adverse reactions to a particular drug. Both the Swedish and UK regulatory agencies issued warnings, but their substance and emphasis differed, and this may have introduced some bias. Neither regulatory authority, though, emphasised the problem of peripheral neuropathy, which appears to be the major difference in the profiles of adverse reactions to the drug.

We are grateful to Dr B E Whiholm of the National Board of Health and Welfare, Department of Drugs, Sweden, and Dr B H Ch Stricker of the Netherlands Centre for the Monitoring of Adverse Reactions to Drugs for their help and for supplying national data. We are also grateful to the Committee on Safety of Medicines for permission to quote from the UK Adverse Reactions Register, and to Professor Abe Goldberg for his invaluable help and criticism.

Reference


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SHORT REPORTS

Unusual complication of perforated appendix

Peritonitis, local or generalised, is a common accompaniment of acute appendixitis, and residual septic foci may occur. The incidence of these has decreased over recent years, partly as a result of improved surgical techniques but largely owing to the use of prophylactic antimicrobial agents,1 whether local2 or systemic.4 We routinely use antibiotic peritoneal lavage (using a solution of 1 g tetracycline/100 ml saline) in cases of peritoneal sepsis, with good results.5 6 Residual intraoperative sepsis may still occasionally occur in the form of pelvic, interloop, and subphrenic abscesses, in addition to local sepsis in relation to the appendix stump. We describe an abscess in the scrotum after appendicectomy.

Case report

A 9-year-old boy was admitted with a two-day history of colicky central abdominal pain that localised in the right iliac fossa on the day of admission. He was toxic, and examination suggested acute appendicitis with peritonitis. At laparotomy, through a grid-iron incision, a perforated retrocaecal appendix was found with free pus in the peritoneal cavity. Culture of the pus subsequently yielded a profuse growth of Escherichia coli. After appendicectomy thorough peritoneal lavage with tetracycline in saline was carried out, and the wound was closed without drainage. No other antibiotic was given.

After the operation he had a slight fever, which was attributed to a low-grade wound infection. The sutures were removed on the sixth postoperative day, and on the same day he developed quite suddenly a painful, hot, red swelling of the right side of his scrotum. Torsion of the right testis was provisionally diagnosed and exploration carried out. On opening the tunica vaginalis 5 ml of pus was released, but the testis and epididymis were normal. The wound was drained. His fever settled rapidly and he made an uneventful recovery. Culture of the pus proved sterile. Subsequent outpatient review at three months showed no evidence of further sepsis.

Comment

Co-existence of a perforated appendix and right-sided congenital hydrocele or hernia must be common, yet we could find no report of residual sepsis in the scrotum. Our patient had no history or clinical finding suggestive of a hydrocele or hernia, but presumably a connection between the peritoneal cavity and the tunica vaginalis was present. A patent processus vaginalis was not found at the second operation, but obliteration of the channel after the sepsis is probable. Sources of the sepsis from the abdominal wound into the inguinal canal seems unlikely, as the sepsis in the scrotum was confined within the tunica vaginalis.

Acute epididymo-orchitis after prosthetic surgery is well recognised, infection reaching the testis via the vas deferens. It might be postulated that retroperitoneal infection from a retrocaecal appendix could track alongside the vas, but in this case there was no evidence of infection of the epididymis or testicle.

Opponents of intraoperative peritoneal lavage believe that it may spread sepsis within the peritoneal cavity. We have been unable to find any evidence for this view, but it is conceivable that the lavage encouraged the passage of organisms down a patent processus. It is unlikely that the use of systemic antibiotics would have prevented this occurrence. Recent (unpublished) observations have shown that tetracycline lavage leads to a therapeutic serum tetracycline concentration within an hour, and the pus trapped in the scrotum was shown to be sterile. It is therefore difficult to know how this complication might be avoided, even when a congenital hernia or hydrocele is suspected before operation.

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Hypophosphataemic vitamin-D resistant rickets may need phosphate supplements

The commonest form of vitamin-D resistant rickets is associated with a renal tubular defect producing hyperphosphaturia and hypophosphataemia, and has been treated with vitamin D up to 100 000 units daily together with phosphate supplements.1 Recent reports2,3 suggest that phosphate supplements are not required when treatment is with 1α-hydroxy vitamin D3 (1α-OH-D3), but the results show that serum phosphate concentrations remained subnormal. We describe a child in whom rickets was not controlled with 1α-hydroxy vitamin D3 until oral phosphate supplements were given.

