

tory nerve endings were larger than usual, particularly on the right. The whole of the bone of the cribriform plate was paper thin and mobile. The defect was closed with a fascia lata patch obtained from the thigh. Post-operative recovery was uneventful. Histology of the olfactory bulb showed chronic inflammatory change and gliosis.

Comments

Cocaine is a psychologically addictive drug and its abuse is greatly increasing.³ It is derived from the coca leaf and is used as a local anaesthetic in otolaryngology because of its useful vasoconstrictor activity. The mechanism of this effect is by inhibiting reuptake of catecholamines into the sympathetic nerve ending, thus potentiating their effect.⁴ Chronic abuse of the drug by intranasal sniffing may lead to chronic inflammatory changes and perforation of the nasal septum, owing to the effects of intense vasoconstriction.⁵ Similar chronic damage probably resulted in the changes in the cribriform plate and olfactory nerves in our patient, which ultimately lead to a leak of cerebrospinal fluid.

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- ¹ Shugar JMA, Som PM, Eisman W, Biller HF. Non traumatic cerebrospinal fluid rhinorrhoea. *Laryngoscope* 1981;**91**:114-21.
- ² Ommaya AK, DiChiro G, Baldwin M, Pennybaker JB. Non-traumatic cerebro-spinal fluid rhinorrhoea. *J Neurol Neurosurg Psychiatry* 1968;**31**:214-55.
- ³ Smart RG, Liban C, Brown G. Cocaine use among adults and students. *Can J Public Health* 1981;**72**:433-8.
- ⁴ Pearman K. Cocaine: a review. *J Laryngol Otol* 1979;**93**:1191-9.
- ⁵ Owens WD. Signs and symptoms presented by those addicted to cocaine. *JAMA* 1912;**58**:329-30.

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Phaeochromocytoma: an unusual cause of chest pain

High catecholamine concentrations due to a phaeochromocytoma are known to cause myocarditis and myocardial fibrosis.¹ Electrocardiographic findings suggestive of myocardial infarction with minimal serum enzyme changes have been attributed to a toxic myocarditis in view of the rapid reversibility of these changes after treatment with adrenergic blocking agents.²⁻³ We describe a patient who presented with myocardial infarction, hypertension, and, later, anginal pain. We suggest that coronary artery spasm was the underlying cause due to the high catecholamine concentrations.

Case report

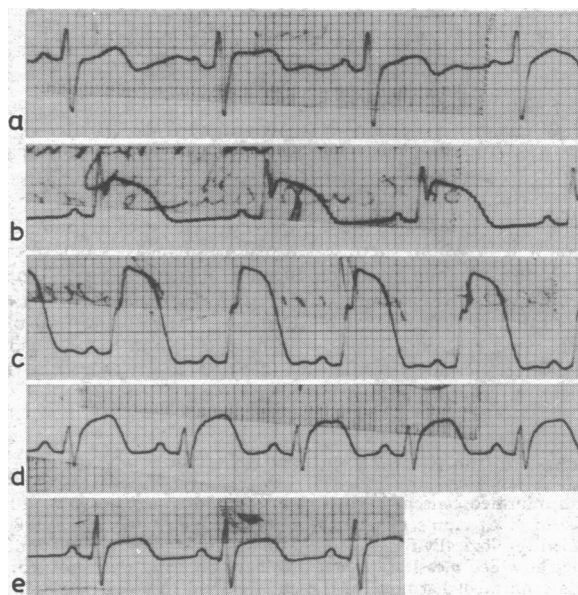
A 38 year old housewife was admitted to hospital in March 1976 because of severe central chest pain. Pulse was 80/min regular and blood pressure 190/140 mm Hg. An electrocardiogram showed appreciable ST segment elevation in the anteroseptal and inferior leads. Twenty four hours after admission serum creatine kinase activity was 1500 IU/l (normal 0-130) and serum lactic dehydrogenase activity 1721 IU/l (normal 115-465).

During the first 24 hours she developed several episodes of severe central chest pain. Monitor printouts (figure) showed considerable variable ST segment elevation. After recovery from the myocardial infarction she was discharged taking propranolol and triamterene, blood pressure being 140/100 mm Hg. One month later the blood pressure was 190/135 mm Hg. After a phentolamine test the blood pressure became normal. Twenty four hour urinary catecholamine excretion was normal. Anginal pain was then

noted, and she was referred for further investigation. Catecholamines in a 24 hour urine collection were raised. This was discounted as it was thought to be due to a drug interaction.

