PHOTO QUIZ

A remarkable ECG of a patient with swollen legs

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CASE REPORT

A 69-year-old male, without any remarkable medical history, was admitted to the hospital because of predominantly rightsided heart failure. Despite initiation of furosemide two weeks before admission, his physical condition did not improve. Physical examination revealed oedematous legs and presacral oedema. His blood pressure was 100/60 mmHg with a pulse rate of 88 beats/min. The laboratory results were as follows: normal peripheral blood cell count, ureum 7.5 mmol/l, creatinine 80 µmol/l, ASAT 38 U/l, ALAT 40 U/l, γ-glutamyltransferase 55 U/l, alkaline phosphatase 118 U/l and C-reactive protein 3 mg/l.

The ECG (figure 1) showed sinus rhythm with microvoltages in the frontal leads and slow R progression in the precordial leads. Echocardiography revealed a concentric hypertrophic heart with moderate left systolic function, based on diffuse hypokinesia. Doppler showed a restrictive diastolic flow pattern. Hypoalbuminaemia (24g/l) was found, while monoclonal gammopathy was absent. Albuminuria of 2 g/day was documented.

Figure 1
The ECG shows sinus rhythm with microvoltages in the frontal leads and slow progression in the precordial leads

WHAT IS YOUR DIAGNOSIS?

See page 77 for the answer to this photo quiz.
DIAGNOSIS

The combination of a hypertrophic myocardium with diffuse diminished left ventricular function and glomerular involvement (macroalbuminuria) was suspicious for a systemic disease. The ECG pattern was concordant with cardiac amyloidosis. Invasive work-up confirmed restriction with square root sign, elevated pulmonary capillary wedge pressure and left ventricular end-diastolic pressure. Myocardial amyloidosis was histologically confirmed by endomyocardial biopsy. This case demonstrates the two most common and diagnostically useful ECG patterns in primary amyloidosis: pseudoinfarction pattern (sensitivity 63 to 85%) and low QRS voltage (sensitivity 60 to 93%).

Figure 1

The ECG shows sinus rhythm with microvoltages in the frontal leads and slow progression in the precordial leads

REFERENCES