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(Accepted 22 July 1983)

Ulceration of the hand secondary to a radial arteriovenous fistula: a model for varicose ulceration

M L WOOD, G D REILLY, G T SMITH

Abstract

A patient in whom a radial arteriovenous fistula was constructed in preparation for haemodialysis subsequently developed ulceration on the dorsum of the hand. The lesion failed to heal despite antibiotic treatment, and so the fistula was closed. The lesion healed within three weeks. Subsequent construction of a fistula at the right wrist was followed 12 months later by the development of similar ulceration of the right hand.

This complication of arteriovenous fistulas is similar to varicose ulceration of the leg and provides a unique opportunity to study the effects of sustained venous hypertension on the skin.

Introduction

Ulceration of the hand may develop as a complication of arteriovenous fistulas. We report on a patient with this complication.

Case report

A side to side arteriovenous fistula was constructed at the left wrist between the dorsal radial artery and vein in preparation for haemodialysis in a 48 year old woman with chronic renal failure secondary to diabetic nephropathy. Her renal failure was managed conservatively, and the blood urea concentration remained raised at around 40 mmol/l (240 mg/100 ml). Eighteen months later she presented to the dermatology outpatients department with swelling of the left hand and an erythematous and indurated area on the dorsum of the hand between

Biopsy specimen showing thick walled capillaries with plump endothelial cells in upper dermis. Haematoxylin and eosin $\times 210$.

the thumb and the index finger, which had been present for about six months. She was not feverish and was otherwise well.

A skin swab grew *Staphylococcus pyogenes*, and she was given a course of oral erythromycin 250 mg four times a day. The lesion failed to heal, and a small ulcer developed over the proximal phalanx of the index finger. A biopsy specimen of the edge of the ulcer (figure) showed thick walled capillaries with plump endothelial cells in a mildly oedematous dermis. Larger vessels showed intimal proliferation. The thickened eosinophilic capillary walls and pericapillary tissue showed focal positive staining for fibrin by the peroxidase antiperoxidase technique. There was a light mixed inflammatory cell infiltrate including eosinophils in the dermis.

Because of the persisting ulceration the fistula at the left wrist was closed; within three weeks the lesion had healed. A further fistula was fashioned at the right wrist, and 12 months later she began to develop a similar erythematous, indurated area on the dorsum of the right hand.

Discussion

Ulceration of the hand as a complication of arteriovenous fistulas has been described previously.¹ It is thought to result from retrograde venous arterialisation, the high pressure in the

Rupert Hallam Department of Dermatology, Royal Hallamshire Hospital, Sheffield S10 2JF

M L WOOD, MB, MRCP, senior registrar
G D REILLY, MA, MRCP, registrar

Department of Pathology, Royal Hallamshire Hospital
G T SMITH, MB, MRCPATH, lecturer

Correspondence to: Dr M L Wood.

veins draining the hand causing swelling, induration of the skin, and eventual ulceration. Construction of a fistula linking the end of the vein to the side of the artery, with ligation of the venous branch draining the dorsum of the hand, may help avoid this complication.

Sustained venous hypertension on the skin capillaries of the canine hind limb causes an increase in the number and permeability of dermal capillaries with increased leakage of fibrinogen into the interstitium.² A recent study on ulcer bearing skin of the leg in man showed layers of fibrin surrounding the dermal capillaries, and it has been postulated that extravascular fibrin stimulates tissue fibrosis and blocks the diffusion of oxygen to the overlying epidermis, resulting in ulceration.³ Histological examination of our patient's skin showed this capillary proliferation and the pericapillary fibrin that are the hallmark of venous hypertension.

We believe that this complication of arteriovenous fistulas is under reported and provides a unique opportunity to observe the effects of sustained venous hypertension on the skin in man similar to that seen in varicose ulceration of leg.

