Thyroid storm presenting as status epilepticus and stroke

Sir, Patients with thyroid storm typically present with hyperthermia, tachycardia, and central nervous system dysfunction such as confusion, lethargy, and psychosis culminating in stupor and coma. Ver (140 beats/min) and stroke is the sole clinical manifestation. To our knowledge, there has been only one report of status epilepticus during the clinical course of thyroid storm.2 We experienced a woman with thyroid storm who initially presented with status epilepticus followed by stroke.

A 56-year-old woman was taken to the emergency room as a result of recurrent generalised seizures which had occurred four times for an hour on the day of admission. After the first seizure at home, she had become confused and lethargic. On arrival at hospital, she was still confused and irritable, and then she experienced her fifth generalised seizure of the day. After this seizure, intravenous sodium valproate (1200 mg/day) was started. The history revealed Graves’ disease, diagnosed three years earlier, although she had discontinued antithyroid medication about two years before. In the past year, she had had a brief episode of frank psychosis. For the most recent three days, she had reported upper respiratory symptoms. Examination revealed a heart rate of 40 beats/min (24195), sweating, and an enlarged thyroid. Neurological evaluation showed confusion, irritability, generalised rigidity, and neck stiffness. Enhanced computed tomography (CT) and cerebrospinal fluid study were normal. Electrocardiographic monitoring documented persistent sinus tachycardia.

Immediately after the brain CT scan, she was found to have right hemiplegia and she became stuporous. The attending physician then managed her as a thyroid storm. Thyroid function test showed free thyroxine 59.6 pmol/l (normal 9–24) and thyroid-stimulating hormone 0.05 mU/l (normal 0.4 to 0.6). An electroencephalogram (EEG) showed diffuse slow waves in the delta and theta range. In the next morning, she developed a high fever that lasted for three days. A T1-weighted magnetic resonance imaging (MRI) of the brain showed a small lacuna in the left frontal subcortical white matter. MR angiography (MRA) showed mild stenosis at the bilateral terminal internal carotid arteries and the left middle cerebral artery.

Ticlopidine was started to prevent ischaemic stroke. On the fourth day, single-photon-emission computed tomography (SPECT) using 99m-Tc-HMPAO showed perfusion defects in the right fronto-parietal and left frontal areas of the brain. The follow-up MRI on the tenth day revealed cerebral infarcts including watershed areas between the right middle and posterior cerebral artery territories, and between the left anterior and middle cerebral artery territories. The follow-up SPECT and MRA showed no changes. However, serial thyroid function tests and the follow-up EEG showed marked abnormalities for stroke risk factors showed no abnormality. In the fifth week, she was discharged home in satisfactory condition.

Having uncontrolled Graves’ disease and a superimposed respiratory infection, our patient with thyroid storm initially presented as status epilepticus. Both status epilepticus and stroke are very rare in thyroid storm.3,4 Possibly, the past history of brief psychosis might also be thyroid storm. We could not completely exclude the possibility that status epilepticus caused thyroid storm in our patient, although theoretically this is almost nil.

Since 1956, seizures secondary to thyrotoxicosis have rarely been reported. Experimentally, in mice, thyroxine has been shown to lower the seizure threshold.5 In the previous report of thyroid storm with status epilepticus, the seizures were not controlled by diazepam, but responded to chlormethazol. In contrast, the seizures in our patient were well controlled with sodium valproate and did not recur after starting antithyroid therapy.

The hemagglutination after enhanced CT scan cannot be sufficiently explained by the initial MRI finding of the lacune. Subsequently, brain SPECT revealed large perfusion defects which were not compatible with the major arterial territories. We suppose that these were related to the metabolic derangement (increased metabolic demand) and aggravated by iotinated contrast dye which precipitates thyroid storm. In a report of thyroid storm with recurrent stroke, despite the lack of the embolic source, the authors suggested thromboembolic events recurring after stopping heparin.1 In contrast, our patient recovered without heparin, and antifibrinolytic vasospasm could be excluded by MRA. However, venous infarction or vasculitis could not be excluded. Hitherto the exact mechanism(s) of ischaemic stroke in thyroid storm remains uncertain.

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4 Seyfried T, Glaser GH, Yu RK. Thyroid hormone influence on the susceptibility of mice to audiogenic seizures. Science 1979; 205: 598 - 600.

Endocarditis caused by Lactobacillus

Sir, In the absence of intravenous substance use, a-haemolytic streptococci remain the most common aetiology of infective endocarditis. A variety of uncommon organisms may also cause endocarditis, however. We report a case of endocarditis due to Lactobacillus. Only a few cases of endocarditis related to this organism have been reported.1-3

An 80-year-old woman with a past history of hypertension and dementia was admitted to our Medical Center with shortness of breath and uncontrolled diabetes. Physical examination revealed an anterograde patient with poorly maintained dentition, a Gr III/VI pansystolic murmur at the apex radiating to the axilla, and bibasilar rales. No peripheral signs of infective endocarditis were present. The chest X-ray revealed mild pulmonary oedema and cardiomegaly. An electrocardiogram showed atrial fibrillation.

On the second hospital day, the patient was noted to have a temperature of 101°F with no change in physical findings. A blood culture drawn on that day and one on the following day were found to be positive for Lactobacillus jensenii. A transoesophageal echocardiogram revealed a large vegetation on the aortic valve with moderate to severe mitral regurgitation and mild to moderate aortic regurgitation. The patient was given ampicillin (2 g intravenously every four hours) and gentamicin (60 mg every eight hours (3 mg/kg daily) for four weeks. Follow-up blood cultures drawn during and after treatment remained sterile. Lactobacillus species are ubiquitous and generally considered non-pathogenic inhabitants of the oral cavity, gastrointestinal tract and female genital tract. Review of 23 reported cases of Lactobacillus endocarditis shows that it typically occurs in patients with pre-existing heart disease and often with some
form of recent dental infection or manipulation. Our patient also had very poor dentition and valvar heart disease. Various combinations of antibiotics like penicillin and streptomycin, cephalosporin and streptomycin, penicillin, tetracycline and streptomycin have been successful in achieving cure, however, at present high dose penicillin (>25 mU/day) in combination with an aminoglycoside for six weeks is favoured.

