Non-bacterial thrombotic endocarditis and subclinical myopericarditis in a patient with advanced rectal cancer

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DESCRIPTION

A 57-year-old man presented to the surgical assessment unit, with rectal pain. He had a 6-week history of painless rectal bleeding and 45 kg of weight loss. On examination, he had a circumferential exophytic rectal mass.

Blood results showed a microcytic anaemia with an inflammatory picture. A CT of the thorax, abdomen and pelvis, revealed an invasive rectal mass (figure 1) and foci of left renal infarction (figure 2). MRI staging was T4N2M0 (figure 3).

The preoperative ECG for a palliative de-functioning end-colostomy showed T-wave inversion in the anterolateral leads (figure 4).

Troponin-I was positive at 1437 ng/L without any clinical features of myocardial ischaemia. To investigate this, a transthoracic echocardiogram (TTE) was performed. This showed a 13×8 mm mobile vegetation on the anterior mitral valve (figure 5).

There were no stigmata of infective endocarditis and the modified Duke criteria were not satisfied as blood cultures were negative.1 Culture-negative endocarditis was thereafter excluded.

A cardiac MRI was subsequently performed, which revealed left ventricular myopericarditis (figure 6). This explained the positive troponin-I result. However, work up for this was unremarkable.

Differentials of non-infectious cardiac lesions were then considered. The hypercoagulable state in malignancy coupled with renal embolic phenomena made non-bacterial thrombotic endocarditis (NBTE) the definitive diagnosis (figure 7). Heparin reduces thrombus size and the incidence of
Figure 5  Initial transthoracic echocardiogram. (A) A 13×8 mm mobile mass on the anterior mitral valve leaflet (arrow), which lies between the left ventricle (LV) and left atrium (LA). (B) Large regurgitant jet of blood (blue) from the LV to the LA, indicative of mitral regurgitation.

Figure 6  Cardiac MRI showing global late gadolinium enhancement within the anterior, mid and apical left ventricular (LV) segments indicative of myopericarditis.

Figure 7  Pathophysiology of non-bacterial thrombotic endocarditis by applying Virchow’s Triad; the three factors of hypercoagulability, endothelial damage and abnormal blood flow in advanced malignancy contribute to the formation of a sterile vegetation on a normal cardiac valve (TNF, tumour necrosis factor; IL, interleukin).
Therefore, therapeutic low-molecular-weight heparin (LMWH) was started. There was little change in the patient’s bleeding. A TTE repeated 4 weeks later showed that the vegetation was non-existent with no evidence to suggest embolisation (Figure 8).

Postoperative histopathological analysis of the rectal biopsy showed well-differentiated rectal squamous cell carcinoma (Figure 9). The patient was discharged on lifelong LMWH.

**Competing interests** None declared.

**Patient consent** Obtained.

**Provenance and peer review** Not commissioned; externally peer reviewed.

**REFERENCES**


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**Learning points**

▸ Myopericarditis describes a primary pericarditic syndrome with minor myocardial involvement. Viral infections are the most common cause. Most cases present subclinically without a defined aetiological agent found. The diagnosis is based on elevated serum cardiac markers in the absence of another cause, and evidence of myocardial inflammation on cardiac MRI, or new left ventricular (LV) systolic dysfunction on echocardiography. There are a variety of atypical ECG changes possible. Treatment is largely conservative.

▸ When a valvular mass is found on a transthoracic echocardiogram, infective endocarditis (IE) must first be ruled out by applying the modified Duke criteria. If blood cultures remain persistently negative, culture-negative endocarditis (CNE) must then be ruled out by considering prior antibiotic exposure and intracellular fastidious bacteria—*Bartonella* spp, *Coxiella burnetti* and *Tropheryma whipplei*. If CNE and by extension systemic infection are excluded, this effectively indicates that the said mass is sterile.

▸ Non-bacterial thrombotic endocarditis (NBTE) is rare and is most commonly diagnosed on post mortem. It is characterised by the deposition of aseptic thrombi on normal cardiac valves. In 80% of cases, malignancy is the underlying aetiology. There are no pathognomonic features as patients are usually asymptomatic. The antemortem diagnosis is by first excluding IE and CNE, and, thereafter, by contextualising the cardiac vegetation against the patient’s background. In patients with advanced cancer, cardiac vegetations in the absence of systemic infection provide strong evidence to diagnose NBTE. Definitive treatment includes antitumour therapy and indefinite systemic anticoagulation with unfractionated or low-molecular-weight heparin. Vitamin K antagonists such as warfarin are less effective in preventing thromboembolic recurrence.

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**Figure 8** Transthoracic echocardiogram 4 weeks after the initiation of low-molecular-weight heparin. (A) Non-existence of the previous thrombus with a normally functioning mitral valve. (B) Mild mitral regurgitation (blue).

**Figure 9** Rectal biopsy showing a well-differentiated squamous cell carcinoma. 1: Normal epidermis composed of uniform and regularly spaced squamous cells; 2: infiltrative island of squamous cells with enlarged atypical nuclei, mitotic activity and disorder indicative of squamous cell carcinoma.