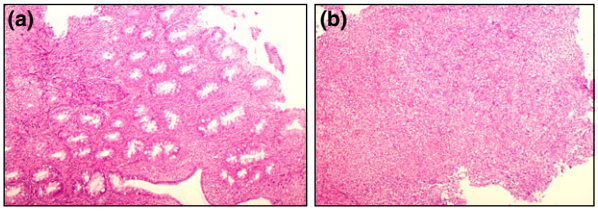

Figure 1 Histopathological examination of the biopsy section taken from the proximal rectum ulceration, (a) dense mixed inflammatory infiltrate of the intestinal mucosa, (b) non-specific granulomatous inflammatory tissue (haematoxylin and eosin staining, original magnification ×20).

Considering the above-mentioned symptoms typical for LGV and a history of unprotected receptive anal intercourse, blind rectal swab for *C. trachomatis* NAAT was repeated and blood for Chlamydia serology was taken. Repeated NAAT was negative again, but microimmunofluorescence test for *C. trachomatis* was positive in IgG (1 : 512) and IgA (1 : 16) antibodies, while IgM antibodies were borderline positive. A paraffin block from a recently performed biopsy was used to confirm the diagnosis. DNA was isolated by Qiasymphony® DSP DNA mini kit from five 5-μm-thick sections cut from the block. Detection of *C. trachomatis* was performed by touchdown enzyme time-release PCR with primers amplified in the 16S rRNA region.[6] Amplicons were analysed in 2% agarose gel with ethidium bromide, sequenced and compared with BLAST database. LGV serovar was confirmed by SeePlex® STI Master Panel 5. Doxycycline treatment 100 mg twice a day led to a gradual resolution of symptoms and was terminated after 21 days. In the control colonoscopy, regression of ulcerations was evident.

We are reporting a case of LGV proctocolitis mimicking IBD in the HIV-positive MSM in whom diagnosis was established by LGV biovar-specific bacterial DNA detection from a bowel biopsy specimen despite repeated negative NAAT for *C. trachomatis from rectal smears*. LGV usually causes diffuse proctitis, but different endoscopic findings were described including multiple mucous ulcers.[4] Mucous ulcers without diffuse proctitis are probably less common manifestation. The situation is even more complicated because endoscopic and histological findings in IBD and LGV can be similar, and thus, histological examination alone is not adequate to establish the diagnosis.[4] Few reports were published about retrospective testing of bowel biopsy samples for *C. trachomatis* DNA and LGV serovar in patients with confirmed or suspected LGV.[4,7] Our case shows that, in patients with ulcers in the proximal rectum, standard rectal swab is likely to fail to identify the disease. In our patients, sampling swab was repeatedly negative as it is normally only about 15 cm long. Whether proximal ulcers without diffuse proctitis are only a rare presentation of the disease or whether these patients are misdiagnosed remains unknown. These patients may be incorrectly diagnosed as having IBD despite standard recommended STI testing.

We would like to thank our colleagues from Department of Pathology, Na Bulovce Hospital, for the biopsy images.

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Pretibial myxoedema: a case report with scanning electron microscopy

Editor

Pretibial myxoedema (PTM) results from the accumulation of hyaluronic acid in the dermis and subcutis and is commonly associated with Graves' disease (GD). It occurs in up to 5% of patients with GD and in 13% of patients with GD and ophthalmopathy. PTM occasionally occurs in thyrotoxicosis but is much less frequent in patients with Hashimoto thyroiditis, primary hypothyroidism and euthyroidism. Clinically, PTM may manifest as waxy, erythematous nodules or plaques, often with evident follicular openings located on the tibial region. Diagnosis is

confirmed by light microscopy.[1] We examined the dermis of a PTM case with a scanning electron microscopy (SEM), which identified unreported findings of this condition.

A 39-year-old White male patient, diagnosed with Graves' disease with thyrotoxicosis and exophthalmos, under treatment with propylthiouracil, presented with waxy, yellowish or slightly erythematous plaques, with prominent follicular openings on the anterior aspect of his legs (Fig. 1a). After 6 months, he underwent radioactive iodine therapy progressing to hypothyroidism. Histopathological samples obtained by incisional biopsy of lesions revealed accumulation of mucin in reticular dermis leading to separation of collagen bundles. Star-shaped fibroblasts were seen under haematoxylin and eosin staining (H&E) (Fig. 1b) and more evident with Alcian Blue (Fig. 1c). Intralesional infiltration with triamcinolone was used with significant improvement and subsequent partial relapse.

