Letters to the Editor

Botulism

Sir,

Dr P.D. Welsby's condensed review of botulism\(^1\) contained statements that could be misconstrued and do not reflect the experience gained from recent outbreaks.\(^2\)\(^3\) He describes the onset with oculo-bulbar symptoms as the usual presentation 'if they are not found dead', and suggests that gastroenteritic symptoms are notable for their absence. Oculo-bulbar manifestations with a descending paralysis provide the most characteristic presentation. This is true of all types including type B, which is the commonest form of botulism in Europe and was the cause of the hazelnut yoghurt outbreak. The development in some patients of weakness in the lower limbs before that in the arms may help to differentiate the condition from Guillain–Barre syndrome (GBS). However, in approximately 40% of patients more protein parasympathetic symptoms such as blurring of vision, dizziness and hypotension preceded the development of muscular weakness.\(^3\) One-third developed abdominal cramps, vomiting, diarrhoea or constipation. Gastro-intestinal manifestations are a potent source of danger and were the cause of death in the one patient who died in that outbreak. She succumbed to an acid pneumonia following sudden gastric dilatation with regurgitation in the presence of oropharyngeal paralysis. Another patient developed gastric dilatation as his first symptom and was unable to do up his trousers. If the airway is protected and ventilation maintained the mortality from type B botulism should not approach 10%. There was no evidence, even retrospectively, of unexplained deaths that might have been attributed to botulism. However, with types A and E a more rapid progression would have been expected, with the risk of a higher mortality.

The presence of fever\(^2\) or drowsiness\(^2\) does not exclude botulism. Many patients and their doctors interpreted a red, dry throat as tonsillitis. In the early stages a raised protein or cell count in the cerebrospinal fluid may also be absent in GBS. A false-positive edrophonium test can certainly be obtained in botulism.\(^5\)^\(^6\) Positive electromyographic tests showing post-tetanic facilitation can be diagnostic but may be difficult to obtain in the most severely affected.\(^7\)

Antitoxin therapy is not of proven value except in type E intoxication, and carries the added danger of an anaphylactoid reaction from horse serum. If used, it should be given early and should precede the early use of guanidine or 3,4-aminopyridine, which may enhance the uptake of toxin at the neuromuscular junction. Some patients, including children, reported a subjective improvement after its use at a relatively late stage.\(^2\) Antibiotics are best reserved for secondary complications except in the presence of infantile or other toxico-infective forms of botulism.

E.M.R. Critchley
Department of Neurology,
Royal Preston Hospital,
P.O. Box 66,
Sharee Green Lane,
Preston PR2 4HT, Lancashire, UK.

References

Gynaecomastia after long-term administration of domperidone

Sir,

Domperidone, a dopamine antagonist that does not freely cross the blood–brain barrier, has found wide use as an anti-emetic in cancer chemotherapy and as a useful promotility agent in patients with upper gastrointestinal symptoms. The peripheral effects of the drug include an increase in the tone of the gastro-oesophageal sphincter and an increase in the rate of gastric emptying.\(^1\)

Adverse reactions to domperidone are uncommon. However, those that have been reported are consistent with its action as a dopamine antagonist. An oculogyric crisis has been reported in an infant given the drug, and one case of gynaecomastia and galactorrhoea in a male infant has been recorded.\(^2\)^\(^3\)

The anterior pituitary lies outside the blood barrier, and in common with other dopamine antagonists domperidone causes a rise in serum prolactin. There have been no previous reports of clinical adverse effects of dopamine-induced hyperprolactinaemia in adults. We report the first case of gynaecomastia in an adult after long-term administration of domperidone.

The patient, a 63 year old man, presented with a 3-month history of tender left gynaecomastia. There was no history of trauma or alcohol abuse. Twenty years previously he underwent a Pölya gastrectomy for a benign gastric ulcer. One year previously he had a gastroscopy for symptoms of nausea and vomiting and had been started on domperidone 20 mg 3 times a day with symptomatic improvement. His liver function tests, serum gonadotrophins and human chorionic gonadotrophin estimations were all normal. His serum prolactin was raised on 2 occasions one month apart at 894 µmol/l and 609 µmol/l (NR 50–360 µmol/l). X-rays of his pituitary fossa were normal. After withdrawal of the drug his prolactin fell to 88 µmol/l with a resolution of his gynaecomastia. We recommend caution in the long-term administration of domperidone.
usage of domperidone. It must now be added to the long list of causes of drug-induced gynaecomastia.

