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Elevation of serum cholesterol and triglycerides as a complication of amiodarone therapy

Sir,

I would like to report the case of a 62-year-old man who was started on amiodarone for lone atrial fibrillation in October 1983. Prior to this the fibrillation had not been controlled by propranolol hydrochloride or sotalol hydrochloride. The fibrillation was quickly brought under control with amiodarone 200 mg daily. All possible causes of episodic fibrillation had been excluded before starting any therapy.

In February 1988 he remained free of attacks and attended for routine check up. It was decided to include him in a check of cholesterol and triglyceride levels in those with cardiac problems. His cholesterol level was found to be 8.6 mM and triglyceride level 4.1 mM. His high density lipoprotein cholesterol level was 1.4 mM and cholesterol: HDL cholesterol ratio was 6.4. A check on his diet showed that in virtually all respects it was exemplary. Both his parents were alive and in their nineties. Perusal of his case notes showed that in April 1987 his cholesterol and triglyceride levels were normal. After consultant referral it was decided to reduce the amiodarone dosage to 100 mg daily and add flecainide acetate 50 mg twice daily. In addition he was advised to tighten up on his diet still further despite his weight being satisfactory.

By July 1988 there was a significant reduction in levels and in December 1988 cholesterol and triglyceride levels were normal at 6.2 mM and 2.2 mM, respectively. Up to the time of writing there has been no recurrence of atrial fibrillation. The patient has lost weight (between four and seven pounds) with minimal alteration in diet.

In March 1988 the patient's amiodarone level was 1.1 mg l^{-1} (range 0.6–2.5) and desethylamiodarone level 1.1 mg l^{-1} (range 0.6–2.5). In September 1988 his amiodarone level was 0.4 mg l^{-1} and flecainide level 224 mg l^{-1}.

Perusal of the literature reveals that this side effect of amiodarone has been reported on several occasions.[1,2] Patients commencing treatment with amiodarone should have their cholesterol and triglyceride levels checked before commencing treatment and then at regular intervals. It seems reasonable to assume that the elevation of lipid levels with this form of therapy is atherogenic.

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Dangers of steam inhalations: case report

Sir,

The patient, a busy mother, was in her second day of an upper respiratory tract infection. She was particularly troubled by a blocked nose and decided to make herself a menthol inhalation. She placed some water in a large mug and added a blob of Vick's mentholated preparation and placed it in her microwave to heat for a couple of minutes.

With her seven month old child balanced on her left hip she removed the bowl of mentholated water with her right hand and noticed that there was a skin of Vick on the surface. She immediately reached for a metal teaspoon to stir the Vick into the hot water while lowering her face to inhale the steam. The contents exploded into her face. Fortunately, the child was unharmed but the mother received first degree burns of her neck, lower jaw, right zygoma and forehand. She saw the contents bursting out and thinks she closed her eyes as a reflex response. Her burns have taken three weeks to settle.

Mentholated steam inhalations are widely advocated for upper respiratory tract infections. With the increasing use of microwaves in homes it is to be expected that they will be used for quick preparation of such inhalations. Although the mechanism of this reaction is not clear this case serves as a reminder of the danger of such procedures.

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Referral of women with chronic pelvic pain

Sir,

Dr Guirgis' letter (December 1988 *Journal*, p.567) highlights the lack of knowledge about the aetiology and management of chronic pelvic pain in women. He describes well the ritual of investigations to which these women are subjected and points to the considerable waste of resources and the adverse psychological effects on women who show no improvement and questions whether all the investigations are justified. While this is a valid question it is not possible to answer until the aetiology of the pain is known in the large number of women who have no obvious cause for it at present. While our work[1] confirms the limitations of laparoscopy as anything but a means of excluding visible gynaecological pathology, I cannot agree with his implication that there is a 'psychogenic' rather than 'organic' cause for chronic pelvic pain.

All our work[2] suggests that vascular congestion associated with ovarian dysfunction, detectable on pelvic venography and ultrasound scanning is the cause of the pain. It is true that psychotherapy results in a significant improvement in the severity and frequency of attacks of pelvic pain[3] but that is true of all forms of pain elsewhere in the body. We have recently completed a randomized control trial of medroxyprogesterone acetate (50 mg a day) given orally continuously for four months, designed to partially supress ovarian function. This treatment has resulted in a significant reduction in pain among women with pain resulting from pelvic congestion despite a strong placebo effect.[4]

There is a basic flaw in the logic of modern medical thinking which assumes that if an organic cause for pain is not revealed by modern methods of investigation, the likely cause for the pain is 'psychological'. Such a conclusion is the refuge of those who find it difficult to tell their patients that they do not know the cause of pain, and seriously damages the self-esteem of the patient. Hopefully, in time, pelvic congestion will come to be accepted as a form of pathology that may well have its origins in emotional disturbance but which is eminently treatable.

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