Myocardial infarction in a young man

N P Singh, S Anuradha, Piyush Ranjan, S N A Rizvi

A 31-year-old male smoker, presented with a six-hour history of sudden onset, severe, retrosternal pain, associated with profuse sweating. There was no past or family history of ischaemic heart disease. The patient had a history of 'swelling' all over his body for the past two years. There was no haematuria or oligo-anuria. He had been prescribed only diuretics for his swelling, which he was taking off and on. On admission, the patient was anxious, in pain, and diaphoretic. His heart rate was regular, 92 beats/min, blood pressure 160/100 mmHg and he had pedal oedema. The chest and cardiovascular examination was normal. A standard 12-lead electrocardiogram (ECG) on admission is shown in figure 1. The results of the laboratory investigations are summarised in the table. Echocardiography done on the fourth day revealed apical hypokinesia. Further investigations were deferred. Three months later, a coronary angiography demonstrated entirely normal coronary vessels. A kidney biopsy was done and the histology is depicted in figure 2.

Table 1  Laboratory investigations

<table>
<thead>
<tr>
<th>Investigation</th>
<th>Normal range</th>
<th>First admission</th>
<th>After 3 months</th>
</tr>
</thead>
<tbody>
<tr>
<td>Haemoglobin (g/dl)</td>
<td>14–16</td>
<td>14.2</td>
<td>13.0</td>
</tr>
<tr>
<td>Serum albumin (g/dl)</td>
<td>4–6</td>
<td>1.6</td>
<td>1.9</td>
</tr>
<tr>
<td>24-h urinary protein excretion (g)</td>
<td>-</td>
<td>6.3</td>
<td>3.4</td>
</tr>
<tr>
<td>Serum cholesterol (mg/dl)</td>
<td>150–250</td>
<td>336</td>
<td>351</td>
</tr>
<tr>
<td>Serum triglycerides (mg/dl)</td>
<td>100–200</td>
<td>324</td>
<td>592</td>
</tr>
<tr>
<td>Low-density lipoproteins (mg/dl)</td>
<td>86–138</td>
<td>223</td>
<td>183.4</td>
</tr>
<tr>
<td>Apo B (mg/dl)</td>
<td>63–114</td>
<td>—</td>
<td>173</td>
</tr>
<tr>
<td>Blood urea (mg/dl)</td>
<td>20–40</td>
<td>20.0</td>
<td>25.0</td>
</tr>
<tr>
<td>Serum creatinine (mg/dl)</td>
<td>0.8–1.6</td>
<td>1.1</td>
<td>0.8</td>
</tr>
<tr>
<td>Serum fibrinogen (mg/dl)</td>
<td>150–400</td>
<td>—</td>
<td>526.0</td>
</tr>
<tr>
<td>Antithrombin III (%)</td>
<td>80–120</td>
<td>—</td>
<td>60.0</td>
</tr>
<tr>
<td>Protein C (%)</td>
<td>70–140</td>
<td>—</td>
<td>126.0</td>
</tr>
<tr>
<td>Platelet adhesiveness to ADP and ristocetin</td>
<td>—</td>
<td>—</td>
<td>enhanced</td>
</tr>
</tbody>
</table>

Questions

1 What is the primary diagnosis?
2 What is the complicating event and what is the underlying aetiology?
3 How will you manage such complications?
Answers

QUESTION 1
The primary diagnosis is nephrotic syndrome as evidenced by proteinuria (6.39 g/24 h), hyperlipidaemia (serum cholesterol 336 mg/dl, triglycerides 324 mg/dl), hypoalbuminaemia (serum albumin 1.6 g/dl) and oedema. The diffuse glomerular basement membrane thickening seen on kidney biopsy is characteristic of membranous glomerulonephritis, which is the underlying histological abnormality.

QUESTION 2
The patient suffered an acute anteroseptal myocardial infarction as the complicating event. This is evidenced by the ECG, raised levels of cardiac enzymes and echocardiography. Since no evidence of coronary artery disease was found on subsequent angiography, in-situ thrombosis of the coronary artery secondary to the hypercoagulable state of nephrotic syndrome, (demonstrated by hyperfibrinogenemia, reduced antithrombin III levels and enhanced platelet aggregability) was the likely event.

The nephrotic syndrome is associated with a hypercoagulable state. It is associated with profound changes in fibrinogen levels, zymogens and cofactors, the fibrinolytic system, coagulation inhibitors and platelet functions, summarised in boxes 1 and 2. All these alterations are proposed to contribute to the hypercoagulable state, which is an important risk factor for the genesis of the thromboembolic complications of the nephrotic syndrome. Other contributory risk factors for the thromboembolic episodes are listed in box 3.

QUESTION 3
It is generally agreed that all patients with nephrotic syndrome and a proven, life-threatening thromboembolic complication should be anticoagulated. Anticoagulation should continue for at least six months after the proteinuria remits or until the serum albumin is lower than 2 g/dl. The role of prophylactic anticoagulation in preventing the occurrence of thrombotic events is not clearly established. While some researchers propose routine anticoagulation for all patients, especially those with membranous nephropathy, most physicians advocate an individualised approach. There is growing evidence for the role of thromboprophylaxis with antithrombotic agents like aspirin.

In addition to the above, control of attendant risk factors like hypoalbuminaemia and proteinuria, hyperlipidaemia, hypertension and smoking, are an integral part of the management strategy.

Discussion
Thromboembolic episodes are well-recognised, serious, and at times life-threatening complications of the nephrotic syndrome. Although venous thromboses are more common, thrombotic occlusions of nearly all arteries have been described in the literature. Isolated coronary artery thrombosis in the nephrotic syndrome, in the absence of coronary artery disease, has rarely been documented. The hypercoagulable state of the nephrotic syndrome is proposed to be responsible for these complications, as discussed earlier.

Normal coronary vessels on angiography in patients with a proven myocardial infarction is a well-recognised entity, especially in younger patients. Normal angiography in a patient with a myocardial infarction could be due to a number of causes, including a hypercoagulable
Learning points

- Nephrotic syndrome is a hypercoagulable state.
- Thrombotic episodes are frequent manifestations of the hypercoagulable state in nephrotic syndrome.
- Although less common than venous thrombosis, arterial thrombosis may be life-threatening.
- Normal coronary vessels may be seen in young patients with acute myocardial infarction.
- A hypercoagulable state is a possible contributory factor to coronary artery thrombosis.
- Normal coronary angiography should prompt a search to rule out an underlying hypercoagulable state.

Final diagnosis

Nephrotic syndrome complicated by acute anteroseptal myocardial infarction.

Keywords: Nephrotic syndrome; myocardial infarction.

A dislocated finger

C H Gerrand, P M Jarrett

A 64-year-old man presented 12 hours after he fell onto his hand whilst intoxicated. X-Rays are shown in figure 1.

Questions

1. What does the radiological appearance suggest?
2. What is the anatomy of this injury?
3. What is the correct treatment?
Myocardial infarction in a young man.

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