Case report

A girl delivered at full term after breech presentation weighed 3150 g and was bottle fed. Welfare clinic multivitamin drops were given daily for one year. Her parents, brother, and sister were healthy and there was no family history of bone disease. She was very active, had a good appetite, and the milestones of development were normal. She walked at 13 months but her gait was waddling and her legs became increasingly bowed. When she presented at 23 months her height was —3 SD and weight —2 SD from the mean (76 cm and 9.6 kg). Radiographs showed evidence of severe rickets affecting wrists and knees. Results of investigations were: serum calcium concentration 2.5 mmol/l (10.1 mg/100 ml); phosphate concentration 0.7 mmol/l (2.1 mg/100 ml); alkaline phosphatase activity grossly raised;

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parathyroid hormone concentration 0.6 ng/ml (normal <1.0); and 25-
hydroxycholecalciferol concentration 46 nmol/l (18-9 ng/ml) (normal 10-75
nmol/l; 4-30 ng/ml). Blood gases, amino-acids, urea and electrolyte concent-
trations, and results of ophthalmic slit-lamp examination were normal.
Urine pH was 5.0; calcium concentration was under 1 mmol/l (4.4 mg/100 ml)
and phosphate concentration 13-4 mmol/l (41 mg/100 ml).

The patient was treated with vitamin D and oral phosphate, which were
serially adjusted to heal the rickets and prevent hypercalcaemia and hypo-
phosphataemia (figure). When she was aged 5 years 1α-hydroxy vitamin D3
gather with phosphate supplements. The likelihood exists that phos-
phate will prove equally necessary in the treatment of other forms of
renal hypophosphataemic rickets.

1 Gardiner LI. Endocrine and genetic diseases of childhood and adolescence.
vitamin D resistant rickets with massive doses of 1α-hydroxyvitamin D3
3 Peacock M, Heyburn PJ, Aaron JE. Vitamin D resistant hypophosphata-
emic osteomalacia: treatment with 1α-hydroxyvitamin D3. Clin Endocrinol

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Role of tartrazine in chronic urticaria

Reactions to azo dyes, particularly the yellow dye tartrazine, are often
reported. The proportion of patients with chronic urticaria who
develop an exacerbation after taking tartrazine has ranged from
5 to 36%.1 2 3 Salicylates, usually in the form of aspirin, will cause
similar exacerbations, affecting 20-45% of patients with chronic
urticaria.4 Many patients who have a reaction to salicylate in an active
phase of chronic urticaria can take salicylates again when the urticaria is
clear.

When patients with chronic urticaria are challenged with a battery
of substances, including salicylates and tartrazine, those who react
to one are frequently affected by the other.5 We tested 317 patients:
40 (13%) reacted to tartrazine and 28 of these also reacted to sali-
cylates. The mechanisms of tartrazine and salicylate reactions in
chronic urticaria are probably similar and non-immunological. This
view would be strengthened if it was shown that patients who had
previously developed exacerbations of urticaria owing to tartrazine
no longer reacted once their urticaria was clear.

Patients, methods, and results

From 40 patients with chronic urticaria who had previously reacted
to 10 mg tartrazine we selected 20 who lived near Bristol. Thirteen agreed
to come to hospital for further tests but one defaulted, so that the tests
were carried out in 12 patients (seven women, five men; average age 44 years,
range 17-71). Each patient was given three identical capsules—lactose,
tartrazine 10 mg, and lactose—which were taken on alternate mornings. One
to four weeks later a test dose of 1 mg tartrazine was given interspersed
with two other colours as part of a different investigation.

The development of widespread weals between two and eight hours after
the capsule was taken was recorded as a positive reaction. Five patients who
had been clear of urticaria for one-and-a-half to seven years had no reaction
to 10 mg and 1 mg tartrazine; two who had been clear for two to three years
except for minor episodic attacks had no reaction to 10 mg and 1 mg
tartrazine; and three who had been clear for three to eight weeks showed a
reaction to 10 mg tartrazine but none to 1 mg tartrazine. Two patients still
with active urticaria (present for three and seven years) showed reactions
to both 10 mg and 1 mg tartrazine.

Comment

In the 12 patients tested reactions to tartrazine occurred when the
urticaria was in an active phase or shortly afterwards, and did not
occur when the urticaria had been clear for a long period. The effect
is quantitative since the two patients reacting to 1 mg tartrazine were
the only ones with active urticaria. In another series of 56 patients
with chronic urticaria immediate and 48-hour patch tests were carried
out to 2% tartrazine. These were all negative, including in four
patients who had reacted to oral challenge tests of 10 mg tartrazine.