During 1978 she had three admissions to hospital with severe central chest pain and one episode of paraesthesia in the left arm and hand. In 1979, when taking prazosin, acebutolol, and triamterene, she underwent an uneventful cholecystectomy for gall stones.

In 1982, after further anginal episodes, she was readmitted. She was taking prazosin, acebutolol, and hydrochlorothiazide, and her blood pressure was 120/70 mm Hg. Left ventricular angiography showed a discrete apical aneurysm with clot in the aneurysmal sac. The coronary arteries were normal. Three measurements of 24 hour urinary catecholamine excretion were 2.6, 2.9, and 7.03 μmol (normal <0.65 $\mu\text{mol}/24$ h). Computed tomography of the adrenals showed a lesion in the left adrenal measuring 4x4 cm and lying behind the pancreas in front of the renal sinus. Renal arteriography showed the lesion to be lying on the anterior surface of the left kidney, and appearances were suggestive of a phaeochromocytoma. The tumour was excised under alpha-blockade and confirmed pathologically as a phaeochromocytoma. She made an uneventful recovery and was discharged taking warfarin because of the apical aneurysm; blood pressure was 120/80 mm Hg without antihypertensive treatment. Six weeks post-operatively she had no chest pain.



Changes in ST segments associated with chest pain (monitor lead). (a) 3 15 am on 31 March 1976; blood pressure 210/150 mm Hg; no pain. (b) 4 00 am; blood pressure 170/130 mm Hg; severe chest pain with appreciable ST segment elevation; intravenous diamorphine given. (c) 4 30 am; massive ST segment elevation; (d) 8 00 am; ST segment elevation settling; (e) 12 noon; ST segment elevation similar to (a).

Comment

Peripheral vascular ischaemia severe enough to cause gangrene has been described in association with phaeochromocytoma.³ Transient cortical blindness and transient neurological defects have been described,³ but so far as we are aware coronary artery spasm has not been reported in patients with a phaeochromocytoma. Hausmann and Getzowa⁴ were the first to describe myocardial degenerative and fibrotic lesions associated with tumours of the adrenal medulla, which were probably phaeochromocytomas. Van Vliet *et al* described "active catecholamine myocarditis" in a high proportion of patients with phaeochromocytomas proved at necropsy.¹ The cardiomyopathy associated with phaeochromocytoma subsided clinically after resection of the tumour.¹ Electrocardiographic changes of myocardial infarction have been shown in patients with phaeochromocytoma, but subsequent necropsies have shown only severe myocarditis or diffuse myocardial damage.²

In this patient, who presented with a myocardial infarction and severe hypertension, the cause of the myocardial infarction and subsequent angina was probably severe coronary artery spasm as shown by appreciable variation in ST segments. Subsequently, a left ventricular apical aneurysm with normal coronary arteries were shown. Symptoms ceased on removal of the left adrenal phaeochromocytoma.

- ¹ Van Vliet PD, Burchell HB, Titus JL. Focal myocarditis associated with pheochromocytoma. *N Engl J Med* 1966;**274**:1102-8.
- ² Pelkonen R, Pitkanen E. Unusual electrocardiographic changes in pheochromocytoma. *Acta Med Scand* 1963;**173**:41-4.
- ³ Radtke WE, Kazmier FJ, Rutherford BD, Sheps SG. Cardiovascular complications of pheochromocytoma crisis. *Am J Cardiol* 1975;**35**:701-5.
- ⁴ Hausmann VM, Getzowa S. Adenoma of organ of Zuckerkandl. *Schweiz Med Woch* 1922;**52**:889-92.

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Measles serology in children with a history of measles in early life

Since the introduction of routine measles vaccination in 1968 the yearly uptake of the vaccine in England and Wales has not exceeded 54%.¹ The failure to protect against this disease is reflected in the 139 485 cases reported to the Office of Population Censuses and Surveys in 1980.²

In many children who were admitted to a north London district hospital and in whom there was a history of measles before the age of 12 months we found that the duration of the rash was often stated to be only two or three days and a history of cough frequently absent or very mild. Hence we were led to question the diagnosis of measles in these children. Since a history of measles is one of the reasons for withholding measles vaccine, we decided to measure the serum measles antibody titres in children with such a history.

