COMMENT

The initial moderate headache and mild pyrexia in the present case was not alarming, in the absence of meningism. CT at that stage might have allowed an attempt at further decompression, but the acute deterioration was not preceded by warning signs. Chemical meningitis has been reported from intracranial rupture of several types of cystic structures, including intracranial dermoid cysts,¹ cholesteatoma following radical mastoidectomy, remnant after geniculate ganglion surgery, and epidermoid cysts in the cerebellar pontine angle, as well as from a craniopharyngioma or cyst of Rathke's pouch.^{2,3} Accumulation of the oily irritant material within a cyst lined by squamous epithelium is an irregular phenomenon: some remain quiescent before and after decompression. Radiotherapy may or may not affect cyst recurrence. Long-term review of these patients is often in the hands of endocrinologists, who need to arrange intermittent scans to identify cyst recurrence.⁴

REFERENCES

- 1 Stendal R, Pietila TA, Lehmann K, Kurth R, Suess O, Brock M. Ruptured intracranial dermoid cysts. *Surg Neurol* 2002;**57**:391–8
- 2 Kulkarni V, Daniel RT, Pranatiriharan R. Spontaneous intraventricular rupture of craniopharyngioma cyst. *Surg Neurol* 2000;**54**:249–53
- 3 Satoh H, Uozumi T, Arita K, *et al.* Spontaneous rupture of craniopharyngioma cysts. A report of five cases and review of the literature. *Surg Neurol* 1993;**40**:414–19
- 4 Clayton RN, Wass JAH. Pituitary tumours: recommendations for service provision and guidelines for management of patients. Summary of a consensus statement of a working party. *J R Coll Phys Lond* 1997;**31**:628–36

Inguinal lumps misidentified as ectopic testis

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J R Soc Med 2004;**97**:586–587

If an apparently hypoplastic or cystic intra-inguinal testicular nubbin is not subjected to full histological analysis, an intra-abdominal testis can be missed.

CASE HISTORIES

Case 1

At age 20 a man with an absent left testis was referred with a swelling in the left inguinal canal which was removed as a

suspected undescended testis. The histopathology was reported as a 3.5 cm × 2.5 cm thick-walled cyst with firmer nodular area and lined by flattened cuboidal epithelium with smooth muscle in the wall. Epididymal tissue was present in the wall but no testicular tissue was seen. It was thought that any testicular remnants had become atretic and no further action was taken. Having been symptom-free for 13 years the man began to experience intermittent swelling and pain in the left groin. Abdominal CT revealed a large complex mass in the left retroperitoneal region involving the psoas muscle and the left ureter. The mass was removed and proved to be a seminoma. This was presumed to have arisen from an abdominal left testis.

Case 2

A man aged 48 known to have a right undescended testis reported a few months of slight discomfort and a mass in the right inguinal canal. A right inguinal orchidectomy led to resolution of his symptoms. On microscopy the 1.5 × 1 cm mass showed fibrovascular soft tissue, vas deferens and epididymis with cystic dilatation of tubules and retained secretions. No unequivocal residual or remnant of testis was seen. Subsequent abdominopelvic MRI revealed a well-defined mass at the level of the right internal ring (Figure 1). This was removed intact laparoscopically and proved to be a testis with histopathological features of atrophy.

COMMENT

Testicular descent is a complex process and this accounts for the variety of positions observed in cases of maldescent.¹ Undescended testis occurs in 4–5% of males at birth and of these 15–20% are intra-abdominal.

The phenomenon of an absent testis in association with inguinal hypoplastic vessels and/or vas deferens is well

Figure 1 T1 weighted CT scan, Case 2. Large arrow, ectopic right testis; small arrow, colon

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documented² and is presumed to be due to a vascular event *in utero* or infancy. In such cases, however, it is important to be aware that the testis may be in a different position.³ On the macroscopic level it is vital to be aware that residual epididymal and vas deferens tissue may undergo cystic changes with time and hence an inguinal mass observed at surgery may mimic but not represent a testis. Furthermore, such tissue may be associated with a symptomless intra-abdominal testis which may or may not be hypoplastic. Such tissue must be recognized and removed since, as in case 1, it can undergo malignant change. When a mass that is clinically an undescended testis shows no histologically proven testicular tissue we advocate further imaging and consideration of laparoscopy.

