colonic anastomosis, and an incidental finding was spondylolisthesis of the fifth lumbar and first sacral vertebrae (L5/S1).

After several weeks of severe back pain MRI of his lower back showed inflammation in the L5/S1 disc but no connection between the disc and the fistula (Figure 1 and Figure 2). Aspiration of the disc yielded pus from which pseudomonas species were grown. The patient was treated with long-term antibiotics and became free from back pain.

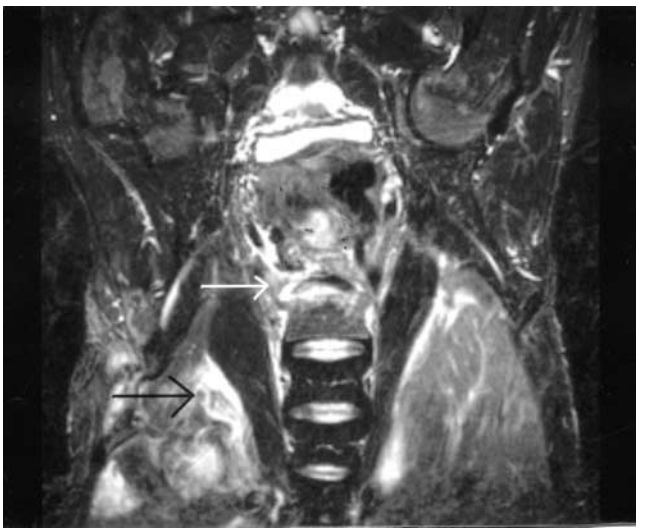

Figure 1 **Coronal STIR MRI.** High signal lateral to the left psoas muscle indicates inflammation in this area; however, the inflammation is not seen to extend directly into the L5/S1 disc space (black arrow). High signal at the L5/S1 disc space represents discitis (white arrow)

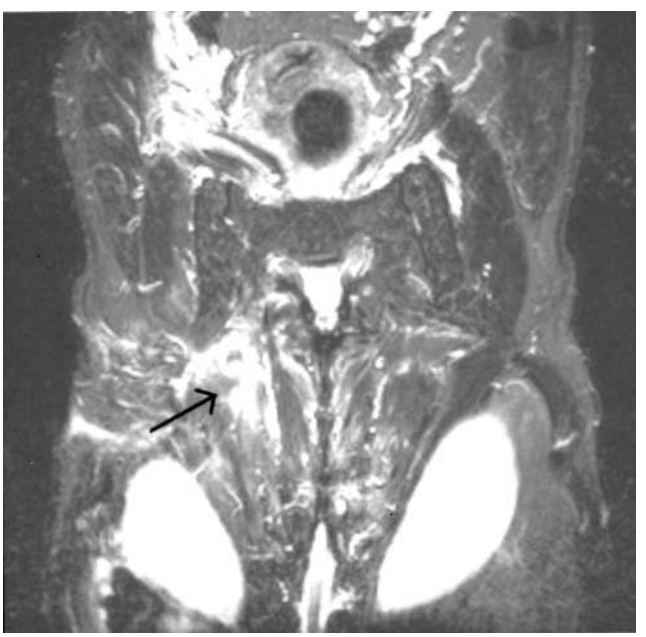

Figure 2 **Coronal STIR MRI through posterior abdominal wall.** High signal intensity lateral to left erector spinae muscles (arrow) indicates inflammation; however, this inflammation is not directly extending into the L5/S1 disc space

The fistula also healed. Subsequent colonoscopy revealed inflammation of the transverse and ascending colon, and biopsy appearances were compatible with Crohn's disease. He was started on azathioprine and the ileostomy was closed without incident.

COMMENT

The striking feature of this case was the apparent lack of a fistula from the gut to the infected disc. Pande and co-workers[2] described a patient not unlike ours with a presacral abscess and osteomyelitis of the L4 and L5 vertebrae, but the route of infection in that case was clearly an enteric fistula. We postulate that in our patient the route of infection was haematogenous, through the vertebral venous system and then retrogradely into the vertebral venous plexus.

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Facial eczema and sight-threatening glaucoma

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Topically applied steroids are effective in the treatment of eczema, but when applied regularly to facial lesions can cause sight-threatening glaucoma.

