chorea occurs when the function of the corpus striatum is disrupted, the specific abnormality in Sydenham's chorea is unknown. The response to valproic acid suggests that the defect affects the GABA neurotransmitter system. Possibly the difference in effectiveness of valproic acid between Sydenham's chorea and Huntington's chorea reflects the degree of severity of the pathologic process or, alternatively, indicates a difference in the pathogenesis of the two diseases.

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Vibrio alginolyticus peritonitis associated with ambulatory peritoneal dialysis

Vibrio alginolyticus is an unusual human pathogen. It is related to *V parahaemolyticus* and its natural habitat is the sea. Human infections reported to date have been mostly wound and ear infections in swimmers and others with marine contact. We believe that the following is the first reported case of *V alginolyticus* peritonitis.

Case report

A 20-year-old man with end-stage renal failure, who had been receiving treatment for six months with continuous ambulatory peritoneal dialysis, presented with signs and symptoms of peritonitis. Several days previously he had been scuba-diving off the South Australian coast and had changed his peritoneal dialysis fluid on the beach without taking adequate precautions against infection.

He presented with severe abdominal pain, appreciable fever, and signs of peritonism. Samples of macroscopically turbid peritoneal fluid were taken immediately from the Tenckhoff catheter for microscopy and culture. Gram staining showed pus cells but no organisms. Treatment was started with continuous peritoneal lavage with flucloxacillin (50 mg/l) added to the dialysis fluid. His symptoms improved but oxidase-positive, Gram-negative bacilli resistant to flucloxacillin (420 colonies/ml) were cultured from the first sample of dialysis fluid collected. The treatment was changed to gentamicin 4 mg/l as *Pseudomonas* was considered to be the likely infecting organism.

Further identification was carried out using routine biochemical tests. The initial reactions suggested that the organism was possibly a member of the *Bacillus* genus. Since Gram staining showed large Gram-negative rods, however, the organism was resubmitted for appareil procédés d'identification (API) and conventional tests. As the API system indicated that the organism was a vibrio (*?V cholerae*) his history was reviewed and marine vibrio suspected. Further identification procedures showed the organism to be *V alginolyticus*, sensitive to aminoglycosides, co-trimoxazole, cephalothin, and cephalexin but resistant to ampicillin.

He made a satisfactory recovery over this period, and after four days cultures of the peritoneal fluid were negative and the gentamicin was stopped. He was discharged from hospital taking oral cephalexin and remained in good health.

Comment

Peritonitis is the most important complication of ambulatory peritoneal dialysis. The infective organism is usually from the patient's own flora and infection occurs after breakdown of the aseptic technique. In this case the infecting organism, *V alginolyticus*, was of marine origin. It has a worldwide distribution[1] and can be isolated from fish and crustacea.[2] It differs from *V parahaemolyticus* in that it ferments sucrose, is Voges-Proskauer positive, will grow in both 8% and 10% sodium chloride, and swarms on blood agar. An unusual cause of human infection, *V alginolyticus* has usually been associated with skin and ear infections[1-5] acquired in a marine setting. To our knowledge *V alginolyticus* peritonitis has not been reported previously.

Patients with indwelling Tenckhoff catheters, and especially those receiving ambulatory peritoneal dialysis, should be advised of the infective risk of swimming in salt water. When such patients with marine contact develop peritonitis the possibility that the causative organism may be a marine vibrio, such as *V alginolyticus*, should be considered.

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Respiratory and bulbar paralysis with relapsing hyperthyroidism

Bulbar paresis occurring in patients with hyperthyroidism is a well-recognised phenomenon.[1 2] Reports have cited only difficulty in swallowing and aspiration pneumonia as serious complications of the bulbar weakness. We report a case of hyperthyroid bulbar myopathy that required artificial ventilation to support life.

Case report

A 49-year-old Chinese woman presented with intermittent diplopia and difficulty in swallowing of three weeks' duration. Swallowing fluids often resulted in coughing and spluttering. She noticed that she could no longer whistle. One week after the onset of the symptoms ethinyloestradiol (50 μg) daily had been prescribed. In 1958 she had undergone partial thyroidectomy for hyperthyroidism in Hong Kong. This had been repeated in 1968 because of a recurrence of hyperthyroidism.

Examination showed a fit woman with a resting pulse of 96 beats/min and normal blood pressure. There was no thyromegaly or thyroid bruit. Diplopia with weakness of the right inferior rectus muscle was present intermittently. No exophthalmos or lid lag was noted. Definite bilateral facial weakness, poor palatal movement, and bilateral tongue weakness were evident. Mild shoulder girdle weakness was present. Tendon reflexes and sensory testing were entirely normal. Routine haematology, electrolyte concentrations, and liver function tests, including creatine kinase activity, were normal. Electromyography and nerve conduction studies were normal, and an edrophonium test (10 mg) was negative. Barium swallow showed no peristalsis. Direct laryngoscopy showed no movement of the cords. Thyroid function tests showed: thyroxine concentration 237 nmol/l (18·4 μg/100 ml) (normal 70-140 nmol/l; 5·4-10·9 μg/100 ml); unbound thyroxine binding globulin 95% (85-110% of normal pool); and triiodothyronine 2·8 nmol/l (182 ng/100 ml) (normal 1·1-2·6 nmol/l; 72-169 ng/100 ml). A thyroid pertechnetate scan showed increased uptake in the right and pyramidal lobes. The antithyroid (microsomal) antibody titre was 1/6400 and thyroglobulin antibody titre 1/320.

Soon after admission to hospital she required endotracheal intubation and artificial respiration as her vital capacity had fallen below 600 ml, she could no longer swallow, and arterial blood-gas analysis showed oxygen pressure 8·1 kPa (61 mm Hg), carbon dioxide pressure 7·2 kPa (54 mm Hg), and pH 7·33. Ventilation was required for six weeks. At the end of this period bulbar and respiratory muscle power had almost recovered. During her admission treatment consisted of carbimazole 20 mg daily and prednisolone 100 mg on alternate days. Thyroid function (biochemically) had returned to normal two weeks before she could be weaned off the ventilator. At follow-up three months later she was well with no symptoms.

Comment

In patients with hyperthyroidism weakness may be due to a thyrotoxic myopathy, hypokalaemic periodic paralysis[3] (particularly