Continued medical education:
Hypertension and systolic dysfunction (day case)

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Presentation

Mrs Grech, a 61 year lady woman was accompanied to my clinic by her daughter due to progressive dyspnoea. She had enjoyed good health until 3 months previously when she started to complain from exertional dyspnoea with minimal activity. Lately this complaint was accompanied by an increase in her weight and some swelling of her feet. Since the previous week her dyspnoea worsened and she noted orthopnea and some chest discomfort. She had been an ex-smoker since 7 years and did not abuse alcohol. She had also been on antihypertensive treatment for over 10 years but with poor blood pressure control. She was taking atenolol 100mg and hydrochlorothiazide 50 mg daily.

Clinical examination

She was obese (weight 79 kg, height 157cm) and tachypneic at 39 respirations per minute. Her BP was 165/95 mm Hg, with a regular tachycardia at 105 bpm. There were no carotid bruits but the jugular pressure was raised. Cardiac examination showed a laterally displaced apex beat at the sixth intercostal space, a 2/6 systolic murmur and a third heart sound. Lung auscultation revealed significant bilateral crepitations. She had pitting oedema of her legs, with normal pedal pulses. The rest of the examination was unremarkable.

Investigations

A chest X ray showed significant cardiomegaly and pulmonary oedema (Figure 1). On ECG, there was sinus tachycardia and a complete LBBB (Figure 2). Routine haematological and blood chemistry were normal except for an elevated cholesterol (total cholesterol: 7.9mmol/l, LDL: 5.1mmol/l, HDL: 0.98mmol/l) and triglycerides 3.2mmol/l.

Management

This lady was treated with an initial dose of IV frusemide and started on oral ACE inhibitor (enalapril 10mg daily), a diuretic (bumetanide 2mg daily) and aspirin 75mg daily (which was changed to a coumarin after the echocardiogram confirmed regurgitation). She was referred to a cardiologist who performed an echocardiogram which showed marked left ventricular hypertrophy (LVH), septum 12mm, posterior wall 11mm and left ventricle end-diastolic diameter 70mm. There was also evidence of marked global systolic dysfunction, with an ejection fraction of about 22% . There was also moderate mitral regurgitation and mild tricuspid regurgitation. Systolic pulmonary pressure was 48 mm Hg.

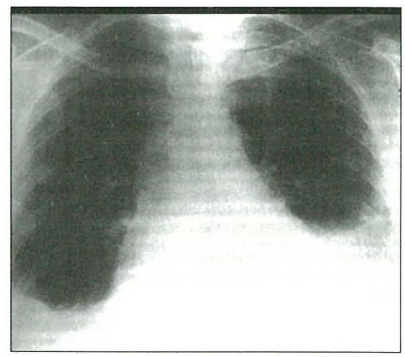

Figure 1

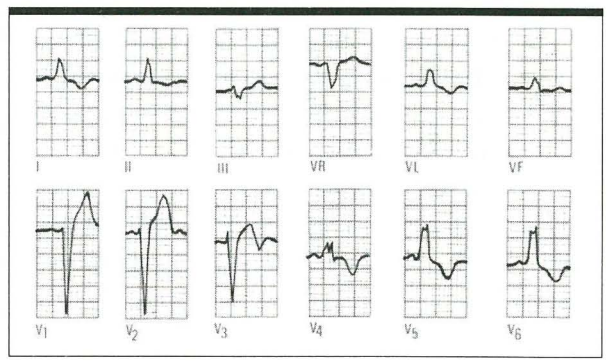

Figure 2

At a second review two weeks later, she was found to have no tachypnoea and no chest discomfort and a BP of 145/ 90. The ACE inhibitor dose was increased to 20mg daily and the diuretic dose was decreased to 1mg daily, a statin was also added in view of hypercholesterolaemia. An angiogram was booked to rule out ischaemic disease.

Questions

1. How does congestive heart failure develop during hypertension?
2. How could hypertensive heart disease be detected in such a patient?
3. What is the best treatment protocol and the prognosis for this patient?

Answers on page 41 After reviewing the answers you can claim 1 CME point by quoting code MFD/CME 001 on your application for accreditation.

Answers to questions on Page 13

1. Hypertension is known to be the most common risk factor for congestive heart failure(CHF). Although systolic function is usually preserved in hypertensive heart disease, diastolic function soon becomes abnormal causing pressure overload. The deterioration from left ventricular hypertrophy (LVH) with compensated cardiac function, to symptomatic CHF may be imperceptible, but diastolic filling is usually impaired. End stage hypertensive heart disease is characterized by systolic dysfunction, with dramatic worsening of symptoms. Reduction of BP often results in dramatic improvement in clinical symptoms. One must always rule out other causes of CHF such as occlusive coronary artery disease, hence the need for an angiogram in this case (apart from the fact that presence of a LBBB renders ECG interpretation of ischaemic changes dubious).

2. Identification of hypertensive heart disease rests on proper clinical assessment and non invasive diagnostic investigations. One may suspect LV dilatation and systolic dysfunction by finding lateral displacement of the apical impulse (from the 5th intercostal space, 8-9cm from the mid-line) and the presence of a third heart sound. A sustained apical impulse and a fourth heart sound may suggest LVH.
   Some experience with ECG interpretation may be rewarding by finding evidence of LVH and left atrial enlargement, the latter is a good sign reflecting diastolic dysfunction. The ECG also provides good information on arrhythmias, defective conduction and ischemic changes. Echocardiography is as excellent tool to diagnose hypertensive heart disease. It gives good information about the structure of the LV and determines the presence of LVH and its geometric model, whether it is concentric or eccentric. Concentric LVH is the predominant form in the elderly and middle- aged patients whereas eccentric LVH is uncommon in those under 50 years but can occur in up to 30% of patients over 60. CT scanning and magnetic resonance imaging provide high definition measurements of cardiac function and size but are not indicated for routine use.

3. Results of clinical trials done in the 1990s show that adding an ACE inhibitor to standard treatment decreases the high risk of hospitalizations and mortality related to CHF. The CONSENSUS trial in patients with severe heart failure demonstrated a 12 month mortality rate of 52% in the placebo group compared to 36% in the enalapril group(RR31%). Mortality rates in patients NYHA class II or III in the placebo and enalapril groups of the SOLVD trial were 39.7% and 35.2% respectively.
   Recently, blockers and spironolactone have also been shown to reduce mortality when added to ACE inhibitors, digoxin and diuretics. More recently still, ARBs have been marketed to replace ACE inhibitors in those patients with cough problems. Although we have a vast array of treatments which have evolved over the recent years allowing a better quality of life to our patients with CHF, the morbidity and mortality rates of patients with CHF remain still exceedingly high.

References and Further Reading

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