REFERENCES

- 1 Hutson JM, Hasthorpe S, Heyns CF. Anatomical and functional aspects of testicular descent and cryptorchism. *Endocr Rev* 1997;**18**:259–80
- 2 Grady RW, Mitchell ME, Carr MC. Laparoscopic and histologic evaluation of the inguinal vanishing testis. *Urology* 1998;**52**:866–9
- 3 Wolfenbittel KP, Kok DJ, Den Hollander JC, Nijman JM. Vanished testis: be aware of an abdominal testis. *J Urol* 2000;**163**:957–8

Severe salicylate poisoning treated conservatively

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J R Soc Med 2004;**97**:587–588

Severity of salicylate poisoning has been classified in terms of blood concentrations. However, in decisions on treatment, other factors must be taken into account.

CASE HISTORY

A woman of 86 was brought to the emergency department having been found unresponsive in her chair by a relative. Her medical history included polymyalgia rheumatica, transient ischaemic attacks and depression. Her regular daily medication was aspirin 75 mg, prednisolone 5 mg and nitrzapam 10 mg. On examination she was apyrexial but

tachypnoeic with oxygen saturation 92% on air. She opened her eyes and moved all her limbs to command. Routine blood examination showed a raised white cell count with neutrophilia and deranged renal function (urea 23.8 mmol/L, creatinine 288 μ mol/L). Arterial blood analysis revealed a pH of 7.50, pCO₂ 2.23 KPa, pO₂ 12.2 KPa, bicarbonate 19 mmol/L (base deficit 4.5 mmol/L) and lactate 2.7 mmol/L. CT of the head was normal. Because of the respiratory alkalosis salicylate was measured and proved to be 850 mg/L.

A regional toxicology service advised that, in a patient with salicylate >700 mg/L, haemofiltration would normally be indicated. The patient was given activated charcoal to minimize gastrointestinal salicylate absorption and transferred to the intensive care unit where a bicarbonate infusion (1.4% sodium bicarbonate at 300 mL/h) was set up to combat the metabolic acidosis and alkalize the urine. Urinary pH was maintained at >7.5. Within 4 h of the infusion being started, the patient's level of consciousness had improved greatly, blood gases were returning to normal and the salicylate concentration was lower. She later told us that she had taken an intentional overdose of aspirin (tablets of 300 mg, not the 75 mg tablets she received on prescription).

COMMENT

Salicylates when taken in excess cause respiratory alkalosis by direct stimulation of the respiratory centre in the medulla, followed by a metabolic acidosis due to uncoupling of oxidative phosphorylation and blocking of the Krebs cycle. Once absorbed, they remain in the ionized form and hence do not diffuse easily into tissues unless a metabolic acidosis supervenes; they are then converted into the non-ionized form which readily diffuses into the brain. Similarly, the non-ionized form is readily reabsorbed from the renal tubules, slowing elimination. The effects vary from nausea, vomiting, tinnitus, deafness and confusion to coma.^{1,2}

The severity of salicylate toxicity has been classified by blood concentration—mild 300–500 mg/L, moderate 500–700 mg/L, severe >700 mg/L. However, there is a view that concentration is not the only or even the major criterion. Additional criteria relating to severity include age over 70 or under 10 years, central nervous system features, hyperpyrexia, metabolic acidosis, pulmonary oedema and late presentation.³ Standard treatment includes the use of activated charcoal or gastric lavage, prompt rehydration and correction of any electrolyte abnormalities. A bicarbonate infusion should be started to combat acidosis, which aids in conversion of the non-ionized form of the drug to the ionized form, minimizing tissue toxicity and increasing renal elimination. Although haemofiltration has been

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