Comment

A previous report of two similar cases of urticaria controlled by warfarin did not include a placebo challenge.¹ Our case differed from those cases in that we had to keep the prothrombin time within the therapeutic range.

The mechanism of this response to warfarin is obscure, but there may be a link between urticaria and the coagulation system.^{2,5} Ryan proposed that plasmin may contribute to urticaria through activating the complement cascade, activating kallikrein, and producing degradation products of fibrin which also promote inflammation.³ There are also several reports of chronic urticaria and angio-oedema responding to the plasmin inhibitors tranexamic acid^{3,4} and aprotinin.⁵ Warfarin could affect plasmin activity in two ways: firstly, by reducing the formation of fibrin

and, secondly, by reducing protein C concentrations. Reduced formation of fibrin may also be important in itself as fibrin may perpetuate inflammation in chronic urticaria.²

We suggest that further investigation of the role of the coagulation system and the use of warfarin in chronic urticaria would be of value.

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Anaesthetic complications of a weight reducing regimen

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Hypokalaemia during anaesthesia may cause cardiovascular instability and arrhythmias and adversely affect muscle activity. I report on a 16 year old girl who required emergency appendectomy but was found to be hypokalaemic as a result of a weight reducing regimen that included thiazide diuretics.

Case report

A 16 year old girl was admitted with acute appendicitis. She did not have a history of diarrhoea or vomiting. General questioning elicited that eight months before admission, and without her general practitioner's knowledge she had started taking a strict reducing diet and treatment (phentermine 15 mg daily and an unlabelled white tablet for "water") prescribed by a private clinic. Her weight had decreased from 69 kg to 53 kg in two months but returned to 69 kg after school examinations and parties caused a break in the regimen. Two weeks before admission she had restarted the regimen, and on admission her weight was 59 kg.

Routine blood investigations showed hypokalaemia (serum potassium concentration 2.9 mmol/l; normal range 3.5-5.0), and operation was deferred owing to the possibility that the anaesthetic might precipitate a dangerous cardiac arrhythmia. To improve the serum potassium concentration in the short term 27 mmol potassium chloride was infused intravenously over 90 minutes under electrocardiographic monitoring, and this raised her serum potassium concentration to 3.5 mmol/l. A further 13.5 mmol was given before anaesthesia, and 5 mmol potassium in one litre of compound sodium lactate intravenous solution was given during the operation.

Anaesthesia was induced with thiopentone sodium; maintained with nitrous oxide, oxygen, and isoflurane; and supplemented with fentanyl. Muscle relaxation was by suxamethonium chloride and vecuronium. Anaesthesia was uneventful, no cardiac arrhythmias being recorded during continuous monitoring.

The serum potassium concentration two hours after operation was 3.2 mmol/l. A further 27 mmol potassium chloride was infused over eight hours, after which the serum concentration was 3.1 mmol/l. Intravenous supplements were continued until oral potassium salts could be started. Before discharge a

serum potassium concentration of 4.2 mmol/l was maintained without supplementation, indicating that most of the total potassium in the body had been restored.

Comment

Patients with hypokalaemia may require an increased dose of depolarising muscle relaxant during anaesthesia,¹ while the action of non-depolarising relaxants is enhanced. Reversal with neostigmine is less predictable,^{1,2} and the risk of inadequate reversal or recurarisation is compounded in the out of hours emergencies. Intermittent positive pressure ventilation during anaesthesia increases intrathoracic pressure and causes a drop in blood pressure, which normally recovers with the resumption of spontaneous breathing. In hypokalaemic patients, however, this return to normal may not occur. The patients are also more likely to develop and less able to deal with postoperative hypotension.³

In hypokalaemia the myocardium is thought to become more susceptible to arrhythmias owing to increases in automaticity, conductivity, and excitability. It is unclear at what degree of hypokalaemia this becomes important, and the only prospective study in humans shows no evidence of chronic hypokalaemia predisposing to arrhythmias during operation.⁴ Hypokalaemia may, however, increase the mortality associated with a serious arrhythmia.⁵

This patient's own doctor was not informed of her self referral to an obesity clinic or of her drug treatment, and so he was unable to supply this information to the hospital. No blood tests had been done by the clinic. Drugs were dispensed unlabelled, so possible drug interactions with anaesthetic agents and with antibiotics could only be guessed.

Diuretics cause hypokalaemia and should not be used without regular monitoring of serum potassium concentrations. Communication between members of the medical profession is important, particularly on the part of doctors who provide treatment in clinics to which patients refer themselves.

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