metastases. She was aware of her diagnosis but declined further treatment and was discharged home.

Comment

This patient had clear evidence of ectopic adrenocorticotrophic hormone syndrome secondary to oat-cell carcinoma of lung. In association with this and as an initial feature she had a spontaneous cure of lifelong psoriasis. We think that these two events were related and can find no other reports of this unusual phenomenon. Just as the onset of certain skin lesions may herald an underlying neoplasm² before it is symptomatic, so may the disappearance of one, as in our case.

We are grateful to Mr W C Barnsley for permission to report this case.

¹ Ayvazian LF. Extrapulmonary manifestations of tumours of the lung. *Postgrad Med* 1978;**63**:93-9.

² Haynes HA, Curth HO. Cutaneous manifestations associated with malignant internal disease. In: Fitzpatrick TB, Eisen AZ, Wolff K, Freedberg IM, Austen KF, eds. *Dermatology in general medicine*. New York: McGraw-Hill, 1979:1343-52.

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Carbon monoxide poisoning secondary to inhaling methylene chloride

Carbon monoxide poisoning usually results from inhaling fumes from engine exhausts, poorly ventilated heating devices, or gas refrigerators. With the advent of natural gas, which is free of carbon monoxide, the risk of accidental poisoning has decreased. We report a case of severe carbon monoxide intoxication caused by inhaling methylene chloride from a commercial paint remover.

Case report

A 20-year-old art student developed nausea and severe, throbbing headache while using a commercial paint remover in a poorly ventilated, unheated room. About an hour later she left the room, feeling dizzy and unwell, and shortly afterwards lost consciousness. She was admitted to the casualty department conscious but disorientated in time and space. She was distinctly flushed with cherry-red colouring of skin and mucosae. Radial pulse was 98/min, and blood pressure 120/70 mm Hg. Cardiovascular system, respiratory system, and abdomen were normal. Haemoglobin concentration was 13.7 g/dl, and white blood cell count $7.3 \times 10^9/l$ ($7300/mm^3$; normal differential). Carboxyhaemoglobin concentration on admission was 50%. Chest radiograph and electrocardiogram were normal.

This patient's symptoms were surprisingly mild for the strikingly high carboxyhaemoglobin concentrations. She was treated with 60% oxygen 4l/min, and after 12 hours the carboxyhaemoglobin concentration was 20%. She was discharged from hospital on the third day.

Comment

Using a paint remover containing methylene chloride in an enclosed room may lead to a large amount of the solvent being absorbed with prompt metabolism to carbon monoxide and a consequent increase in blood carboxyhaemoglobin concentration.¹⁻³ Methylene chloride is released slowly from body tissues, resulting in carbon monoxide remaining in the circulation for a prolonged period.⁴ Hence the carboxyhaemoglobin concentration after inhaling methylene chloride remains high longer than after inhaling carbon monoxide.

Carbon monoxide poisoning is a serious hazard to patients with coronary artery disease, since only marginal increments in carboxyhaemoglobin concentration necessitate greatly increased coronary

blood flow to prevent ischaemia.⁵ Though our patient responded to 60% oxygen, higher concentrations (ideally 100%) are mandatory, especially in a patient with coronary artery disease.

Methylene chloride is present in several paint removers, and any patient with coronary artery disease likely to be exposed to this agent should be warned.

¹ Kubic VL, Anders MW, Engel CH, Barlow T, Cauthey WS. Metabolism of dihalomethanes to carbon monoxide. *Drug Metab Dispos* 1974;**2**:53-7.

² Ratney RS, Wegman DH, Elkins HB. In vivo conversion of methylene chloride to carbon monoxide. *Arch Environ Health* 1974;**28**:223-8.

³ Stewart RD, Fisher TN, Hosko MJ, Patterson JE, Baretta ED, Dodd HC. Experimental human exposure to methylene chloride. *Arch Environ Health* 1972;**25**:342-8.

⁴ Stewart RD, Habe CL. Paint remover hazard. *JAMA* 1976;**235**:398-401.

⁵ Scharf SM, Thames MD, Sargent RK. Transmural myocardial infarction after exposure to carbon monoxide in coronary artery disease. *N Engl J Med* 1974;**291**:85-6.

