ischio-rectal mass which was not fluctuant. Sigmoidoscopy to 20 cm showed no intrinsic rectal lesion. At exploratory operation the entire ischio-rectal fossa was replaced by woody hard tumour, with surrounding inflammation. No pus was obtained and bacteriological swabs of the mass gave no growth on culture. Histological examinations of biopsy specimens showed fat and muscle infiltrated with poorly differentiated squamous carcinoma consistent with an origin in the bronchus. The chest x-ray film had disclosed an opacity in the right hilum, suggestive of bronchial carcinoma. Subsequent bronchoscopy confirmed the presence of a carcinoma arising from the right bronchus—biopsy specimens showed that it was a squamous-cell carcinoma.

Discussion

In a review of published work we have been unable to find any recorded cases of carcinoma of bronchus spreading to the ischio-rectal fossa, though bronchial carcinoma metastasising to the gluteus maximus and presenting as a gluteal abscess has been reported.¹ Furthermore, a review of 16 394 necropsies (747 cases of bronchial carcinoma) found no evidence of metastasis to the ischio-rectal fossa but noted secondary deposits in the rectum in 2.2% of cases.² The mechanism of spread is uncertain. The tendency of malignant cells to metastasise to areas of trauma or inflammation is well known.³ Possibly a sub-clinical episode of infection in the ischio-rectal fossa occurred, with resultant deposition of tumour cells locally. The histological similarity of biopsies taken and the negative findings at sigmoidoscopy excludes the possibility of two primary tumours.

We are grateful to Mr D Pratt for his comments and for allowing us to report this case.

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Rebound phenomenon during gradual withdrawal of clonidine

Clonidine is a powerful antihypertensive agent. If long-term treatment is suddenly discontinued it may precipitate a withdrawal crisis, in which sharp increases in blood pressure and urinary and blood catecholamines occur.¹ We investigated the possibility that this syndrome could be prevented by gradual withdrawal of the drug in a patient who had been taking excessive doses of clonidine.

Case report

A 21-year-old man was admitted to hospital for readjustment of his anti-hypertensive treatment. Three years previously essential hypertension had been diagnosed. His blood pressure was 200/110 mm Hg and clonidine treatment was started. Whenever he forgot to take the clonidine he became acutely agitated. He also complained of flushes, and therefore increased the doses of clonidine spontaneously. On admission he was taking 3 mg/day and his blood pressure was 120/80 mm Hg. Since he seemed unreliable and had a history suggestive of repeated withdrawal crises, we decided to start him on a new course of treatment. A careful withdrawal schedule was planned.

Urine was collected for 24-hour measurement of catecholamines (maximum normal values: noradrenaline 0.55 $\mu\text{mol}/24\text{ h}$ (100 $\mu\text{g}/24\text{ h}$); adrenaline 0.12 $\mu\text{mol}/24\text{ h}$ (20 $\mu\text{g}/24\text{ h}$), measured fluorometrically). Supine blood pressure was recorded four times a day. The means of systolic and diastolic pressure were calculated daily. Clonidine dosage was decreased by 0.3 mg for the first five days, and by 0.15 mg for the next 10 days.

During withdrawal blood pressure gradually increased to a maximum mean value on day 14 of 212/126 mm Hg (figure). The maximum value recorded was 242/134 mm Hg, which greatly exceeded the initial pretreatment values. Catecholamine concentrations were low during the first 10 days, but started to rise gradually on day 11, reaching a maximum value of 1 $\mu\text{mol}/24\text{ h}$ (190 $\mu\text{g}/24\text{ h}$) on day 15. When catecholamine concentrations were maximal the patient was anxious and could not sleep.

Mean blood pressure values and urinary catecholamine concentrations during and after gradual withdrawal of clonidine.

Conversion: SI to traditional units—urinary catecholamines (as adrenaline) 1 $\mu\text{mol}/24\text{ h}$ = 183 $\mu\text{g}/24\text{ h}$.

After withdrawal of clonidine the blood pressure gradually decreased and he showed the clinical picture of essential hypertension with neurogenic symptoms. He was then given propranolol 60 mg/day on day 18. On day 20 catecholamine concentrations had greatly decreased. Clinical signs gradually declined and disappeared around day 20 except for repeated episodes of flushing. His progress has been good on propranolol 120 mg/day, the blood pressure being within the range of 150/100 mm Hg.

Increases in catecholamine concentrations and blood pressure were closely related; the correlation coefficient was 0.74 for systolic pressure and 0.83 for diastolic pressure ($P < 0.001$).

Comment

Several reports have shown that the sudden withdrawal of long-term clonidine treatment may precipitate a hypertensive crisis^{2,3} accompanied by a rise in catecholamine concentrations, which may reach the levels found in pheochromocytoma.¹ This phenomenon might be prevented by gradual reduction of clonidine dosage.⁴ Nevertheless, a typical rebound phenomenon has been described after gradual withdrawal of clonidine,⁵ though the patient was also undergoing beta-blockade—a combination that may independently cause an important rise in blood pressure. Our patient also showed a typical rebound phenomenon when the daily dosage of clonidine was gradually reduced. Blood pressure and urinary catecholamine concentrations were greatly increased; the increases were statistically closely correlated. Our findings suggest that even gradual withdrawal of clonidine may precipitate a rebound phenomenon.

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