Apical rotating flow and right ventricular cerebral compression

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A 59-year-old male was admitted to the emergency room with signs of heart failure. The echocardiogram showed an extensive apical infarction with large mobile thrombi in the left ventricle. Doppler examination demonstrated apical rotating flow. Despite adequate anticoagulant therapy, the patient suffered a massive right-sided cerebral infarction leading to right ventricular cerebral compression. The thrombogenic risk of apical rotating flow and the need for anticoagulation are discussed.

KEYWORDS
Apical rotating flow; Thrombus; Cerebral infarction

A 59-year-old male known with diabetes type 2 and a family history of sudden cardiac death was admitted to the emergency room with abdominal pain, progressive dyspnoea d’effort, and orthopnoea which had started a week ago. Physical examination showed signs of left (LV)- and right ventricular heart failure. The ECG was abnormal with low voltages in the extremity leads and signs of an old anteroseptal myocardial infarction.

The echocardiogram (see Supplementary data online, Movie) showed an extensive apical infarct with large mobile thrombi in the LV and a severely depressed LV-ejection fraction. The continuous wave Doppler and colour M-mode Doppler signal (Figures 1 and 2) indicated permanent counter clockwise revolution of blood flow along the lateral wall towards the apex (continuous red colour in the colour M-mode tracing) and away from the apex along the ventricular septum.

A subsequent coronary angiogram indicated severe three vessel disease without any therapeutic options. The patient was then discharged with, among others, low molecular weight heparins and oral anticoagulation.

Despite adequate anticoagulation, the patient was readmitted to the neurology department 2 weeks later with a hemiparesis left due to a massive right-sided cerebral media infarction (CT-cerebrum, Figure 3). Dislodgement of the before mentioned mobile thrombi in the LV probably caused an occlusion of the arteria cerebri media.

Rapidly, oedema due to the infarct evolved to subfalcine and subtentorial herniation of the brain with compression of the right ventricle and midline shift to the left (Figure 3). Because of the very poor cardiac prognosis, no hemicraniectomy was performed and the patient died a few days later.

Apical rotating flow with low blood velocities creates a highly thrombogenic substrate.1 Protruding thrombus configuration and free mobility of the thrombus predict the risk of embolism and can be of value in weighing benefits and disadvantages of anticoagulant therapy.2

Supplementary data
Supplementary data are available at European Journal of Echocardiography online.

Conflict of interest: none declared.

References
Figure 1 Transthoracic echocardiogram: continuous wave Doppler signal demonstrating flow along the lateral wall towards the apex during diastole and, which is abnormal, also during systole (arrow).

Figure 2 Transthoracic echocardiogram: colour M-mode Doppler signal showing apical rotating flow (continuous red colour on tracing indicating permanent counter clockwise revolution of blood along the lateral wall towards the apex).

Figure 3 CT cerebrum: right-sided cerebral media infarction with subfalcine and subtentorial herniation and compression of the right ventricle with midline shift to the left.