case the diagnostic problem was not great because of the finding of acid-fast bacilli in the sputum, the subsequent growth of *M. tuberculosis* in sputum culture, the histological appearance, as well as the dramatic response to antituberculous treatment. Tuberculous skin lesions may, however, present a diagnostic problem when not accompanied by abnormalities on the chest roentgenogram (Kleid and Rosenberg, 1970). Tissue stains, cultures, and guinea-pig inoculations are negative in most cases. Furthermore, an abnormal chest film and the presence of a skin lesion usually, and justifiably, call to mind the diagnosis of actinomycosis. It is hoped that the present case may serve as a reminder that one should also consider tuberculosis in this setting.

The pathogenesis of the skin ulcer in this case is not clear. Most extrapulmonary tuberculosis represents haematogenous spread from a primary focus in the lung. Tuberculous lesions of the skin were once thought to arise from this lymphatic spread along the path of an intercostal nerve tracking out at the lateral branch. Two recent cases of tuberculosis involving the skin of the chest at the site of blunt trauma were attributed to silent primary tuberculosis and bacillaemia (Stead and Bates, 1971). In these cases the only clinical evidence of pulmonary involvement was hilar adenopathy, there being no roentgen manifestations of parenchymal or pulmonary involvement. The present patient had cavitary tuberculosis including pronounced pleural thickening immediately subjacent to the tuberculous skin ulcer. For this reason we suggest that this skin ulcer might have been caused by contiguous spread from the pleura.

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Ethambutol and a False-positive Screening Test for Phaeochromocytoma

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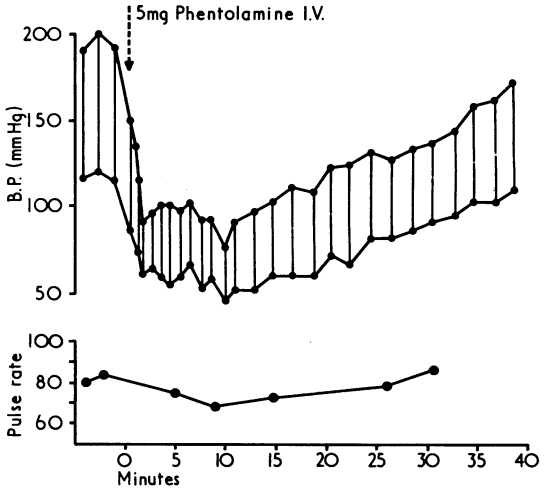
Case Report

A 46-year-old woman presented with renal tuberculosis. Urogenital tuberculosis was first diagnosed in January 1968, but despite chemotherapy and left nephrectomy tubercle bacilli continued to grow from her urine. On examination in February 1971 she weighed 55 kg and her blood pressure was 130/80 mm Hg. The blood urea was 46-60 mg/100 ml, and the creatinine clearance 40-48 ml/min. Tubercle bacilli recovered from the urine were sensitive to streptomycin, para-aminosalicylic acid, ethambutol, and rifampicin and highly resistant to isoniazid. Treatment was begun with streptomycin 500 mg, ethambutol 1,400 mg (25 mg/kg body weight), and rifampicin 600 mg daily. In May the ethambutol was reduced to 800 mg daily (15 mg/kg body weight). The blood pressure was 140/80 mm Hg. Streptomycin was discontinued in July owing to a persistent rash.

In August the blood pressure was found to be persistently raised at 170/120 mm Hg and a retinal haemorrhage was present. No history suggestive of a phaeochromocytoma was obtained. Renal function was unchanged. The hypertension was investigated. Phentolamine (Rogitine) 5 mg intravenously produced a marked depressor effect lasting more than 30 minutes (see Fig.). Glucagon provocation test negative, plasma volume 2·7 litres, total 24-hour exchangeable sodium 2,228 mEq (normal for weight 2,250 mEq), maximum urine osmolarity after intramuscular vasopressin (Pitressin) 508 mOsm/l., maximum urine pH after ammonium chloride loading (1 mg/kg body weight) 5·4, vanillomandelic acid excretion on two occasions not increased, plasma adrenaline, noradrenaline, and catecholamines within normal limits. After these investigations ethambutol and rifampicin were withdrawn. Over a two-week period the phentolamine test became negative. The hypertension was then controlled with guanethidine given by mouth.

Six weeks later, after temporary withdrawal of the guanethidine, the phentolamine test remained negative, but three days after reinstituting treatment with ethambutol 800 mg and para-aminosalicylic acid 12 g daily a depressor response to intravenous phentolamine was found. The drugs were withdrawn and after two weeks the phentolamine test was again negative. Para-aminosalicylic acid combined with rifampicin 600 mg/day did not result in a positive phentolamine test. When ethambutol was added to this drug regimen the phentolamine test became positive within three days.



Effect of intravenous phentolamine on blood pressure and pulse rate.

Comment

False-positive phentolamine tests in uraemia are well known (Sheps *et al.*, 1966), but although this patient had a damaged kidney her blood urea did not rise above 60 mg/100 ml over the whole period of observation. A phaeochromocytoma is a difficult tumour to exclude and may be present in the absence of raised catecholamine excretion (Litchfield and Peart, 1956). In this patient the timing of positive phentolamine tests in relation to ethambutol administration and the normal plasma adrenaline and noradrenaline levels (taken immediately before a positive phentolamine test) made the existence of a chromaffin cell tumour most unlikely.

