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ACUTE FULMINANT NECROTISING LYMPHOCYTIC MYOCARDITIS IN A PATIENT WITH MIXED CONNECTIVE TISSUE DISEASE: A RAPID CLINICAL RESPONSE TO IMMUNOSUPPRESSION

Editor

Myocarditis is an uncommon condition encompassing a spectrum from asymptomatic cases to fulminant heart failure. Acute fulminant myocarditis is characterised by severe haemodynamic compromise often necessitating circulatory support. The diagnosis and management of myocarditis remains challenging with uncertainty surrounding the role of immunosuppression therapy. We describe a case of biopsy-proven acute necrotising lymphocytic myocarditis which responded rapidly to steroids, mycophenolate and immunoglobulins.

A 53 year old male was admitted to a District General Hospital with a 4-day history of chest pain and ‘flu-like symptoms. He had a history of mixed connective tissue disease (MCTD). Physical examination revealed sinus tachycardia (126bpm) and mild pulmonary oedema.

The ECG on admission showed sinus rhythm with Q waves in the anterior chest leads and T wave inversion in leads I, aVL and V3-V6. High-sensitivity troponin T (hsTNT) was 5220ng/L and the C - reactive protein (CRP) was 328mg/L. Within 24 hours he developed cardiogenic shock with severe pulmonary oedema, left bundle branch block and severe left ventricular systolic dysfunction (LVSD). Transfer was arranged due to clinical instability. At cardiac catheterisation, the aortic pressure was 83/55mmHg with a left ventricular end-diastolic pressure of 35mmHg. Coronary angiography showed no obstructive disease and an intra-aortic balloon pump (IABP) was sited.

With the progressing ECG abnormalities, echocardiographic findings and rising biomarkers, a diagnosis of acute myocarditis was made. Urgent right ventricular endomyocardial biopsies were undertaken with frozen section analysis confirmed acute necrotising myocarditis. There was no evidence of vasculitis and giant cells were absent on histopathology (Figures 1 & 2). Immunohistochemistry was negative for Epstein Barr virus (EBV) and parvovirus. Viral polymerase chain reaction (PCR) was weakly positive for both. Screening for hepatitis B, C, cytomegalovirus (CMV), erythrovirus B19, streptococcus pneumoniae and picornavirus was negative.

Oral prednisolone (40mg OD) and mycophenolate mofetil (500mg BID) were commenced on rheumatological advice. A total dose of 300g of Human Immunoglobulin [Privigen® (CSL Behring, PA, US)] was administered over 5 days.

A rapid clinical improvement ensued, facilitating IABP removal and discontinuation of inotropes after 72 hours. Standard heart failure therapy was commenced. Repeat echocardiography by day 9 showed only mild global left ventricular systolic impairment. Temporary interruption in mycophenolate therapy occurred due to shingles (treated with Ganciclovir). He was discharged on day 15.

DISCUSSION

The diagnosis of myocarditis should be considered in any patient presenting with acute heart failure. Non-invasive imaging modalities (ECHO and Cardiac MR) are helpful in establishing the diagnosis. Ultimately, myocarditis is a histopathological diagnosis. Multiple endomyocardial biopsy samples are required as sampling error can occur in the setting.

Fig 1. Histology specimen from the endomyocardial biopsy demonstrating myocardium with a dense infiltrate of inflammatory cells (L)-(dark blue nuclei) separating the myocytes (M)
of patchy disease. The most common histopathological form of acute myocarditis is a lymphocytic pattern. The mainstay of treatment in acute myocarditis is inotropic agents and circulatory support. The efficacy of intravenous immunoglobulin (Ig) and immunosuppression remains unproven. Studies have demonstrated mortality benefits with early Ig and steroid administration.

The authors have no conflicts of interest

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MOLECULAR PROFILING OF GLIOMAS - TIME FOR A REGIONAL SERVICE.

Editor,

Gliomas form a heterogeneous group of intrinsic primary brain neoplasms in terms of pathological and clinical features. Low-grade (WHO grade II) gliomas (e.g. astrocytomas and oligodendrogliomas) inevitably recur and progress to higher grade (WHO III-IV) anaplastic tumours. Although they have traditionally been classified using histological criteria, there is increasing evidence that gliomas can be further subtyped based on molecular profile which can predict prognosis and response to treatment.

Longterm follow-up data has demonstrated a significant survival advantage with anaplastic oligodendroglioma (AO)/oligoastrocytoma (AOA) tumours co-deleted for chromosomal arms 1p and 19q following combined chemo-radiotherapy compared with non-1p19q codeleted cases. These findings validated in both European (EORTC 26951) and North American trials (RTOG 9402) have meant that 1p19q status predicts post-surgical treatment. The current standard of care is that co-deleted cases receive chemoradiotherapy while non-deleted cases receive only radiotherapy due to the lack of efficacy of combined treatment in this group.

Prior to this recent change in practice, all patients with anaplastic oligodendrogial tumours were treated with radiotherapy upfront and received chemotherapy (typically procarbazine, lomustine and vincristine) on relapse. The aim of this regional retrospective study was to establish as a baseline NI clinical outcomes using this pre-1p 19q stratification as a comparator for future outcomes studies.

RESULTS

Clinical, pathological and molecular profile data (available in 20 cases) were analysed in 58 consecutive patients with a histological diagnosis of anaplastic oligodendrogial tumour diagnosed over a five-year period (2007-2012). The median survival of all patients was found to be 53 months (95% CI 22-84 months). The median survival of patients with AO (n=38) was found to be 81 months (95% CI 37-125 months). The median survival of patients with AOA (n=20) was found to be 19 months (95% CI 14 to 25 months). Log-rank analysis confirmed that AO patients had a significantly longer median survival than those with AOA tumours (p=0.023) comparable with other reports (Fig. 1).