unusual locations of heterotopic pancreas, such as the lungs, gallbladder, spleen, and papilla of Vater. Of lesions found in the stomach 85–99% are located in the antrum and are within 5–6 cm from the pylorus [1–3].

Most cases of heterotopic pancreas are asymptomatic, but non-specific gastrointestinal symptoms have been described in 30–40% of cases [3]. Most patients complain of epigastric pain, nausea, vomiting, and gastrointestinal bleeding. Complications are similar to those that occur in the pancreas itself and include acute pancreatitis, pancreatic cancer, insulinomas, gastrinomas and cystic degeneration [4,5].

Asymptomatic cases of heterotopic pancreas are seldom recognized at a preoperative stage and are usually discovered during surgery. Preoperative diagnostic studies include upper gastrointestinal series, gastro-duodenoscopy, computerized tomography and endoscopic ultrasound. Gastro-duodenoscopy is an indispensable tool for the investigation of patients with upper gastrointestinal symptoms, however it is generally difficult to produce an accurate endoscopic diagnosis of submucosal tumors such as heterotopic pancreas because biopsy specimens often fail to include the tumor tissue beneath the normal mucosa. Despite its characteristic features such as central umbilication in the tumor, this condition is difficult to diagnose endoscopically, because in tumors of less than 1.5 cm that umbilication is often absent. Endoscopic ultrasound is helpful for detecting small submucosal tumors (<2 cm), but it is not specific and cannot exclude other pathologies, such as cardinoid, fibroma, eosinophilic granuloma or leiomyoma [5].

Surgical exploration is required for a definitive diagnosis and to exclude neoplastic lesion for symptomatic patients [2]. When the lesion appears benign, local excision with confirmatory frozen section is the treatment of choice [2]. However, in cases of malignancy or when the diagnosis is uncertain, more formal gastric resection is mandatory. Similarly, in cases of heterotopic pancreas found incidentally, it is advisable to resect intraoperatively to avoid late complications and a second operation. In cases of a definitive and certain diagnosis, asymptomatic patients should remain under observation since the risk of malignant changes is no greater in heterotopic pancreas than in the pancreas itself [2,3,5].

References

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**Alendronate-Induced Lichen Planus**

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**Key words:** alendronate, lichen planus, drug eruption

Alendronate is an oral amino derivative of the bisphosphonates, used for the treatment of bone diseases characterized by increased osteoclastic resorption. It is widely used for the prevention and treatment of osteoporosis, as well as for reducing pain and hypercalcemia in malignant neoplasms of the bone, Paget's disease and several other conditions. Gastrointestinal symptoms are the most frequent side effects observed during treatment with alendronate. Reports in the literature of cutaneous side effects are scarce. We describe a patient with osteoporosis who developed hypertrophic lichen planus during treatment with alendronate.

**Patient Description**

A 54 year old woman attended our outpatient clinic due to an itching rash on her trunk and extremities. The rash appeared 5 months prior to her consultation, and 2 months after the beginning of treatment with alendronate (Fosalan M.S.D) for osteoporosis. The patient was otherwise healthy and was not taking any other medications or food supplements.

On dermatologic examination, livid flat papules as well hypertrophic prurigo-like papules were seen on her sacrum, flexural aspect of the arms and on the lower extremities. The oral and genital mucosa were not affected. The clinical features were compatible with hypertrophic lichen planus. Laboratory investigations including complete blood count, liver and kidney...
function tests, antinuclear antibodies, complement 3 and 4, aspartate aminotransferase, serology for hepatitis A, B and C, viral serology for Epstein-Barr virus and cytomegalovirus and serology for syphilis (VDRL) were all normal or negative. Patch testing with the standard series (TRUE Test) was negative. The histopathologic findings from a lesional biopsy demonstrated hyperkeratosis, hypergranulosis and marked acanthosis (Figure). A dense, band-like, lymphocytic infiltrate at the dermo-epidermal junction and in the upper dermis was seen. Vascular degeneration and eosinophilic Civatte bodies were observed at the basal layer. The histopathologic features were those of hypertrophic lichen planus. Clinically drug-induced hypertrophic lichen planus was suspected.

Withdrawal of the incriminated drug, alendronate, and treatment solely with a topical steroid resulted in complete resolution of the lesions within 3 weeks. On follow-up no relapse of the lichen was observed for more than one year after withdrawal of the offending drug. The patient refused any further investigations including provocation tests or in vitro testing.

**Comment**

Drug-induced lichen planus is often very similar to idiopathic lichen planus [1]. Identification of a drug as an etiologic agent for lichenoid drug eruption is crucial for successful treatment, namely withdrawal of the offending drug. Our patient presented with lesions resembling hypertrophic lichen planus distributed symmetrically on the trunk and extremities but without mucosal involvement. The symmetric distribution and sparing of the mucous membranes have been described as clinical features that are observed more frequently in lichenoid drug eruptions [1]. The histopathologic features in our case were compatible with hypertrophic lichen planus. These hypertrophic lesions could be the result of severe and constant rubbing of the skin, induced by the severe pruritus experienced by the patient.

The latent period before the onset of the eruption, the complete resolution of the pruritus and the hypertrophic lichen planus only 3 weeks after withdrawal of the incriminated drug, as well as the lack of recurrence after discontinuing treatment with the offending drug, led us to the diagnosis of alendronate drug-induced lichen planus.

Furthermore, in our patient there was no evidence of other etiologic agents related to lichen planus, such as bacterial and viral infections, systemic disease or contact allergy.

Cutaneous adverse effects to alendronate reported in the literature are few and include pruritus, petechiae, fixed drug eruption, hypersensitivity, dysgeusia [2], rash [3] and allergic reaction [4]. Recently there has been a report of patients with oral ulcers [5] that were caused by direct mucosal injury from the drug.

Although spontaneous remission could occur in the course of lichen planus, in our patient the clinical features, the prompt resolution of the lichen after withdrawal of the offending drug, and the lack of recurrence are arguments in favor of a drug eruption. Our patient appears to be the first one with a lichen planus induced by alendronate.

### References


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**Capsule**

**A barrier to colon cancer?**

The gastrointestinal (GI) tract is lined by a layer of mucus that acts as a physical barrier between the luminal contents and the intestinal epithelium. This mucus is comprised of highly glycosylated proteins called mucins, whose precise roles in normal physiology and disease are poorly understood. Velcich et al. made the surprising observation that mice deficient in Muc2, the most abundant GI mucin, show an increased rate of intestinal epithelial cell growth and migration and spontaneously develop invasive tumors in the small intestine and rectum.

*Science* 2002;295:1726