Ethambutol excretion is about 80% through the kidneys (Place and Thomas, 1963). The plasma concentration in this patient 24 hours after 800 mg (15 mg/kg body weight) was 2·1 μg/ml, implying impaired excretion (Strauss and Erhardt, 1970).

It is assumed that ethambutol in high concentration reacts in some way with phentolamine. The nature of the interaction is obscure.

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This case is reported to draw attention to this false-positive phentolamine test which might be found in other patients being investigated for hypertension while taking ethambutol.

I am indebted to Mr. Howard G. Hanley for permission to report this case. The plasma catecholamines were estimated by Dr. Malcolm Carruthers, of the Institute of Ophthalmology

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Unusual Case of Paratyphoid Fever

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Salmonella paratyphi C is not normally isolated in Canada or the U.K. The present report concerns a case of paratyphoid that occurred and was treated in British Columbia. Investigations failed to trace the source of infection.

Case Report

A 14-year-old white schoolboy with no previous illnesses of any note became ill with a fever which lasted about two weeks. He had fainted once during the course of this time but had not had any abdominal pain, vomiting, or diarrhoea. There were no similar cases in the area at the time.

Examination showed no abnormalities except a temperature of 39·4° C and a sinus tachycardia of 110/min. He was admitted to hospital and the following investigations were done: chest *x*-ray picture negative; urine, routine analysis and culture negative six times; E.C.G. normal on each of three times performed; anti-streptolysin O titre 50 units; throat swab negative; Paul-Bunnell test negative twice; haemoglobin 14 g/100 ml. Blood measurements for days 1, 6, and 8 are given in the Table.

Haematological Picture during Treatment

Day	W.B.C. (/mm³)	Poly-morphs	Stab Cells	Lympho-cytes	Mono-cytes	E.S.R. (mm/hr)
1 ..	8,000	54%	13%	29%	4%	35
6 ..	39,000	27%	49%	19%	5%	42
8 ..	7,000	44%	3%	48%		104

PROGRESS

While in hospital his temperature fluctuated between 38° and 40°C until he was started on intravenous medication. Blood cultures were taken immediately on admission but grew nothing for the first few days.

Day 3.—He developed a discharge in the posterior nasopharynx which was somewhat haemorrhagic. It was not readily moved by swabbing. Sinus *x*-ray films were negative. A culture from the posterior nasal discharge grew *Diplococcus pneumoniae*.

Day 4.—His posterior nasal discharge was still present and he had a number of small shallow ulcers on the nasal septum. He also developed two large *scrotal infarcts*, one about 1 in (2·5 cm) long and the other about ½ in (1·3 cm). The skin became rapidly dark and black and then sloughed in these areas. At that time the question of diphtheria arose and it was noted that he had been immunized against diphtheria, but consultation with an E.N.T. specialist and an internal medicine physician was obtained largely to try to exclude this seemingly remote possibility. Both physicians felt confident that this was not a case of diphtheria although the diagnosis was obscure at the time. The possibility of infectious mononucleosis was again raised and consideration of Wegener's granuloma, septicicaemia possibly due to pseudomonas, and also a vasculitis due to periarteritis nodosa were discussed.

Day 6.—By this time it seemed evident that some sort of septicaemia was involved and intravenous ampicillin 500 mg and cloxacillin 500 mg every four hours was instituted, since the organism was unknown.

Day 9.—There was noticeable clinical improvement with a fall in the fever and the mucosal and skin lesions started to heal. On this day the blood culture taken from day 1 was read as positive and a probable salmonella organism was identified.

Day 10.—The cloxacillin was stopped and ampicillin continued.

Day 17.—The organism was identified as *Salm. paratyphi* C (1). He was clinically a great deal better and the intravenous ampicillin was changed to 500 mg every six hours by mouth.

Day 24.—Stool cultures grew the same *Salm. paratyphi* C (1) three times. He was feeling well but developed a new lesion in the mouth. It appeared to be a simple vesicle but was approximately 4 mm on its longest side. Since there had been earlier oral mucosal lesions and it was difficult to be certain about the cure of this condition it was elected to discontinue the ampicillin and he was started on co-trimoxazole in a dose of two tablets twice a day. This had just become available in the hospital on an experimental basis.

Day 26.—It was obvious that this was a simple vesicle in the mouth and he was discharged to continue with the co-trimoxazole for two weeks at home.

He was seen in the course of the next two weeks as an outpatient and made a smooth recovery with healing of his scrotal lesions. Three stools were tested at about six weeks after his discharge and they were reported as negative.

Comment

Systemic salmonella infections are increasing in incidence. Focal lesions have been described in numerous organs and tissues in such cases but I have seen no record of involvement such as we have had in this case with lesions of the nasopharynx and nasal septum and actual infarcts of the skin of the scrotum. The importance of early blood cultures in such cases is obvious and the long period of incubation (nine days) of the paratyphoid organism is noteworthy. The way in which these lesions are produced is not clear, but since they seemed to start about two weeks after the onset of the illness it raises the possibility of circulating antigen-antibody complexes being responsible.

I wish to thank Dr. D. Klassen and Dr. R. W. Van der Flier for their help with this case.

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