and lymphocytes and having marked proliferation of blood vessels in the submucosa. The histological findings in the patient we reported here are consistent with those reported by Nishijima et al<sup>1)</sup>, and the polyps of our case can thus be diagnosed as fibroepithelial polyps. Although the pathogenesis of fibroepithelial polyps remains unresolved, chronic inflammatory processes may play a key role. Several chronic inflammatory etiologic



**Figure 3** endoscopic findings after one year



**Figure 4** histopathological findings

factors, such as chronic smoke inhalation, chronic inflammation in patients with asthma and chronic obstructive pulmonary disease (COPD), and repeated airway infections may be potential causes<sup>2</sup>. However, the nasal cavity is habitually exposed to such external stimuli as infections, smoke, etc., and chronic inflammation is always present, making it difficult to believe that it would act as a specific trigger.

Although the pathogenesis of fibroepithelial polyps remains unresolved, chronic inflammatory processes may play a key role. Several chronic inflammatory etiologic factors, such as chronic smoke inhalation, chronic inflammation in patients with asthma and chronic obstructive pulmonary disease (COPD), repeated airway infections may be potential causes<sup>2</sup>. On the other hand, as the nasal cavity is often stimulated by infection and smoking, chronic inflammatory always exist and is hard to consider it as specific precipitants.

The main symptoms are nasal occlusion and epistaxis, and patients commonly go to an ENT clinic due to the nosebleeds. Nasoendoscopic findings indicate multiple, pedunculated, benign tumors, which differ from the inferior turbinate edematous mucosa that is associated with allergic rhinitis. There are scattered references to bronchial fibroepithelial polyps in the published literature, and the histological possibility of neoplasms<sup>3</sup> and the risk of recurrence<sup>4</sup> have been pointed out. In our present case, there were no findings suggestive of a neoplasm, and no clear evidence of recurrence was seen at 6 months following the surgery. Nevertheless, long-term observation of this patient will be necessary.

Nasal polyps can be treated conservatively or surgically, depending on their size and the amount of bleeding. Small polyps were reported to resolve in response to drug therapy using corticosteroids and antibiotics<sup>5</sup>. However, large polyps and lesions that do not respond to drug therapy are treated by excision by endoscopic surgery<sup>6</sup>, with residual lesions treated by argon plasma coagulation or laser therapy<sup>7</sup>. Patients such as our present case, with polyps in the inferior turbinate, can probably be best treated by biopsy and excision of the lesions, with postoperative administration of corticosteroids and antibiotics.

#### Conflict of interest statement

We declare that we have no conflicts of interest to disclose.

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