all three patients the hands were particularly deformed and affected several years before the feet. While the patients we describe had had serologically active systemic lupus erythematosus for several years there did not appear to be a correlation between the development of arthritis and any clinical or serological pattern.

The process that results in the deformity is not understood but may result from inflammatory changes in tendons or periarticular structures. Occasionally a patient with systemic lupus erythematosus is noted to have pain and erythematous tenderness along the path of tendons. Inflammation and fibrosis have been described in tendon sheaths, and this may in turn result in acute or insidious contracture of the tendon.

We thank Dr N B Bowley, radiology department, and Dr G R V Hughes, rheumatology unit, for help and advice.


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Hyponatraemia in legionnaires’ disease

Hyponatraemia is common in legionnaires’ disease, and has been attributed to the syndrome of inappropriate secretion of antidiuretic hormone.¹ Our results suggested, however, that an alternative explanation is more likely.

Patients, methods, and results

Between December 1976 and May 1979, 24 patients with acute pneumonia were found to have legionnaires’ disease. Thirteen were hyponatraemic on admission (serum sodium concentration lower than 130 mmol(mEq)/l), and none was diabetic or receiving diuretics or corticosteroids. All had normal serum sodium concentrations (135-145 mmol/l) by the time of discharge. Before treatment five patients had measurements of sodium and osmolality, using standard methods, on simultaneous blood and urine samples.

Findings on admission in the five patients studied

<table>
<thead>
<tr>
<th>Case</th>
<th>Osmolality (mmol/kg)</th>
<th>Sodium (mmol/l)</th>
<th>Hyponatraemia</th>
</tr>
</thead>
<tbody>
<tr>
<td>No.</td>
<td>Serum</td>
<td>Urine</td>
<td>Serum</td>
</tr>
<tr>
<td>1</td>
<td>268</td>
<td>400</td>
<td>127</td>
</tr>
<tr>
<td>2</td>
<td>265</td>
<td>817</td>
<td>129</td>
</tr>
<tr>
<td>3</td>
<td>279</td>
<td>806</td>
<td>128</td>
</tr>
<tr>
<td>4</td>
<td>370</td>
<td>614</td>
<td>129</td>
</tr>
<tr>
<td>5</td>
<td>268</td>
<td>530</td>
<td>129</td>
</tr>
</tbody>
</table>

Conversion: SI to traditional units—Osmolality: 1 mmol/kg = 1 mosmol/kg. Sodium: 1 mmol/l = 1 mEq/l.

Patients were considered to be hyponatraemic if jugular venous pulsation became visible only with the patient lying at an angle of less than 5° and systolic blood pressure was at least 20 mm Hg greater in the supine than in the standing position.

The table shows results of investigations on admission. All five patients were hyponatraemic, with high urine osmolality and urinary sodium concentrations below 8 mmol/l. In a group of 12 patients with legionnaires’ disease 10 were hyponatraemic. One of the two exceptions had normal circulating...
Acute hypertension after traumatic renal artery thrombosis with high circulating concentrations of angiotensin II

Hypertension is a well-recognised complication of traumatic renal artery thrombosis. It occurs in up to half of patients who do not undergo early surgery but is rarely detected within the first few weeks of injury. A possible role for the renin-angiotensin system was suggested by Von Knorring et al. We describe a patient who became severely hypertensive 48 hours after traumatic renal artery thrombosis and in whom plasma concentrations of renin, angiotensin II, and aldosterone were measured serially.

Case report
A 48-year-old man was admitted to hospital after a road traffic accident. He was shocked (blood pressure 70/40 mm Hg with a standard sphygmomanometer, diastolic phase V) and had multiple fractures of the lumbar transverse processes and lower ribs. Intravenous pyelography showed a non-functioning right kidney, and complete occlusion of the right renal artery was confirmed by renal arteriography. He was resuscitated with intravenous fluids and whole blood. On day 3, however, his blood pressure rose progressively to a maximum of 260/156 mm Hg (figure) and remained poorly controlled despite administration of propranolol 80 mg three times daily together with intravenous infusions of clonidine 0.5 mg/h and hydralazine 20 mg/h. Despite adequate fluid replacement his urine output fell progressively to 400 ml on day 5 with a corresponding rise in serum creatinine concentration to 289 μmol/l (10.5 mg/dl).

He was transferred to the renal unit at Glasgow Royal Infirmary on day 6, where his blood pressure was finally controlled with sodium nitroprusside 0.5 ng/kg/min. Preoperative plasma concentrations of active renin and angiotensin II were grossly raised at 599 mU/l (normal range 9-50 mU/l) and 600 pmol/l (normal range 5-35 pmol/l) respectively. Plasma aldosterone concentration was less notably increased at 639 pmol/l (23 ng/100 ml) (normal <500 pmol/l (18 ng/100 ml)). At laparotomy on day 7 a severely ischaemic right kidney was removed. Histological examination showed almost complete infarction and confirmed thrombotic occlusion of the main renal artery.

Thirty minutes after the operation it was possible to stop all antihypertensive drugs, and thereafter blood pressure and plasma renin, angiotensin II, and aldosterone concentrations returned to normal (figure). He remained oliguric for 45 days, however, and received daily haemodialysis during this period. Respiratory failure requiring prolonged artificial ventilation was a major complication. He subsequently made a gradual but sustained recovery, and when last seen at the clinic six months after his first admission his serum creatinine concentration was 200 μmol/l (2-26 mg/100 ml) and blood pressure 124/82 mm Hg without treatment.

Changes in plasma concentrations of active renin, angiotensin II, and aldosterone, blood pressure, serum creatinine concentrations, and urinary output after traumatic renal artery thrombosis. Intravenous infusions of clonidine (C), hydralazine (H), and nitroprusside (Np) were administered before nephrectomy.

Conversion: SI to traditional units—Serum creatinine: 1 μmol/l = 11.3 μg/100 ml. Angiotensin II: 1 pmol/l = 1 pg/ml. Aldosterone: 1 pmol/l = 36 pg/100 ml.

Comment
Although hypertension is a well-recognised complication of traumatic renal artery thrombosis, its incidence is uncertain, probably because most patients undergo early surgery and have other features that may modify blood pressure. Cass et al found no evidence of hypertension in 31 consecutive patients with renal pedicle injuries, including seven who did not undergo nephrectomy or vascular repair, and whose blood pressure remained normal for a mean of 29 months. On the other hand, Stables et al noted hypertension in five out of 10 patients who did not undergo surgery.

Generally, where hypertension has occurred the interval between injury and its detection varies from 13 weeks to three years; thus the