Immunosupression for patients of endocarditis with glomerulonephritis

Sir,

The case report on post-infectious glomerulonephritis following infective endocarditis, which was amenable to immunosuppression, sheds light on an unusual but highly significant aspect in the management of infective endocarditis.[1] Immunosuppression in infective endocarditis is usually not indicated, and if given without adequate antibiotics, may lead to disastrous results. So, why use immunosuppression in such patients? In this letter, we would like to reiterate those clues that would prompt us for the use of immunosuppression in addition to antibiotics, in a case of subacute bacterial endocarditis (SBE) with renal disease.

Subacute bacterial endocarditis, possibly due to circulating immune complexes, can cause a host of immunological problems. Prolonged infections can cause serological abnormalities, just as hepatitis B is associated with polyarteritis nodosa and hepatitis C with cryoglobulinemia. Infective endocarditis has been associated with positive cytoplasmic anti-neutrophil cytoplasmic antibody (c-ANCA) of the anti proteinase-3(PR-3) type which may mimic ANCA associated vasculitis with endocardial involvement.[2-4] c-ANCA positivity is considered pathogenic and can also cause features resembling granulomatosis with polyangitis (Wegener's disease), presenting with proliferative glomerulonephritis, lung nodules and pulmonary angiitis, along with rapid clinical deterioration despite appropriate antibiotics.[2,3,5] Other antibodies which may be found are low levels of rheumatoid factor, cryoglobulins, anti-nuclear antibodies and anti-cardiolipin antibodies. Such patients may not respond to antibiotics alone. As a corollary, an apparent case of ANCA associated vasculitis may actually be an infective endocarditis, which in certain instances is usually misdiagnosed due to an absence of a significant heart murmur. Early initiation of immunosuppressants in such case may cause catastrophic sepsis. Therefore, determining the underlying cause of ANCA positivity is essential.[2]

Histological examination of involved organs in infective endocarditis, with multiple autoantibodies formation, is necessary to determine the treatment regime. It has been suggested that post-infectious glomerulonephritis, and the presence of low titer of PR3-ANCA (eg., less than 25 IU/ml), usually responds to appropriate antibiotics alone. Alternatively, high titer of PR3-ANCA (eg., more than 50 IU/ml) or failure to respond to antibiotics within a usual period of time usually warrant antibiotics with corticosteroids or immunosuppressants.[2,3] Similarly, pauci-immune crescentic glomerulonephritis or vasculitis may need immunosuppressants along with antibiotics. Interstitial nephritis may be drug induced, especially β-lactams or ANCA associated, and usually requires immunosuppression. Renal disease with anti-phospholipid positivity may require warfarin.[2,3]

Another indication of immunosuppression, apart from infective endocarditis with ANCA positivity failing to respond to antibiotics alone, is culture negative endocarditis secondary to a vasculitis, the echocardiographic picture of which closely resembles SBE.[2]

Therefore, in a case of SBE with worsening renal function, tissue biopsy is essential, along with evaluation for autoantibodies like PR3-ANCA. Secondly, in a case of endocarditis with ANCA positivity, it is necessary to determine the cause of endocarditis as vasculitis or infection, as the latter would probably require immunosuppression along with antibiotics.

K. Chatterjee, C. Sen[1]

Department of Medicine, Dr. Ram Manohar Lohia Hospital and Post Graduate Institute of Medical Education and Research, Guru Gobind Singh Indraprastha University, New Delhi, [1]Medicine, Medical College and Hospital, Kolkata, West Bengal, India

Address for correspondence:
Dr. Chetana Sen,
B-7/11, Diamond Park, PO-Joka,
Kolkata-700 104, West Bengal, India.
E-mail: chetana87@gmail.com

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