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

Sandeep Basavarajaiah MBBS¹

Paul Sigston FRCP FRCA² Kuno Budack MRCP¹

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 µmol/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

Departments of ¹General Medicine and ²Intensive Care Medicine, Kent and Sussex Hospital, Tunbridge Wells TN4 9AT, UK

Correspondence to: Dr S Basavarajaiah

E-mail: drsandy270478@yahoo.co.uk

recommended for patients with a salicylate exceeding 700 mg/L, our patient's age and frailty led us to try urinary alkalization first and she showed rapid clinical improvement. A previous paper⁵ has compared the use of bicarbonate infusion with haemofiltration in a single patient following two episodes of severe salicylate poisoning. The initial rate of decline in the salicylate concentration was faster with alkalization of urine.

REFERENCES

- 1 Bari N. Salicylate poisoning. *J Pak Med Assoc* 1995;**45**:160–1
- 2 Krause DS, Wolf BA, Shaw LM. Acute aspirin overdose: mechanisms of toxicity. *Ther Drug Monit* 1992;**14**:441–51
- 3 National Poisons Information Service (NPIS). *Salicylates—Features and Management* [http://www.spib.axl.co.uk]
- 4 Wrathall G, Sinclair R, Moore A, Pogson D. Three case reports of the use of haemodiafiltration in the treatment of salicylate overdose. *Hum Exp Toxicol* 2001;**20**:491–5
- 5 Higgins RM, Connolly JO, Hendry BM. Alkalization and hemodialysis in severe salicylate poisoning: comparison of elimination techniques in the same patient. *Clin Nephrol* 1998;**50**:178–83

Recurrent laryngeal nerve palsy, dysphagia and aortic fistula

Charles Bailey MRCS¹ Andrew Toner BM BCh¹
John Nottingham MRCPath² David Hunter FRCS¹

J R Soc Med 2004;**97**:588–589

In an elderly patient the most likely cause of recurrent laryngeal nerve palsy and dysphagia is a malignant tumour. Among the other conditions to be considered is tuberculosis.

CASE HISTORY

A woman aged 89 consulted her general practitioner after four weeks of hoarseness, sore throat and difficulty with swallowing. She also reported pain in the right side of the neck and recent breathlessness. Her sister had died from laryngeal cancer at age 93 and she was concerned that she might have the same condition. On referral to the ear, nose and throat department she was found to have a right

recurrent laryngeal nerve palsy. Subsequent oesophago-gastroduodenoscopy revealed a 3 cm ulcerated lesion in the upper oesophagus with what was considered to be an associated tracheo-oesophageal fistula (Figure 1). The provisional diagnosis was oesophageal squamous carcinoma and her dysphagia was relieved by a 12 cm stent placed under endoscopic and X-ray guidance. Biopsies of the ulcer showed fragments of oesophageal mucosa with a squamous epithelial covering. Focal ulceration was associated with granulation tissue but there was no evidence of malignancy. A chest X-ray showed no abnormality apart from slight cardiomegaly. One week later she developed severe back pain and died. At necropsy the cause of death was found to be a massive gastrointestinal bleed from an aorto-oesophageal fistula. Although the macroscopic appearance was typical of a squamous carcinoma, histological examination revealed the fistula to be caused by oesophageal tuberculosis with erosion into the aorta. The patient had lived in England all her life and had not recently travelled abroad.

COMMENT

Tuberculosis can affect the oesophagus either indirectly by ulceration from infected mediastinal or hilar lymph nodes or directly as part of a miliary spread. Oesophageal tuberculosis poses a considerable diagnostic challenge since the presentation and macroscopic appearances are very similar to those of carcinoma and only 20% have evidence of tuberculosis on chest X-ray.¹ In the present case, even when malignancy had been excluded by biopsy, there were other possibilities such as peptic ulceration and Crohn's disease. In a review of 54 cases only 12 had the diagnosis made on mucosal biopsy alone;¹ in others diagnosis depended on

Figure 1 3 cm ulcerated lesion in upper oesophagus

Departments of ¹General Surgery and ²Histopathology, Northampton General Hospital, Northampton NN1 5B, UK

Correspondence to: Charles Bailey, c/o Mr Hunter's Secretary, Northampton General Hospital

E-mail: emhbailey@doctors.org.uk