On examining the dermal surface of biopsy punch using SEM, three distinct areas could be visualized: areas with normal collagen bundles without deposit (×650) (Fig. 2a), a transitional area with normal and slightly distorted collagen bundles (arrow) and mucin deposition (2.200×) (Fig. 2b). In the most affected area, in the deep reticular dermis, the deposit was tightly packed, covering, separating and disrupting the bundles and the collagen fibres (850×) and (8.500×) (Figs. 2c,d).

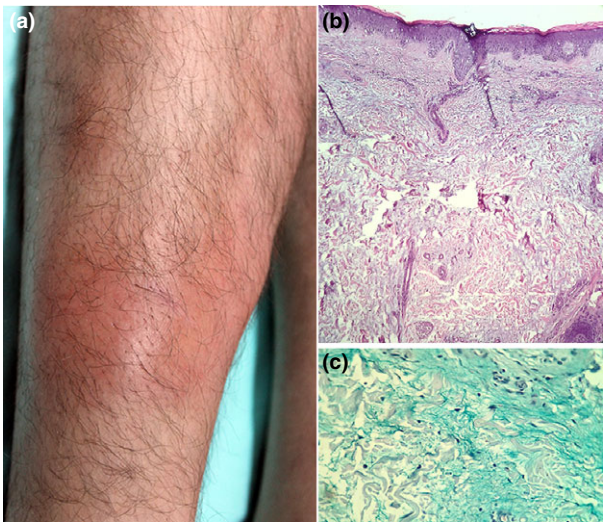

Figure 1 (a) Erythematous plaques on the anterior aspect of the legs (b) Normal epidermis; dermis with extensive interstitial deposits of mucin (amorphous, amphophilic material), permeating and pushing away the collagen fibres, more evident in the reticular dermis HE-40× (c) normal amount of star-shaped fibroblasts, Alcian Blue 400×.

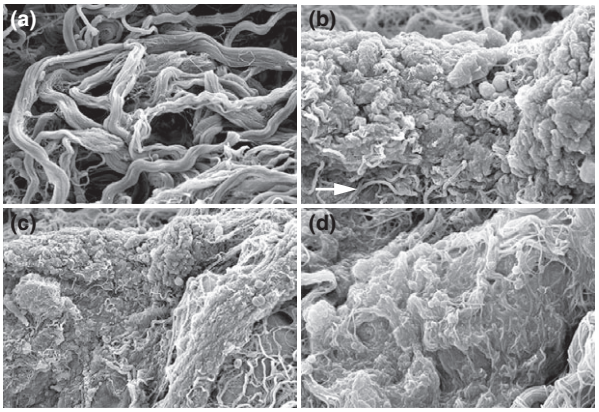

Figure 2 Scanning electron microscopy (a) normal area with collagen-forming bundles (×650), (b) transition of normal bundles (arrow) to the affected area (×2.200); (c) affected area with 'packing' of collagen with some loose bundles and fibres (×850); (d) higher magnification showing the 'packing' of collagen by deposits (×8.500).

Light microscopy can establish PTM diagnosis. The deposit can be visualized using special staining (Alcian Blue, colloidal iron or toluidine blue), which produces spaces among the collagen fibres. Normal amount of fibroblasts can be evidenced, some of them with a star-shaped outline.[2]

Even though the exact cause is still elusive, it is known that besides immunological effects, mechanical factors contribute to the formation of PTM. Antibodies bind to thyroid-stimulating hormone receptor and stimulate the dermal fibroblasts, increasing the production of glycosaminoglycans. The accumulation of these polysaccharides separates the collagen fibres and obstructs the lymphatic vessels, causing oedema. The predisposition of the pretibial region could be explained by trauma.[2]

Pretibial myxoedema has been already examined by transmission electron microscopy. It was observed that the bundles and the collagen fibres were separated by wide electron-lucent gaps. Fibroblasts have a star-shaped appearance with extensive formation of cytoplasmic processes.[1]

Scanning electron microscopy has been shown to be helpful in studying the ultrastructure of dermal diseases with deposits, as previously reported in other studies. In this report, we were able to reveal the accumulation of mucin in reticular dermis. It separates, disrupts and covers most of the collagen fibres, but it does not assume a peculiar morphological appearance as it does in amyloidosis (stone-like appearance)[3] and in lichen sclerosus and atrophicus (a pearl necklace appearance).[4] SEM is not used routinely; however it can provide additional ultrastructural information of conditions with dermal involvement.

