commended, however, in the generation of hypotheses. The two cases presented here may point to a genetic link in the development of gut lymphoma in support of previous reports.^{1,2} On the other hand, the EB virus titres were surprisingly high, and one patient had a history of malaria. The association of EB virus, malaria, and Burkitt's lymphoma is well known,³ and studies of non-Burkitt's lymphoma have yielded complexes of low molecular weight RNA and reverse transcriptase.⁵ Further study of intestinal lymphoma may yield more clues about the cause of this group of diseases.

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Arthritis simulating thrombosis on urokinase scan

^{99m}Tc-labelled urokinase is used to diagnose thrombosis in leg veins because, unlike ¹²⁵I-labelled fibrinogen, it detects clot that has already formed and can disclose thrombi in the thigh as well as the calf.¹⁻³ Despite these advantages the specificity of the technique is not known. We report two cases where arthritis of the knees caused a false-positive diagnosis of venous thrombosis.

Case reports

A 56-year-old woman with an eight-year history of rheumatoid arthritis presented with clinical and electrocardiographic evidence of myocardial infarction. Both knees were warm and painful, and small effusions were detectable. In the calves there was no tenderness, swelling, or superficial venous distension. The RA latex test result was positive. Although scanning with ^{99m}Tc-labelled urokinase disclosed increased radioactivity over both knees (see figure) bilateral venograms performed the next day showed normal results.

A 60-year-old woman with a six-year history of seropositive rheumatoid arthritis was admitted to hospital for investigation of anaemia. Both knees showed radiological features of rheumatoid arthritis—that is, narrowed joint spaces and marginal lipping—but the patient did not complain of pains in her joints. The legs were wasted, without any features of venous thrombosis. Scanning with ^{99m}Tc-labelled urokinase showed increased activity over both knees (figure) although bilateral venograms performed two hours later were normal.

Comment

The classic clinical features of deep venous thrombosis in the legs are only infrequently present, and various techniques—for instance, venography, isotope scanning, doppler ultrasound, and plethysmography—have been used to detect thrombi that are not clinically apparent. The results of using the plasminogen activator urokinase have proved promising. Necropsy studies in dogs have shown that ^{99m}Tc-labelled urokinase localises in blood clots⁴ and in man there is a good correlation between thrombi detected by venography and thrombosis detected by ^{99m}Tc-labelled urokinase.⁵

These two cases indicate that rheumatoid arthritis can masquerade as venous thrombosis when this method of detection is used. Furthermore, the second case illustrates that this may occur in the absence of pain or discomfort in the affected joints. These observations call into question whether other causes of hyperaemia—for example, cellulitis—cause false-positive diagnoses, and also emphasise that

Case 1 Case 2

^{99m}Tc-labelled urokinase scans showing increased activity over both knees in cases 1 and 2.

evidence of arthritis should be sought before anticoagulant treatment is instigated on the basis of increased activity on a urokinase scan.

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Normotensive familial phaeochromocytoma with predominant noradrenaline secretion

Hypertension is a typical symptom of phaeochromocytoma. We report a patient with familial phaeochromocytoma who was normotensive despite a persistently raised urinary noradrenaline excretion.

Case report

A 40-year-old woman was referred to our hospital because of suspected phaeochromocytomas. A phaeochromocytoma had been discovered in her

Catecholamines in urine and blood pressure recordings of the patient (n=123), her sister (n=20), and her son (n=5) during the observation period

	Blood pressure ± SD (mm Hg)		Noradrenaline (µg/24 h)	Adrenaline (µg/24 h)	Dopa (µg/24 h)	Dopamine (µg/24 h)	Homovanillic acid (mg/24 h)	Vanillyl- mandelic acid (mg/24 h)
	Systolic	Diastolic						
Normal values	..	..	60	15	32	400	10.5	6.0
Patient	104 ± 15	71 ± 13	1866.2	< 0.1	ND	338.8	6.9	10.5
preoperative 15/9/74	..	..	553.9	3.8	ND	180.6	7.3	13.8
30/10/74	..	..	458.7	7.1	17.6	76.4	7.2	13.5
20/2/75	..	..	21.8	0.4	ND	241.4	4.1	3.0
postoperative 9/6/75	..	..	..	..	..	..	..	..
Sister	123 ± 12	90 ± 9	481.3	12.2	29	270.8	7.6	13.8
preoperative	..	..	..	..	..	..	..	..
Son	170 ± 18	142 ± 17	737.2	0	ND	99.5	2.8	11.4
preoperative	..	..	..	..	..	..	..	..
Catecholamines in tumour tissue (µg/g*)								
Patient	..	..	304.3	31.3	0.56	4.5	4.68	0.05
Sister	..	..	618.1	21.7	0.47	10.5	0.73	0.70

*Except for noradrenaline, all values are within normal range for adrenal glands.
ND = Not determined.

younger child, an 8-year-old boy, who was in hospital with a hypertensive crisis. She complained of weakness, dizziness, nausea, and occasional episodes of hunger and palpitation. Her blood pressure had always been normal. Both her parents died at an early age of unknown causes. For two years her only sister had had hypertension (around 140/100 mm Hg), which we now know was caused by a pheochromocytoma.