Patients, methods, and results

With informed consent in each case, 80 children aged 6-36 months who were seen at St Ann's General Hospital, London, after April 1980 were examined serologically for measles haemagglutination inhibition antibodies. The age at which measles was stated to have occurred was recorded. Clinic records confirmed that none of the children had received measles vaccine. According to the mothers, 76 of the children had had their measles diagnosed by either the family doctor or the clinic doctor; in four cases the parents or grandparents had made the diagnosis.

Measles was stated to have occurred before the first birthday in 53 children, and in nine of these before the age of 6 months. The table gives the results. Of children whose measles was stated to have occurred before 12 months of age, over two thirds were seronegative (haemagglutination inhibition antibody titre < 1/8). Also of children whose measles was reported after the age of 12 months, just under one third were seronegative.

Number and percentage of seronegative children according to age at which measles was reported

Age measles reported (months)	No examined	No (%) seronegative*
4-12	53	36 (67.9)
13-24	27	8 (29.6)

*Measles haemagglutination inhibition antibody titre < 1/8.

Comment

Measles is a not inconsequential infection, with a moderate to high fever, conjunctivitis, a blotchy rash lasting three or more days, and a troublesome cough. The presence of Koplik's spots is pathognomonic. The clinical diagnosis should not therefore be difficult in most cases, and measles should not be confused with other infections accompanied by a morbilliform rash in early childhood. These are chiefly caused by enteroviruses; in these infections Koplik's spots are not seen, the rash often lasts for less than three or four days, and coughing is seldom protracted or severe. It was surprising that the diagnosis of measles in children under 1 year and even in almost

one third of children aged 12-24 months was so often incorrect as judged by the serological evidence.

As was found by one of us in an earlier study,³ these findings are relevant in attempts to prevent measles by vaccination in Britain. In the earlier study one of the most frequent reasons for failure to be given vaccine was the history of measles before the 15-18 months of age at which the vaccine was due to be given. There is no evidence that measles vaccine is harmful to a child who has had the disease (S Krugman, personal communication, 1981). We therefore suggest that measles vaccine should be administered to children irrespective of a history of the disease in the first year of life. If there is hesitation to give vaccine because of an alleged history of measles the uncertainty of such a clinical diagnosis can be pointed out to the parents and antibody testing offered to clarify things.

We thank Dr Christine Miller for helpful comments.

¹ Department of Health and Social Security. *Health and personal services statistics*. London: HMSO, 1980.

² Office of Population Censuses and Surveys. *Infectious diseases, 1980*. London: OPCS, 1980.

³ Adjaye N. Measles immunization: some factors affecting non-acceptance of vaccine. *Public Health (London)* 1981;**95**:185-8.

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Therapeutic abortion and chlamydial infection

Chlamydia trachomatis is a common cause of genital infection in both sexes.¹ In women, symptomless colonisation of the cervix may be followed by an ascending infection and the development of pelvic inflammatory disease. We set out to determine the incidence of sexually transmitted infections (including *Chl trachomatis* infection) in a group of women attending this hospital for termination of pregnancy.

Patients, methods, and results

A group of women attending the day care unit of the department of gynaecology at this hospital requesting therapeutic abortion within the first seven weeks of pregnancy was studied. Patients who had received antimicrobial drugs during the previous three months were excluded, and a total of 89 entered the study.

Specimens were collected from the cervix before operation and cultured by standard procedures for *Neisseria gonorrhoeae*, *Chl trachomatis*, *Mycoplasma hominis*, and *Ureaplasma urealyticum*; and from the vagina for *Trichomonas vaginalis* and *Candida* spp. A specimen of blood was taken for serological

Micro-organisms isolated from lower genital tract of 89 women before termination of pregnancy

Micro-organisms	No of patients infected
<i>Chl trachomatis</i> alone	1
<i>Chl trachomatis</i> , <i>U urealyticum</i>	3
<i>Chl trachomatis</i> , <i>U urealyticum</i> , <i>M hominis</i>	2
<i>Chl trachomatis</i> , <i>U urealyticum</i> , <i>M hominis</i> , <i>N gonorrhoeae</i>	1
<i>M hominis</i> alone	3
<i>M hominis</i> , <i>U urealyticum</i>	13
<i>U urealyticum</i>	33
<i>T vaginalis</i>	0
<i>Candida</i> spp	16