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Value of routine chest radiography of psychiatric patients

Chest radiography is often performed routinely on patients admitted to psychiatric wards; at one time it was mandatory. A chest radiograph costs about £12.50 (May 1980), and our radiology department suggested that reducing "routine" requests for radiography need not prejudice patient care. Some consultant psychiatrists, however, maintained that chest films were necessary to exclude unsuspected physical disease that might be responsible for psychiatric symptoms. We therefore decided to survey the results of chest radiography of patients admitted to our 60 acute psychiatric beds to find how often unexpected disease was reported.

Method and results

We examined the daily records of the x-ray department for the names of acute psychiatric inpatients who had had chest radiography during 21 May 1979 to 31 March 1980. If an abnormality had been reported we referred to the request form for the patient's age and clinical details, and film and report were then reviewed by a consultant radiologist.

The 746 admissions during the period generated 231 chest x-ray examinations. Twenty-one of these (9%) were reported as showing a "significant" abnormality (broadly defined by us as warranting further investigation or treatment)—namely, active tuberculosis (two cases), other infective changes (seven), abnormal masses (five), cardiac enlargement (five), generalised emphysema (one), and fractured ribs (one). Sixteen of the 21 patients had chest symptoms or signs mentioned on the request card, two had confusional states, and five had a diagnosis of alcoholism. None had "functional" psychiatric conditions without physical symptoms or signs. (A table giving details of these 21 cases is available on request.) Twenty-five other reports mentioned abnormalities of no current clinical relevance, mostly old inactive tuberculosis.

Comment

No chest radiograph disclosed an abnormality entirely unsuspected clinically. Most patients with significant x-ray abnormalities had chest symptoms or a diagnosis of alcoholism. Alcoholism is well known for its association with physical disease, and the two cases of active tuberculosis were in alcoholics. Routine chest x-ray examination may be thought necessary before electric convulsion therapy, for medico-legal reasons, though Abramczuk and Rose¹ found that it did not add appreciably to information obtained from the history and physical examination.

We conclude that chest radiography need not be performed on

patients with functional psychiatric illness in the absence of clinical evidence of physical disease or alcoholism.

We are grateful to Dr P B Guyer and Dr K C Dewbury for help in reviewing the chest radiographs, and Mrs H Hills for helping to collect data.

The table giving details of the 21 patients with relevant radiographic abnormalities may be obtained from Dr Jennifer Hughes.

¹ Abramczuk JA, Rose NM. Pre-anaesthetic assessment and the prevention of post-ECT morbidity. *Br J Psychiatry* 1979;134:582-7.

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Adverse interaction between nifedipine and β -blockade

The calcium antagonist drug nifedipine is hypotensive and produces its systemic effect by reducing systemic vascular resistance.¹ It is also effective in treating angina pectoris, whether caused by coronary artery spasm or by effort.²⁻⁴ β -Blockade is a well-established treatment for hypertension and for angina. In combination with propranolol, nifedipine reduces ST-segment deviations in monitored patients with exertional angina.³ A combination of β -blockade and nifedipine should therefore be especially effective in treating co-existing hypertension and angina.⁴ We studied 15 patients with stable effort angina and hypertension who were treated with nifedipine in addition to pre-existing treatment with β -blockade and a diuretic. One patient developed excessive hypotension.

Patients, methods, and results

Fifteen patients with established hypertension and stable exertional angina were controlled by a standard dose of the cardioselective β -antagonist atenolol (50-100 mg/day) together with diuretic treatment and prazosin (average total dose 9 mg/day in eight patients) as well as intermittent sublingual nitroglycerin as required. The patients had been attending the hypertension clinic for several months or years. After an initial run-in period of six weeks control blood pressure readings were done in the sitting, standing, and lying positions; after walking exercise; and after hand-grip and cold-immersion tests. Nifedipine placebo (1 capsule twice daily) was then added for six weeks and the blood pressure readings were repeated at two-weekly intervals. Then active nifedipine (10 mg twice daily) was substituted for nifedipine placebo for a further six weeks. Finally, active nifedipine was replaced by nifedipine placebo and the readings were again repeated after one month.