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(Accepted 6 July 1983)

Severe diabetic microangiopathy with normal glucose tolerance and pituitary infarction

R W BILOUS, R N POSTON, H KEEN, G C VIBERTI

Abstract

Blood glucose concentrations in a woman with severe retinopathy, nephropathy, and neuropathy were found to be within the normal range. A high random blood glucose concentration was recorded only once, six months before her initial presentation. At necropsy the anterior lobe of the pituitary gland showed old, extensive infarction.

Occult pituitary hypofunction may explain severe diabetic microangiopathy in patients with normal glucose tolerance.

Introduction

In 1936 Houssay described the ameliorating effect of hypophysectomy on hyperglycaemia and ketosis in dogs that had undergone pancreatectomy.¹ Many studies on the influence of pituitary function on diabetes and its complications have since been reported.² We report on a patient with severe retinopathy, nephropathy, and neuropathy in the absence of diabetes mellitus at the time of presentation. Postmortem examination showed infarction of the pituitary gland.

Case report

A 44 year old woman presented to this hospital in July 1978 complaining of blurred vision. In December 1977 she had been admitted

to another hospital with a superficial loin carbuncle, when glycosuria and a random blood glucose concentration of 17.6 mmol/l (317 mg/100 ml) had been recorded. She had also had heavy proteinuria, with a creatinine clearance of 52 ml/min/1.73 m². She had been given a single injection of 20 units of insulin before drainage of the abscess but thereafter had been treated with only dietary restriction. Blood glucose concentrations had returned promptly to the normal range. No formal glucose tolerance test had been performed.

On initial examination at this hospital she was obese (90 kg) with facial hirsutism and hypertension (196/126 mm Hg). She had severe bilateral proliferative retinopathy with acuity reduced to 3/60 on the right and 3/2 on the left. Ankle reflexes and pin prick and vibration sensation were absent in both legs.

After fluorescein angiography she underwent right laser photocoagulation, but a vitreous haemorrhage blinded her in November 1978. Investigations showed proteinuria of 2.5 g/24 h and a glomerular filtration rate (edetic acid labelled with ⁵¹Cr) of 51 ml/min/1.73 m². A 75 g oral glucose tolerance test showed only impaired glucose tolerance (table). Her hypertension responded to beta blockers, and an intravenous urogram was normal. Left percutaneous renal biopsy in January 1979 confirmed diabetic glomerulosclerosis. Blood glucose concentrations on multiple occasions over this period never exceeded 6.1 mmol/l (110 mg/100 ml). By June 1980 the glomerular filtration rate (edetic acid labelled with ⁵¹Cr) had declined to 13 ml/min/1.73 m²; a second oral glucose tolerance test was performed, during which both plasma growth hormone and insulin concentrations were measured (table). Results of thyroid function tests, morning and evening plasma cortisol concentrations, and urinary free cortisol excretion were normal. She declined to undergo insulin stress test. By

Plasma glucose and growth hormone concentrations after 75 g oral glucose tolerance tests at presentation and two years later

Time after glucose load	23 August 1978 Plasma glucose (mmol/l)	16 June 1980	
		Plasma glucose (mmol/l)	Growth hormone (mU/l)
Fasting	5.9	4.1	1
30 min	8.0	5.7	1
60 min	8.0	6.9	1
90 min	8.5	6.8	1
120 min	7.3	7.2	1
150 min		7.2	1
180 min		5.3	1
240 min		4.8	1
300 min		3.9	0

Conversion: SI to traditional units—Glucose: 1 mmol/l $\approx$ 18 mg/100 ml.

Unit for Metabolic Medicine, Guy's Hospital Medical School, London SE1 9RT

R W BILOUS, BSC, MRCP, research registrar

H KEEN, MD, FRCP, professor

G C VIBERTI, MD, senior research fellow

Department of Histopathology, Guy's Hospital Medical School

R N POSTON, MRCPATH, lecturer

Correspondence and requests for reprints to: Dr R W Bilous, St Helier Hospital, Carshalton, Surrey.