CASE HISTORY

A man aged 42 was referred to eye casualty by his general practitioner after two months of intermittent and increasingly frequent blurring of vision in his left eye. He had atopic eczema (present from childhood)

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and mild asthma but said he was not using any medication. The eczema was localized to the face and neck, with areas on the chest and arms; it did not appear symptomatic.

Visual acuities were 6/6 in both eyes. On slitlamp examination there were no keratic precipitates, aqueous flare or cells, nor was there pseudoexfoliation or evidence of pigment dispersion syndrome or any cause of secondary glaucoma. Intraocular pressures (IOPs) were 33 mmHg on the right and 50 mmHg on the left. Gonioscopy showed normal open grade IV angles with no increased pigmentation or rubeosis. His optic discs were healthy and symmetrical with cup-to-disc ratios of 0.2. There was no pathological change in the nerve fibre layer, no sectoral neuroretinal rim defect, and no excavation. Flyover vessels were absent. Humphrey visual fields were also normal as were pupillary reactions. Routine laboratory tests including blood count, erythrocyte sedimentation rate and liver function tests yielded nothing of note. He was treated with oral acetazolamide 250 mg four times daily and topical timolol 0.25%, which was changed to dorzolamide three times daily when the beta blocker aggravated his asthma. The next day his IOPs were 14 and 15 mmHg. On review 5 and 8 days after presentation the IOPs remained about the same. An outpatient appointment was made for two months, with instructions to continue the dorzolamide eye drops. In the absence of any signs of secondary glaucoma, this was thought to be an unusual presentation of primary open-angle glaucoma.

He returned to eye casualty six weeks after his initial referral with headache and blurred vision on the left. IOPs were 27 mmHg (right) and 49 mmHg (left). The symptoms were occurring in episodes of a few minutes, several times a day, progressive in nature. Over the previous week the vision had been particularly cloudy in the morning. The patient was using timolol drops as well as dorzolamide—the timolol drops having been restarted on his own initiative because he was concerned about damage to his vision. Intravenous acetazolamide initially brought the pressure down to 13 and 35 mmHg. Polymerase chain reaction testing for *Herpes simplex* virus was negative. Again he denied the use of any other medication. Over the subsequent 18 days the IOP became refractory to all medical therapy, including intravenous mannitol, and on the 18th day he underwent left trabeculectomy augmented with 5-fluorouracil, under general anaesthesia. This was two months after his initial referral. Postoperatively his IOP stabilized in the teens without medication. Within days, the right eye developed refractory high IOP, and two weeks from the first procedure he underwent right trabeculectomy with 5-fluorouracil. For four more months he attended, and during this time his IOPs remained between 9 and 14. When he then missed follow-up appointments his general practitioner was contacted. It emerged that, before development of the eye symptoms, the patient (who had now left the country) had been issued with a repeat prescription for several steroid ointments, which were for facial and periorbital eczema. Topical medication had been prescribed by the general practitioner for daily use. The patient had been using his ointments less frequently than this (about twice a week) over the past 2 years, and had not been using them regularly in the three months before we first saw him. Hydrocortisone 1%, betamethasone 0.1% and 0.25% and 0.05% with salicylic acid had been prescribed to the patient on five separate occasions over 2 years.

COMMENT

Topical steroids are routinely used in the management of dermatoses.[1] In this case bilateral sight-threatening glaucoma in a young adult was induced by steroid cream and did not resolve with medical therapy. When applied to the face steroid creams (some of which are available without prescription in the UK[2]) can raise the IOP and irreversibly damage the visual field[3]. If the applications are discontinued, the IOP typically falls after a lag phase, but glaucomatous damage may continue nonetheless. The duration of steroid use is important in the development of glaucoma. In previous reports, the raised intraocular pressure has been attributed to direct steroid seepage into the eye.[4,5] Salicylic acid (present in one of the preparations used by our patient) facilitates penetration. Topical steroids are categorized by their potency into four groups, from mild to very potent. Betamethasone 0.1% and 0.05% are potent, hydrocortisone 1% mild and betamethasone 0.025% moderate. There is no warning in the *National Formulary*, or in the specific drug information leaflets, about risk of glaucoma with these preparations. The fact that the patient claimed not to be using a medication illustrates a common misconception regarding topically administered agents.

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