repair was made to the posterior wall and the canal was closed again behind the cord. The patient became pain-free and 3 years later he remains so, with a normal sex life.

Case two

A 53-year-old man had a left inguinal hernia repair (Halsted) and four months later reported pain around the site of the scar. A sharp, burning pain localized to the scar with no radiation to the scrotum occurred only on ejaculation. The patient was otherwise fit and well. He had been sterilized fourteen years previously without experiencing any ejaculatory pains postoperatively. On examination there was decreased sensation to light touch and pin prick beneath the herniorrhaphy scar. As in case one the patient was initially seen in the pain clinic and then referred for exploration when treatment with amitriptyline had failed.

At exploration the vas, which was superficial to the external oblique aponeurosis, was found tethered and twisted at the superficial ring. The vas was untethered and the cut ilioinguinal nerve was traced backwards and cut cleanly away from the scar. The patient became pain free and remains so 4 years post-exploration.

COMMENT

Although painful ejaculation after inguinal hernia repair is evidently rare, painful ejaculation itself is not uncommon, most cases being secondary to urological conditions such as prostatic cysts[2], prostatitis[3] and seminal vesicle calculi[4]. In children, groin surgery often injures the vas deferens and may be a cause of painful ejaculation later in life.

Pain around a herniorrhaphy scar is commonly reported, the pain being variably described as somatic or neuropathic. Somatic pain has been attributed to trauma to the pubic tubercle (possibly by a suture), while neuropathic pain (usually in the area of the genitofemoral or ilioinguinal nerve) may result from operative nerve damage or scar tissue development.

What could be the mechanism of painful ejaculation after hernia repair? In the single previous report, dysfunction of the periurethral structures was postulated, but that patient's symptoms were mild and surgical exploration revealed kinking and scarring affecting both vasa deferentia. Resultant engorgement within the vas, with the rhythmic contractions of ejaculation, might lead to the pain. In the senior author's (MJH) opinion, scarring around the vas is especially common with Halsted inguinal hernia repair, which leaves the cord structures superficial to the external oblique aponeurosis. Neuroma formation at the scar site is a recognized cause of wound pain and our excision of the ends of the ilioinguinal nerves may have contributed to the relief of symptoms in these patients.

We recommend that patients with painful ejaculation after inguinal hernia repair should be considered for exploration, to detect and treat scarring, tethering or kinking of the vas.

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Heart failure with fludrocortisone in Addison's disease

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Autoimmune destruction of adrenal cortex causes Addison's disease and demands lifelong replacement of glucocorticoids and mineralocorticoids. We report a patient with a normal heart who developed heart failure on replacement fludrocortisone.

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CASE HISTORY

On admission to hospital a 47-year-old woman gave a six-month history of intermittent abdominal pain, vomiting and diarrhoea. She was clinically dehydrated and pigmented and her blood pressure was 110/70 mmHg. Examination findings were otherwise normal, as was the chest X-ray. Serum sodium was 122 mmol/L, potassium 5.8 mmol/L, urea 11.2 nmol/L and creatinine normal. Addison's disease was confirmed by the absence of a cortisol response to tetracosactrin 250 μg (serum cortisol 19, 16, and 13 nmol/L at 0, 30 and 60 min) and a baseline corticotropin of 112 ng/L (normal 5–50). Anti-adrenal antibodies were present. Replacement therapy with hydrocortisone (30 mg

daily in divided doses) and fludrocortisone (100 μg daily) was started and she was discharged a few days later having improved clinically and with normal serum electrolytes.

She was readmitted two weeks later with oedema, dyspnoea and a 3 kg weight gain. She was tachypnoeic and basal crepitations were heard in the chest; blood pressure was 120/70 mmHg and she was afebrile. Chest X-ray showed cardiomegaly and pulmonary congestion. On echocardiography the heart valves and chamber sizes were normal but there was a small pericardial effusion. The electrocardiogram was normal. There was no suggestion of non-compliance with the treatment regimen or dispensing error. She was treated with intravenous frusemide and the fludrocortisone dose was reduced to 50 μg daily and then stopped after two days. Over the next four days her cardiac failure improved and diuretic therapy was withdrawn. She was discharged a week after admission. Two months later her serum and urine electrolytes and cortisol day profile were normal. Aldosterone was undetectable in either supine or recumbent posture but fludrocortisone was not restarted. Eight months later, she was admitted with acute addisonian crisis (serum sodium 118 mmol/L, potassium 6.2 mmol/L) following acute gastritis and responded well to intravenous fluid and hydrocortisone. Fludrocortisone was started with a dose of 25 μg daily and increased to 100 μg over two months. On review after four months she was doing well, without fluid retention, and her serum electrolytes were normal.

COMMENT

The association of congestive cardiac failure with fludrocortisone therapy was reported by Knowlton and Baer in 7 of 22 adults with Addison's disease followed for over 30 years[1]. All had unrelated ischaemic, valvular, or hypertensive heart disease and were elderly. The dose of hydrocortisone and fludrocortisone did not differ between the groups with and without heart failure. Derish *et al.* reported an 11-year-old boy with Addison's disease who developed cardiac failure due to a reversible cardiomyopathy[2]; and Willis *et al.* described heart failure secondary to fludrocortisone and hydrocortisone therapy in a 6-year-old boy with Addison's disease[3].

The patient reported here was started on a standard dose of fludrocortisone. There was no suggestion of abnormal myocardial function before the onset of congestive failure. When myocardial function is normal, an increase in circulatory volume is expected to increase contractility but further increases in the volume load will eventually cause it to decline. We attribute the fluid overload and heart failure in our patient to fludrocortisone therapy. The possible explanation could be renal adaptation to chronic salt and water deprivation. With mineralocorticoid replacement, this previously adaptive mechanism may have resulted in sodium and water retention, leading to heart failure. This view is supported by the fact that, over the subsequent months, the patient tolerated a gradually increasing dose of fludrocortisone.

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Osseous metaplasia in caecal adenocarcinoma

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Osseous metaplasia in colorectal adenocarcinoma is a histological curiosity. Until now, the condition has been described only in left colonic lesions. Here we report a patient in whom osseous metaplasia developed in a caecal adenocarcinoma.

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CASE HISTORY

A woman aged 47 was admitted with right iliac fossa pain and a leucocyte count of 12.6×10^9/L. Laparotomy revealed an inflamed, perforated appendix with appendicular mass. Two months later rectal bleeding developed and her haemoglobin was 8.7 g/dL. Skin nodules and a fistula appeared in her abdominal wound. On colonoscopy a large mass was seen in the caecum, reported on biopsy as adenocarcinoma. Computed tomography revealed a 4 cm mass arising from the caecum and invading the abdominal wall with multiple liver secondaries. The patient underwent right hemicolectomy *en bloc* with the involved abdominal