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3 Griffin JK, Daly JS, Dodge RA. Two cases of endocarditis due to Lactobacillus species; antimicrobial susceptibility, review and discussion of therapy. Clin Infect Dis 1992; 14: 250 – 5.

Emergency blood test guidelines

Sir,
The audit study by AG Pennycook1 resulted in considerable savings (40%) on out-of-hours investigations in the Accident and Emergency department in Southampton. We carried out a similar study here seven years ago but achieved a smaller (22%) reduction in on-call investigations over the first few months only.2 Since then the on-call workload for the laboratory has increased steadily year-on-year, despite instruction of doctors in the Accident and Emergency department on the use of our guidelines. We are now reconsidering the wider use of emergency investigation guidelines and contacted the laboratory in Southampton to ask them about the effect of the guidelines on their workload. They were not aware of the audit study conducted in their Accident and Emergency department and their workload figures had not shown any reduction over the years. A possible explanation is that the reduced requesting in the Accident and Emergency department was compensated for by increased requesting for blood tests on patients admitted to other units in the hospital. We are therefore not yet convinced that significant costs savings can be made for the whole hospital by the use of such guidelines but intend to explore this further.

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Cardiopulmonary resuscitation

Sir,
In his interesting article, Kevin Stewart analyses the ethical and moral principles around the issue of how ‘not for cardiopulmonary resuscitation’ decisions (DNR decisions) are made. We would agree that, in most cases where a decision will be put into effect, they can be made on the grounds of futility, i.e., it is not necessary to offer an ineffective treatment. However, a larger number of patients are admitted to hospital unwell but not with a high likelihood of dying, in whom death is nevertheless a possibility. A decision still needs to be made in case of the unexpected cardiac arrest. In our study we did not find any way of being able to predict the 18% of patients who would not want to be resuscitated without asking them first; in addition, we found that 35% of patients wish to be actively consulted while 51% did not mind. Quality of life from the observer’s perspective does not seem to be a valid predictor of the decision made by the patient. Therefore, it is necessary to discuss DNR decisions with competent patients, irrespective of our view of their potential quality of life, if we wish to comply with their wishes. We do not say that this is easy or even practical at the moment in the UK but if it became routine to ascertain the patients’ view on DNR decisions and admission a lot of the sensitivity around discussion would disappear. This could be done initially by routinely informing patients of the hospitals’ ‘opt out’ or ‘opt in’ cardiopulmonary resuscitation policy in hospital literature, although one suspects that many will not read this.

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Octreotide therapy for diarrhoea

Sir,
Intractable diarrhoea complicates systemic amyloidosis in a significant number of patients. The aetiology is thought to be either autonomic neuropathy or direct infiltration by amyloid of gastrointestinal submu cosa. Therapeutic management is frequently unsatisfactory due to resistance to conventional anti-diarrhoeal agents. Only two case reports exist to date in the literature describing successful symptomatic control with the long-acting somatostatin analogue octreotide acetate.3,4 We report the third.

Case report
An 80-year-old woman had a six-month history of weight loss, anaemia and unremitting diarrhoea, unaccompanied by blood or mucus and resistant to all attempts at conventional treatment with codeine, loperamide and su局phasalazine. Routine biochemical and haematological investigations, stool cultures, gastroscopy with biopsy, abdominal ultrasound and barium enema examination were all negative. Histology from rectal biopsies, however, stained well with Congo red and confirmed a diagnosis of amyloidosis, immunohistochemistry demonstrating a monoclonal immunoglobulin light chain (AL type). Institution of octreotide therapy 100 μg subcutaneously three times daily resulted in immediate cessation of her diarrhoea. Transfer to another hospital unfortunately led to octreotide being discontinued with subsequent recurrence of diarrhoea, but re-challenge with the drug again achieved immediate symptomatic control.

This case represents the third reported where octreotide re-challenge resulted in immediate complete symptomatic resolution in a patient with hyposecretion diarrhoea due to amyloidosis. Successful anti-diarrhoeal action has also been described with octreotide in patients with familial amyloidotic polyneuropathy.3 Hypersecretory diarrhoea in amyloidosis is an unlicensed indication for octreotide use, but further similar reports may strengthen the case for more generalised use of this agent in a distressing condition affecting predominantly elderly patients.

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Oxytocteide: clinical indications
- bleeding peptic ulcer
- bleeding oesophageal varices
- gastrointestinal fistula
- pancreatic fistula
- acute pancreatitis
- short bowel syndrome
- ileostomy diarrhoea
- diabetic diarrhoea
- chronic secretory diarrhoea (idiopathic, HIV)
- secretory tumours: pituitary adenomas, gastroinomas, insulinomas, vipomas, carcinoid syndrome

Oxytocteide: modes of action
- reduces splanchic, portal and mucosal blood flow
- inhibits endocrine and exocrine secretions from somatostatin-containing cells in pancreas, stomach and intestines
- stimulates water and electrolyte absorption from gastrointestinal tract
- inhibits gallbladder motility and secretion
- slows gastric emptying and reduces peristalsis in gastrointestinal tract
- inhibits hypothalamic–pituitary hormonal release
- inhibits gastrointestinal tract tumour growth
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doi: 10.1136/pgmj.73.855.61-a

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