J.P. Keating
M. Rees
Department of Surgery,
Basingstoke District Hospital,
Aldermaston Road,
Basingstoke, Hampshire, UK.

References

Recurrence of a reactive arthritis following streptokinase therapy

Sir,

We write to report a case of a delayed reaction to streptokinase therapy, probably an immune-complex vasculitis now increasingly associated with the drug, in contrast to the immediate allergic reaction commonly seen.1-3 Similar vasculitic reactions have been reported with anisoylated plasminogen streptokinase activator complex.4

A nulliparous woman of 46 was admitted as an emergency with a diagnosis of myocardial infarction. She gave a history of hypertension for which she had been treated for 8 years with diltiazem, captopril and frusemide. She was treated with streptokinase 1.5 million units intravenously over 1 h. On the 5th in-patient day, she developed a widespread macular rash, predominantly over the limbs; no purpuric element appeared. Simultaneously, she felt considerable pain in the knees, shoulders and elbows, which were hot and stiff symmetrically but with no effusions. She was not febrile, there was no pericardial rub, and dipstick testing of the urine was normal. The arthritis and rash settled spontaneously over two days on ibuprofen. She recovered and was discharged on the 10th day. At the time of the arthritis, blood film showed a leucocyte count of 14 x 10⁹/l, ESR 54 mm/h. Antinuclear factor was negative and rheumatoid factor (RAPA) borderline at 1/80. C3 and C4 186 and 14 mg/100 ml (elevated and normal, respectively). IgG 19.9, IgA 3.9 (both elevated) and IgM 1.1 g/l. Urine protein excretion 0.26 g/24 h.

She subsequently revealed that she had experienced the same reaction, with arthritis affecting the elbows and knees, and a rash, in 1980 when she had had an episode of pneumonia for which she had received parenteral antibiotics. At that time, no pathogen had been identified, but she had taken penicillin subsequently with no ill effect.

This patient’s problem is interesting because of the previous reaction to a pneumonia, and we may surmise that this was again a reaction to a streptococcal compon-ent antigen, although we have no proof that this was so. The reaction would then appear to be a form of reactive arthritis, and this suggests that investigation of future patients should include studies of synovial fluid (if accessible), anti-streptokinase antibodies and lymphocyte and neutrophil responses to streptococcal antigens.

M.P. Kelly*
C. Bielawska
Whittington Hospital,
Highgate Hill,
London N19 5NF, UK.
*Correspondence and present address:
Manze District Hospital,
P.O. Box 660029, Manze, Zambia.

References

Spontaneous pneumomediastinum following myocardial infarction

Sir,

Spontaneous pneumomediastinum is a rare condition that may simulate the features of myocardial infarction in the absence of actual ischaemic heart disease. We report a case of asymptomatic spontaneous pneumomediastinum that followed acute myocardial infarction.

A 52 year old woman presented with a 2-h history of severe retrosternal chest pain associated with dyspnoea and nausea but no vomiting. Clinical examination was unremarkable but the electrocardiogram showed acute anteroseptal myocardial infarction. She was given intravenous streptokinase infusion. A chest radiograph at admission revealed free air within the mediastinum but no evidence of pneumothorax. The patient had no further chest pain or other complications. Acute myocardial infarction was confirmed by elevated serial enzymes. The radiographic appearance resolved over the following week.

Spontaneous pneumomediastinum is caused by non-traumatic rupture of marginal pulmonary alveoli allowing air to travel along interstitial and vascular routes.1 It occurs in situations where there is a sudden increase in intra-alveolar pressure such as severe coughing, straining or Valsalva manoeuvres, and has been associated with acute asthma,2 violent exercise1 and childbirth.3 This is the first reported case of pneumomediastinum following myocardial infarction and the pathogenesis is uncertain. We do not believe that the concurrence of these condi-
Gynaecomastia after long-term administration of domperidone.

J. P. Keating and M. Rees

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