Examination showed an asthenic woman without neurocutaneous abnormalities. Her blood pressure was 140/90 mm Hg initially; over the next ten days it was always normal (see table). Her pulse rate ranged between 80 and 90/min. The results of numerous routine laboratory investigations were normal. The electrocardiogram was normal. The chest x-ray film showed no enlargement of the heart. A percutaneous abdominal angiographic study disclosed symmetrical, highly vascularised masses in the region of both adrenal glands. The bilateral tumours were removed transabdominally. The right one measured 4 × 3.5 × 3.5 cm and the left 5.5 × 3.5 × 1.5 cm. Histological examination of the tumours showed a benign encapsulated pheochromocytoma.

The results of the determination of catecholamines in urine and tumour tissues of the patient, her sister, and her younger son are shown in the table. It should be emphasised that these urinary concentrations of noradrenaline were obtained when our patient's blood pressure and pulse rate were normal, whereas her sister had intermittent and her son continuous hypertension.

Comment

The absence of hypertension in pheochromocytoma has been reported several times and variously ascribed to (a) predominant secretion of adrenaline, (b) concomitant dihydroxyphenylalanine (dopa) secretion, (c) inactivation of the noradrenaline within the tumour, (d) myocardial insufficiency, and (e) tolerance of tissue receptors to circulating catecholamines. With the first possibility the beta-adrenergic effects of adrenaline secreted by the tumour dominate the picture, and hypotension or even shock might become the major manifestation.¹ In such cases urinary and tumour analyses have shown hypersecretion of adrenaline. Louis *et al*² suggested that concomitant secretion of dopa might counterbalance the hypertensive action of noradrenaline in pheochromocytoma. Crout and Sjoerdsma³ suggested that there are two types of pheochromocytoma: small tumours with "low" content of noradrenaline (NA) and adrenaline (A) have a low urinary vanillylmandelic acid (VMA) : NA + A ratio and a low rate of turnover of catecholamines; large tumours with "high" content of NA and A show a high urinary VMA : NA + A ratio and large amounts of NA + A seem to be metabolised directly in the tumour, which might explain the lack of clinical manifestations.

Our patient's pattern of urinary catecholamine excretion shows that only noradrenaline and VMA levels were raised. Thus concomitant secretion of predominantly adrenaline, dopa, or dopamine cannot account for the lack of hypertension. She also had a low urinary VMA : NA + A ratio. Moreover, metabolites were low in the tumour tissue and hence local inactivation was not evident. Thus none of the mechanisms mentioned above apply to our case. As others have suggested, patients with pheochromocytoma seem to have a different vasoresponse to noradrenaline^{4,5}; some show pronounced tolerance to endogenous circulating catecholamines since the concentration is sometimes so high as to seem incompatible with life. Our case supports this hypothesis. Furthermore, it is intriguing that, though both her sister and her son showed the same urinary catecholamine excretion pattern, only our patient was always normotensive.

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Treatment of hyperlipidaemia in patients on chronic haemodialysis

Abnormalities of lipid metabolism occur often in patients with chronic renal failure, and these are not reversed by dialysis.¹ Few attempts have been made to correct the abnormalities, which may at least partly contribute to the accelerated atherosclerosis in these patients.² We have examined the effects of advice on lipid-lowering diets and treatment with slow-release nicotinic acid (Nicangin) in patients with types IIb and IV hyperlipoproteinaemia being treated with dialysis.

Patients, methods, and results

We had previously measured cholesterol and triglyceride concentrations in 131 patients on chronic haemodialysis (mean age 40.2 years),³ of whom 65% had hyperlipoproteinaemia: type IIa 5%, type IIb 21%, and type IV 39%.

Ten patients with type IIb and 10 with type IV were randomly selected for dietary treatment. Their usual diet, assessed by a dietitian, provided a mean of 10.9 MJ (2600 Kcal) daily, half of which were derived from fat (saturated and monounsaturated:polyunsaturated fat ratio 4:1). For one month, as outpatients, they were asked to modify their diet so that fat comprised 40% of total energy and the ratio of saturated and monounsaturated to polyunsaturated was altered to 1:1, while cholesterol was reduced from 675 to 275 mg daily. Carbohydrate was increased (from 40% to 50% of total daily calories) to maintain energy balance. After one month they reverted to their previous diet. To determine whether patients were following their lipid-lowering diet we measured linoleic acid as a percentage of total triglyceride fatty acids, as the percentage of linoleic acid increases appreciably when polyunsaturated rather than saturated fatty acids comprise most of the dietary fat.⁴

Three patients with type IIb and three with type IV hyperlipidaemia were recommended to take slow-release nicotinic acid, starting with 0.25 g four times a day and increasing to 0.5 g, which was continued for