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STUDIUM PRZYPADKU / *CLINICAL VIGNETTE*

Unsuccessful treatment of accelerated hypertension and persistent hyperkinetic state in a haemodialysed patient with high-output arteriovenous fistula

Nieskuteczne leczenie przyspieszonej fazy nadciśnienia tętniczego i utrzymującego się stanu krążenia hiperkinetycznego u pacjenta hemodializowanego z wysokoprzepływową przetoką tętniczo-żylną

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The case we describe concerns a rapid progression of heart failure (HF) in a patient with hypertension crises (HC), haemodialysed (HD) with the use of a high-output arteriovenous fistula (AVF). The 48-year-old male with a body mass index of 19.27 kg/m^2, renal insufficiency due to glomerulonephritis, treated with HD for two years (3 × 4.0 h/week), and with uncontrolled hypertension was admitted to the Military Institute of Medicine. On examination, reduced exercise tolerance, paroxysmal nocturnal dyspnoea and blood pressure (BP) 190/90 mm Hg were found. Laboratory tests revealed NT-pro B-type natriuretic peptide 302,009.0 pg/mL (n < 300.0). Echocardiography (ECHO) showed left ventricular (LV) hypertrophy (LV mass index [LVMI] 345.81 g/m^2), enlarged LV diastolic diameter [LVDD] 7.15 cm), with slightly decreased ejection fraction (EF 49%), Doppler signs of elevated cardiac output (CO 15.05 L/min; cardiac index (CI) 8.91 L/min/m^2), and pseudonormalised pattern of mitral inflow with elevated LV diastolic pressure (E/E' 31). In ultrasonographic examination of left shoulder AVF, an aneurysmal expansion of the lumen from 5.5 mm to 35 mm (Fig. 1), and significantly increased flow up to 2,532 mL/min (Fig. 2) were detected. Cardiopulmonary recirculation rate (CPR) was 0.17. Computed tomography excluded renal artery stenosis. In polysomnography, heavy central apnoea with an apnoea/hypopnoea index of 35.4/h was found. In 48-h ambulatory BP monitoring (ABPM), BP was 162/95 mm Hg. Treatment of sleep apnoea was implemented by a continuous positive airway pressure device, but this did not improve BP control. Upon discharge from hospital, the patient was overtreated with eight hypotensive drugs.

After a month, HC occurred (BP 220/120 mm Hg) associated with acute coronary syndrome without ST-segment elevation. Coronarography found non-obstructive atherosclerotic plaques. Because of the suspicion of hyperkinetic state related to a lack of BP control, AVF was narrowed by implanting a polytetrafluorethylene graft of 4 mm diameter (Fig. 3). Despite AVF flow being reduced to 1,400 mL/min, after two months another HC (BP 220/125 mm Hg) with pulmonary oedema was presented. The next HC took place three months later. Although ECHO showed features of LV hypertrophy regression (LVMI 335.62 g/m^2), and significant decreases of CO (8.16 L/min) and CI (4.86 L/min/m^2), a decrease of EF (40%) and an LV dilatation enlargement (LVDD 7.7 cm) were also observed. AVF flow increased up to 1,750 mL/min and significant CPR 0.21 was indicated. In 24-h ABPM, BP was 173/105 mm Hg. The hypotensive treatment used has been confirmed by experts and seems to be optimal and safe for the cardiovascular system. Probably, AVF banding was performed too late, and progression of HF and lack of BP control improvement were observed. In hyperkinetic AVF, blood flow exceeds 2,000 mL/min and CPR is over 0.2–0.3. Currently, in cases of difficulties in surgical AVF creation and the necessity of haemodialysotherapy termination as a result of a lack of vascular access, banding or closure of the AVF is a very rare event. However, in some cases, early ligation of AVF may be the only effective procedure, especially when there is rapid progression of HF.

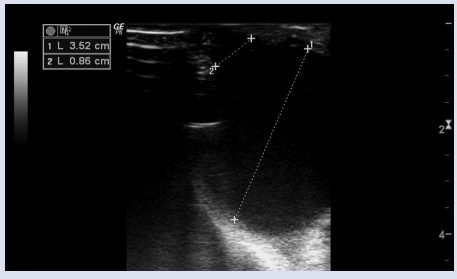

Figure 1. Aneurysmal dilatation of AVF with aneurysm

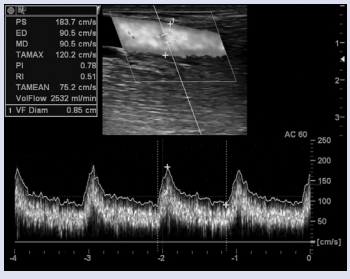

Figure 2. Flow measurement in hyperkinetic haemodialysis AVF

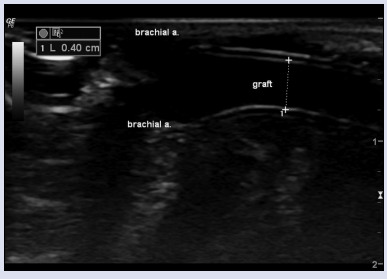

Figure 3. Narrowing polytetrafluoroethylene graft of haemodialysis AVF

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Conflict of interest: *none declared*