plasma immunoreactive "β-MSH" (figure). The decrease in SER was greater in the females (46·3±13·8% SE of mean) than in the males (82·4±13·9% SE of mean) in whom the decrease was not significantly different from the normal ($0·1<P>0·05$).

Discussion

The decreased SER we found in CRF suggests that the hormone measured as immunoreactive "β-MSH" is not sebotrophic in man, though the possibility that sebotrophic activity is inhibited in renal failure cannot be excluded. The melanocyte-stimulating hormone in man has been thought to be a β-MSH, but recent evidence suggests this hormone normally exists only as part of the larger lipotrophin (LPH) molecule,[4] which cross reacts in the present assay of "β-MSH." Hence apparently LPH is not sebotrophic in man even, though ovine β-LPH is strongly sebotrophic in the rat.[1] This conclusion agrees with our evidence from several different clinical states that changes in SER and immunoreactive "β-MSH" are unrelated. The nature of the postulated sebotrophic hormone still remains to be clarified in man. Why there is a decrease in SER is uncertain. One possibility is the decrease in androgen metabolism which occurs in CRF.[5] Only two of our patients were being treated with testosterone; in one the SER was increased and in the other it was low. Further studies are required to establish whether there is an impaired target organ response to androgen in CRF. It is likewise uncertain to what extent the decrease in SER contributes to the "dry" scaly skin of CRF or indeed whether this is a factor in the pruritus. Since, however, these changes may be less obvious in hypopituitarism and hypoadrenalism, despite a comparable or larger decrease in SER,[1] changes in epidermal synthesis of structural lipid may also be important.

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Carcinoma of the pancreas and acute renal failure

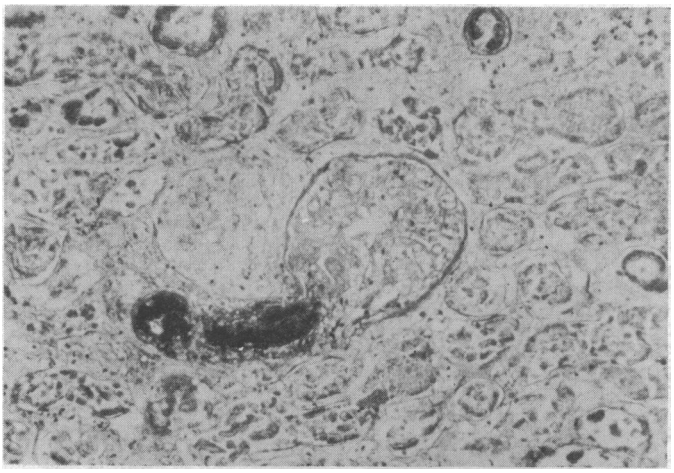
Acute renal failure associated with a pancreatic carcinoma may be caused by diffuse intravascular coagulation[1] or by renal tubular obstruction by mucoproteins.[2] We report a patient with an unsuspected pancreatic adenocarcinoma who presented with ischaemia of her right hand and subsequently developed acute renal cortical necrosis.

Case history

A 57-year-old woman was admitted to St Peter's Hospital, Chertsey, in March 1973 with a three-day history of paraesthesiae in her right hand. Twenty-four hours before admission the fingers of her right hand had become painful and cyanosed. She also complained of six weeks' vague central abdominal pain. Her right hand was ischaemic with an absent radial pulse; a loud systolic murmur was audible over the right clavicle, but there were no other abnormal physical signs. An aortogram showed stenosis of the right subclavian artery at the border of the first rib; no cervical rib was seen and a fibrous band was suspected. She was anticoagulated with heparin 10 000 units six-hourly without improvement. Nine days later her radial and brachial arteries were explored and thrombus removed from the radial artery using a Fogarty's catheter. Despite improved blood flow at operation her fingers became gangrenous.

Twelve days after operation, when apart from her ischaemic fingers she seemed well, she had mild haematuria and became totally anuric until her death six days later. Treatment with warfarin had begun two days earlier and heparin had been discontinued 36 hours after starting warfarin. Investigations at the onset of anuria showed a blood urea of 11·45 mmol/l (69 mg/dl) with normal electrolytes. Her haemoglobin had dropped 5·6 g to 8·6 g/dl since the last estimation ten days previously. Her white count was $33·6\times10^9$/l and platelets 194×10^9/l; the blood film was normal. Blood cultures were negative. Her plasma calcium was 2·38 mmol/l (9·5 mg/dl) and phosphate 1·87 mmol/l (5·8 mg/dl). Her prothrombin time was 120 s (control 12 s). Because of the fall in haemoglobin, a grossly prolonged prothrombin time, and total anuria, a retroperitoneal haemorrhage causing bilateral ureteric obstruction was suspected, but a retrograde pyelogram showed normal appearances. A renal arteriogram showed that both renal arteries were patent but produced no nephrogram.

At necropsy her kidneys showed confluent bilateral cortical necrosis with fibrin thrombi in the afferent glomerular arterioles (fig). A scirrhous mucus-secreting adenocarcinoma had replaced most of the head and body of the pancreas and had metastasised widely throughout the abdomen. Thrombi were present in the lungs, portal vein, submucosal veins of the colon (which showed patchy infarction), and in the right subclavian and brachial arteries.



Photomicrograph of kidney showing fibrin thrombus in afferent arteriole (× 125) stained with Mallory's phosphotungstic acid haematoxylin (PTAH).

Discussion

Cortical necrosis accounts for only 2% of cases of acute renal failure: concealed accidental haemorrhage in pregnancy is much the commonest cause,[3] but it may result from any sudden, severe renal ischaemia. This patient was at no time hypotensive, nor was there any obvious change in her clinical condition at the onset of anuria. Although there was no overt bleeding apart from the one episode of haematuria (reasonably explained by renal infarction), the platelet count was never low—and the blood film was normal—she was found at necropsy to have diffuse intravascular clotting in arteries and veins, large and small. Thrombophlebitis migrans is commonly associated with pancreatic carcinoma, but it is unusual for venous and arterial thromboses to coexist.[4] In this case thrombosis of a large artery was associated with predisposing subclavian artery stenosis.

In unexplained acute renal failure an unsuspected carcinoma must be considered as a possible diagnosis.

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