Case N°2: Pyogenic granuloma in Barrett’s oesophagus

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Clinical history
A 62-year-old man underwent an upper gastrointestinal endoscopy for follow-up of Barrett’s oesophagus.
One year before diagnosis was made of esophagitis with short Barrett’s oesophagus with intestinal metaplasia, negative for intra-epithelial neoplasia. He was treated with proton pump inhibitors.
The patient was on life-long anticoagulation therapy for recurrent episodes of deep vein thrombosis of unknown cause.
Upper endoscopy showed a semipedunculated polypoid lesion with white coating at 25 cm from the incisor teeth. The polypoid lesion had a diameter of about 1 cm.
Endoscopic ultrasound (EUS) revealed no signs of invasion and no local lymphadenopathy.
Endoscopic mucosal resection of the mass was performed. There was no bleeding.
Follow-up upper endoscopy 2 years later revealed no new lesions suggestive of recurrence.

Description of the slide
On the slide that was submitted for the seminar, including the entire polyp removed by endoscopic snare polypectomy (Figure 1), we observe a mass composed of aggregates of angiomatous structures of various sizes with endothelial cell swelling, arranged in a vague lobular fashion, accompanied by fibrous stroma and located in the mucosal layer, covered by normal squamous epithelium (Figure 2, 3, 4). The epithelium was intact, in contrast to a previous biopsy from the lesion that showed focal ulceration. The previous biopsy also showed rather oedematous stroma.
containing variable amounts of inflammatory cells, including neutrophils (Figure 5). Immunohistochemistry showed positivity for CD31 (Figure 6). Immunohistochemistry for D2-40 and HHV-8 was negative (Figure 7). Focal intestinal metaplasia in previous biopsies was consistent with diagnosis of Barrett’s oesophagus (Figure 8).

Figure 1: Endoscopic photograph of the exophytic tumour located at 25 cm from the incisor teeth. We see a semipedunculated polypoid lesion with white coating. The polypoid lesion has a diameter of about 1 cm.

Figure 2: Low power view of the resected fragments (HE).
Figure 3A and 3B: A mass composed of aggregates of angiomatous structures of various sizes arranged in a vague lobular fashion, protruding into the lumen (HE).

Figure 4: High power view showing vascular spacers lined by swollen endothelial cells. Some denser regions with less obvious vascular lumina were also present (HE).
Figure 5: Focal ulceration. In this region the stroma contains variable amounts of inflammatory cells, including neutrophils (HE).

Figure 6: Lining cells displayed positivity for CD31, but not for D2-40 (IHC)
Figure 7: No positivity was observed for HHV8 (IHC).

Figure 8: Focal intestinal metaplasia (HE).

**Diagnosis**
Pyogenic granuloma in Barrett’s oesophagus.
Pyogenic granuloma (also called granulation tissue-type haemangioma) is a polypoid form of lobular capillary haemangioma mostly occurring on the skin, particularly the lips, face and fingers, and also on mucosal surfaces of the oral cavity, especially the tongue and gingiva. It appears as a rapidly growing elevated dark red lesion that may or may not be ulcerated. The size ranges from several millimetres to a few centimetres. Microscopically, it consists of masses of proliferating endothelial cells, with or without discernible lumina, separated by an oedematous stroma containing inflammatory cells. Characteristically, the covering epithelium almost meets at the base of the lesion, forming a collarette. High cellularity and abundant mitotic activity may be present. The lesion occurs at all ages, and sexes are affected equally. Its precise aetiology is unknown and controversial, some considering them neoplasms and other a reactive process. Pyogenic granuloma bears a striking resemblance to granulation tissue with often extensive superficial secondary inflammatory changes (due to ulceration) and was formerly believed to be infectious in origin (septic granuloma, botryomycoma). It is often consequence to a minor trauma. It is also known to be associated with pregnancy. Although benign in nature, the tumour is known to recur frequently after resection (4, 8, 11, 15).

The occurrence of pyogenic granuloma in the gastrointestinal tract in other parts than the oral cavity is extremely rare and only very few cases of gastrointestinal pyogenic granuloma have been reported, mainly in the colon (one patient with multiple lesions), and rarely in the oesophagus and small intestine (1, 5, 6, 7, 9, 10, 12, 13, 16). More in particular, only 6 patients with pyogenic granuloma in the oesophagus have been reported in the English literature (2, 3, 7, 10, 12, 14). The patients present with symptoms of dysphagia, retrosternal pain, weight loss and/or bleeding. On endoscopy the lesion usually occurs as a pedunculated polyp, often with white coating, frequently with ulceration. The lesion is adequately treated by snare polypectomy (as in this case and 2, 3, 7, 10, 12) or by laser-photocoagulation (14). In the gastrointestinal tract recurrences of pyogenic granulomas after local resection have not been described. Moreover, no case of malignant transformation has been reported.

The lesion needs to be distinguished from an angiomatous form of Kaposi sarcoma, an important differential diagnosis (4, 11, 15). Kaposi sarcoma is typically encountered in patients with AIDS. Grossly, Kaposi sarcoma has a darker bluish
colour and is usually covered by a moistly glistening intact mucosa. Microscopically Kaposi sarcoma in not well circumscribed and contains, in addition, at least focal cellular zones of spindled cells, which form the classic slit-like vascular spaces. Immunohistochemistry for human herpesvirus 8 (HHV8) is positive in Kaposi sarcoma and negative in pyogenic granuloma. Included in the differential diagnosis is also bacillary angiomatosis, a vascular proliferation caused by Rochalimaea henselae (a rickettsial organism) (4, 11, 15). This condition occurs almost exclusively in patients with AIDS or other immunosuppressive conditions. The architecture is very similar to pyogenic granuloma. Important is the recognition of granular basophilic or amphophilic material in relation to the inflammatory cells, which consists of short bacilli as shown when stained with Warthin-Starry or Giemsa. Pyogenic granuloma should also be differentiated from well differentiated angiosarcoma, which displays poor circumscription, cellular atypia and dissection in collagen bundles (4, 11, 15).

It remains unclear whether the occurrence of Barrett's oesophagus and pyogenic granuloma observed in our patient are related. In the 6 oesophageal pyogenic granulomas described previously, this combination of Barrett's oesophagus and pyogenic granuloma was found in 2 patients (3, 7). In one of these cases the patient had been treated with repeated dilations because of stricture formation caused by severe esophagitis and the lesion thus could have been developed as a consequence of trauma. In the other reported case and in our patient there was no history of previous dilatations and the polyps did not arise from columnar epithelium. Our patient was diagnosed with Barrett's oesophagus, known to develop on the basis of reflux esophagitis, damaging the epithelium and mucosa. This is however not consistent with the fact that pyogenic granuloma is only rarely observed in the oesophagus, whereas reflux esophagitis is a very common condition (7).

In conclusion, although extremely rare, pyogenic granuloma needs to be considered in the differential diagnosis of a tumoral mass developing in the oesophagus and it is important to emphasize that pyogenic granuloma arising in Barrett’s mucosa can at endoscopy simulate a malignant process (this case and 3, 8). Biopsy together with endoscopic ultrasound (EUS) allows making the correct diagnosis. It is a benign lesion that can be adequately treated by endoscopic resection.
References