Nifedipine was an active hypotensive agent, even in the presence of β -blockade, and was superior to placebo in reducing the sitting, standing, and post-exercise blood pressures as well as the cold-pressor response. Mean ($\pm$ SE) sitting pressures (mm Hg) were as follows. Initial therapy, $162 \pm 7/108 \pm 4$; same therapy + nifedipine placebo, $165 \pm 7/102 \pm 3$; same therapy + active nifedipine, $148 \pm 6/94 \pm 3$; same + placebo nifedipine, $164 \pm 7/105 \pm 4$ ($p < 0.005$ for all comparisons between active nifedipine and placebo). Side effects included headaches and dizziness as well as dilatation of varicose veins in two patients. The addition of nifedipine did not change the severity of angina, as judged by a subjective scoring system.

In one patient (figure) the blood pressure on active nifedipine and β -blockade progressively dropped down to sitting levels of only 90/60 with post-exercise levels of only 80/60. The patient felt extremely weak but his angina was no worse. The atenolol was stopped, his pulse rate and blood pressure rose, and a period of severe unstable angina with persistent chest pain at rest for 24 hours developed while he was taking nifedipine alone. There were no enzymatic changes of myocardial infarction. Stopping nifedipine and restarting β -blockade had little effect on the blood pressure but rapidly relieved the angina. Throughout the observations the patient received no other drugs except cyclopethiazide with potassium chloride one tablet daily and allopurinol 300 mg thrice daily.

Comment

Nifedipine was an active hypotensive agent even when added to β -blockade in these patients. It did little to improve angina, but effort tolerance rose. In one patient the combination of nifedipine and β -blockade progressively lowered the blood pressure over one month

Excessive hypotension produced in hypertensive patient by interaction between nifedipine and beta-blocker.

to such low levels that the patient became extremely weak and unable to exercise. His angina, however, was unchanged. When the atenolol was stopped unstable angina developed, which was relieved with the reintroduction of β -blockade and the cessation of nifedipine. Thus nifedipine was unable to treat the angina of β -blockade withdrawal. The possible advantages of nifedipine alone as an anti-anginal and anti-hypertensive agent in patients with hypertension and angina must still be assessed. When given to such patients who are already receiving β -blockade our results show that (a) it is the hypotensive rather than the anti-anginal effects of nifedipine that predominate; and (b) if excess hypotension develops it is the nifedipine and not the β -blockade that should be stopped.

¹ Olivari MT, Bartorelli C, Polese A, Fiorentini C, Moruzzi P, Guazzi MD. Treatment of hypertension with nifedipine, a calcium antagonistic agent. *Circulation* 1979;59:1056-62.

² Endo M, Kanda I, Hosada S, Hayashi H, Hirokawa K, Konno S. Prinzmetal's variant form of angina pectoris. Re-evaluation of mechanisms. *Circulation* 1975;52:33-7.

³ Oakley GDG, Fox KM, Dargie HJ, Selwyn AP. Objective assessment of treatment in severe angina. *Br Med J* 1979;i:1540.

⁴ Lynch P, Dargie H, Krikler S, Krikler D. Objective assessment of anti-anginal treatment: a double-blind comparison of propranolol, nifedipine, and their combination. *Br Med J* 1980;280:184-7.

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Corrections

Haemangioma of the cord: a further cause of raised maternal serum and liquor alpha-fetoprotein

An error occurred in this paper by Dr A J Barson and others (8 November, p 1252). Maternal serum alpha-fetoprotein concentrations should have been expressed as ng/ml not μ g/ml. The fourth and fifth sentences of the case report should thus have read: "The maternal serum α -fetoprotein concentration at 16 weeks' gestation was raised at 65 ng/ml . . . At 19 weeks' gestation it was 165 ng/ml. . ."

Comparison of nicotine chewing-gum and psychological treatments for dependent smokers

We regret that an error occurred in this paper by Mr Martin Raw and others (16 August, p 481). In table I the figures in the last two rows in the column headed "Nicotine gum" should have read "30(43)" and "35(51)".