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Trichobacteriosis axillaris caused by *Dermabacter hominis*

Editor,

Trichobacteriosis is a frequent superficial bacterial infection of the hair, mainly affecting sweat gland-bearing areas such as axillae and genital region, and also rarely the scalp.[1–3] The disease is characterized by bacterial overgrowth forming nodular concretions, usually of yellow colour (infrequently black or reddish), which are firmly attached to the hair shaft.[1,4] Clinical hallmarks are rancid, acidic odour, unpleasant 'dirty' sensation and staining of clothes. Although most cases have been associated with different species of corynebacteria, the full aetiologic spectrum of this infection is uncertain.[1,5] Here, we report the first case of trichobacteriosis caused by *Dermabacter hominis*.

A 30-year-old otherwise healthy man presented with an unpleasant strong axillary odour and 'dirty' bilateral armpits since 2 months. Physical examination revealed nodular concretions along multiple hair shafts in both axillae. These casts were firmly adhered and could not be removed from the hair shafts. Polarized trichoscopy showed creamy-yellow adherent concretions along the entire hair shafts, which produced a white fluorescence under Wood's lamp examination (Fig. 1a,b). Examination by low-power microscopy revealed that the affected hair was encased by irregular, approximately 100- to 200-μm-wide casts (Fig 1c). Further microscopic examination of

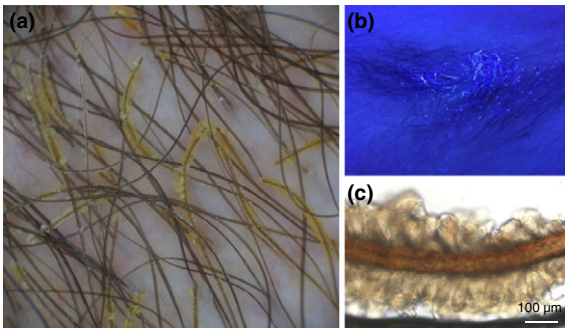

Figure 1 (a) Polarized trichoscopy (10×) of axillary hair showing multiple yellow-creamy concretions. (b) Wood light examination of affected areas displaying white fluorescence. (c) Low-power microscopy of hair with irregular, 100- to 200-μm-wide cast.

granules by Gram stain demonstrated conglomerates of Gram-positive pleomorphic rods surrounding an amorphous matrix (Fig. 2a,b). Hair samples and segregated conglomerates were then separately inoculated onto blood agar (bioMérieux, Marcy l'Etoile, France) and incubated at 37 °C in a 5% CO_2 atmosphere. After 24 h, both cultures showed multiple grey, convex colonies of 0.5 mm, which after 48 h grew to a diameter 1 mm (Fig. 2c). Gram staining of the colonies showed Gram-positive pleomorphic rods without branching (Fig. 2d), morphologically identical to those observed from Gram stains of the casts (Fig. 2a,b). The microorganism was identified by matrix-assisted laser desorption/ionization time-of-flight (MALDI-TOF) mass spectrometry (Vitek MS, bioMérieux) as *D. hominis*. The patient was advised to shave the affected areas and administer clindamycin ointment (1%) twice daily for 4 weeks. After this time,

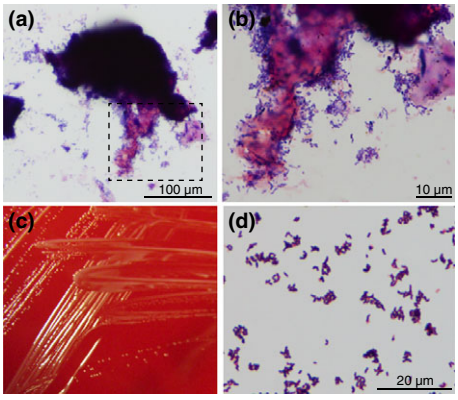

Figure 2 (a) Gram stain of a concretion demonstrating masses of Gram-positive rods surrounding an amorphous matrix; details shown in (b). (c) *Dermabacter hominis* culture on blood agar showing small convex colonies after 48 h. (d) Gram stain of the culture demonstrating Gram-positive pleomorphic rods.