An incidental case of thrombus formation in a patient with a portacath inserted for regular blood transfusions

A 33 year old woman with thalassaemia major was referred to us for assessment of her myocardial iron status by cardiovascular magnetic resonance. Assessment of myocardial iron loading in the heart by the use of T2* measurements is the first non-invasive method that has been successfully validated for this purpose. Requiring regular blood transfusions for which a portacath had been inserted in 1992, she had never been formally anticoagulated. In cases of severe cardiac iron loading portacath insertion is often required to provide intravenous chelation treatment, and can also be used to provide long term intravenous access for the multiple blood transfusions. While being assessed a large mobile structure which appeared to be attached to the tip of the portacath was seen in the right atrium (highlighted by arrow.) The signal characteristics of the mass on various magnetic resonance imaging techniques indicates that this was a thrombus. Thrombus formation, which can then go on to embolise, is a recognised long term complication of a portacath, along with sepsis and leakage. The patient was subsequently treated with warfarin as she did not wish to have the line removed surgically.

M A Westwood
J S Wainscoat
R Mohiaddin
mwestwood@rbh.nthames.nhs.uk

Prolonged asymptomatic sinus pause indicated by implantable loop recording

A 51 year old man, with a previous history of 15 years of hypertension treated with β blockers, complained of nocturnal and early morning seizures. One year ago, he had a syncope thought to be caused by bradycardia. β Blockers were reduced in a first step, and then discontinued. Serial neurological investigations were normal. Therapeutic challenges using sodium valproate or phenytoin did not had any favourable influence. Glucose metabolism was normal.

Cardiac investigations including carotid sinus massage, recording of late potentials, repetitive 24 hour Holter monitoring, and tilt table testing were normal. At electrophysiological testing, AH interval was 80 ms, and HV interval was 42 ms. Sinus node function and anterograde atrioventricular conduction were normal. Programmed atrial and ventricular stimulation did not induce any sustained arrhythmia. The ajmaline test was negative.

A Reveal Plus implantable loop recorder (Medtronic) was implanted. One month later, at the follow up visit, the downloading of the recorded ECGs showed an asymptomatic nocturnal pause of 35 seconds. An antibradycardia pacemaker was implanted in 1999, and during three years of follow up the patient has remained completely asymptomatic.

The originality of the present case report is the length of the documented asymptomatic sinus pause. The asymptomatic three year follow up confirms the relation between the sinus pauses and the symptoms. One may thus be concerned about the length of symptomatic sinus pauses.

The present case also suggests that in patients with convulsive syncope, but no clear diagnosis of epilepsy, an implantable loop recorder may provide useful complementary diagnostic information.

G H Mairesse
B Marchand
drhghmairesse@skynet.be