

tions was mere coincidence, however. While the patient gave no history of severe vomiting or coughing and cardiopulmonary resuscitation had not been performed, it is entirely credible that the pain of her myocardial infarction led to a Valsalva manoeuvre that produced spontaneous pneumomediastinum.

Most patients with spontaneous pneumomediastinum complain of dyspnoea and chest pain while a quarter are asymptomatic. The chest pain is usually of acute onset and can closely mimic myocardial infarction. Furthermore, electrocardiographic abnormalities including ST segment elevation have been reported in patients with spontaneous pneumomediastinum without other evidence of ischaemic heart disease.⁵ Although raised cardiac enzymes subsequently confirmed myocardial infarction in this patient the concurrence of spontaneous pneumomediastinum highlights the importance of correct clinical interpretation of acute chest pain in such a case.

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Non-sustained ventricular tachycardia following clonidine withdrawal

Sir,
Abrupt discontinuation of various antihypertensive agents including clonidine, guanethidine, alpha methyl-dopa and propranolol may lead to a constellation of symptoms resembling pheochromocytoma crisis due to a rebound hyperadrenergic state.¹ We recently encountered a case of sudden clonidine withdrawal presenting with the distinctly unusual manifestation of a recurrent non-sustained ventricular tachycardia (VT).

A 46 year old male presented in the emergency room with a history of repeated spells of palpitations and dizziness for 9 h prior to entry. The patient was a known hypertensive for the last 3 years and was on 0.6 mg of clonidine in divided doses for the last 8 months. He had stopped clonidine about 24 h prior to the onset of symptoms. There was no previous history of ischaemic

heart disease or cardiac arrhythmia, and a treadmill test and echocardiogram done earlier were normal. The patient was seen by another doctor 3 h prior to admission; he was found to have a blood pressure of 180/120 mmHg and was prescribed 10 mg nifedipine orally. On examination, pulse rate was 130 per min with 15-20 ectopics per min and blood pressure was 130/90 mmHg. Initial electrocardiogram revealed multifocal ventricular ectopics and short runs of VT. There was no evidence of myocardial ischaemia on electrocardiogram. The patient was treated initially with 75 mg lignocaine bolus, repeated after 10 min with no effect. Administration of lignocaine infusion produced no significant reduction in ventricular ectopics. Lignocaine was discontinued and 200 mg of labetalol administered orally. A significant reduction in the frequency of ventricular premature contractions was observed within the next hour. An additional dose of 100 mg of labetalol was then given. Two hours after oral labetalol, ectopic activity had totally disappeared and the patient was subsequently free of symptoms. A treadmill test and Holter studies done after 3 weeks did not reveal any abnormality.

Various case reports and prospective trials of abrupt antihypertensive drug withdrawal have been reviewed elsewhere.¹ Clonidine discontinuation syndrome is usually seen within 24-48 h after abrupt cessation of the drug. It is characterized by nervousness, restlessness, anxiety, palpitations, diaphoresis, insomnia, tremors, tachycardia and rebound hypertension. Exacerbation of angina, acute myocardial infarction, hypertensive encephalopathy and sudden deaths have also been reported. Twenty-four to 72 h after withdrawal of clonidine, there is an increase in plasma noradrenaline and urinary catecholamine levels.² The subjective symptoms are observed to be more frequent in patients on higher doses of the drug (> 1 mg/day), in patients on chronic therapy with other antihypertensive drugs² and in withdrawal after prolonged clonidine therapy.³ Goldberg⁴ studied discontinuation syndrome in 9 hypertensive patients on clonidine in a prospective trial. Five out of 9 subjects showed withdrawal symptoms and manifestations of atrial ectopic activity. One patient showed ventricular premature contractions and non-sustained VT.

A hyperadrenergic state of any aetiology may lead to ventricular arrhythmia in patients with ischaemic heart disease or mitral valve prolapse syndrome, and also in individuals with a normal heart.⁵ VT in this patient was possibly due to the hyperadrenergic state observed with abrupt clonidine withdrawal. A failure of lignocaine and prompt control of VT with labetalol further consolidates this observation.

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Anaphylaxis to mustard

Sir,

Mustard has been implicated in very few cases of severe allergic reactions in spite of its widespread use. To our knowledge, just two cases of IgE-mediated anaphylaxis due to mustard have been reported in which serum specific IgE against mustard was demonstrated.^{1,2} Two cases of systemic anaphylaxis after ingestion of mustard, confirmed by *in vitro* methods, are presented.

Case 1 A 47 year old women suffering from seasonal rhinoconjunctivitis since childhood developed 3 episodes of acute severe urticaria and facial angioedema accompanied by nausea, vomiting, dyspnoea, wheezing, chest tightness and hoarseness shortly after ingestion of vegetable sandwiches with mayonnaise and mustard. Prick skin tests with grass and olive pollen were positive. Total serum IgE value was 170 kU/l. Serum specific IgE against mustard gave a strong positive value of 23.6 kU/l (radioallergosorbent test class 4; Phadebas RAST units, Pharmacia Diagnostics). Oral challenges with eggs, olive oil, vinegar, lettuce and tomatoes were negative. A non-specific bronchial challenge with methacholine was done with a negative result, which demonstrates the absence of bronchial hyperreactivity in her basal situation.

Case 2 A 15 year old female with a past history of rhinoconjunctivitis and asthma after exposure to grass pollens presented an episode of generalized urticaria, facial and throat swelling followed by chest tightness, 45 min after ingestion of a vegetable salad containing mustard. Total serum IgE was 536 kU/l. Serum specific IgE against mustard was positive at 18.5 kU/l (radioallergosorbent test class 4). Oral challenges with the rest of the foods ingested that day were negative.

The high antigenic potency of mustard is well known,³ although cases of clinical manifestations are scarce. Our patients have histories of pollen allergy, which were not present in the previously reported patients. Some authors suggest the presence of similar antigenic determinants in grass pollen and mustard seeds⁴ while others think that a similar lecithin activity could be responsible.⁵ More studies are required to clear up this point.

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