Renal lesions in infective endocarditis (an autopsy study of 55 cases).

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INTRODUCTION

The association between bacterial endocarditis and nephritis was established 50 years ago,[2],[4],[14],[19] Initial reports are autopsy studies before the discovery of antibiotic treatment. These emphasized the embolic events from the heart resulting in renal infarcts and microabscess formation[3],[7] Subsequently, it was thought that renal failure was generally associated with diffuse glomerulo-nephritis and rarely with infarcts and abscesses. [10],[22],[23]

Both types of lesions have been noted by most workers. Fifty five autopsied cases of infective endocarditis in the 5 year period from 1977-1981 at the K.E.M. Hospital are reviewed to evaluate the incidence of renal involvement and note the types of renal lesions in these cases.
MATERIAL AND METHODS

Fifty five cases of infective endocarditis were encountered in the 5 year study period from 1977 to 1981, 45 of these were of bacterial endocarditis (BE). It was complicating rheumatic heart disease in 34 cases, congenital heart disease in 9 cases and prosthetic valves in 3 cases. In 2 cases, it had affected previously normal valves. There were 10 cases of fungal endocarditis (FE) complicating prosthetic valves. In 8 cases, it was caused by aspergillus and in 2 cases by Candida albicans. Fungus was identified by histomorphology in sections from vegetations on the cardiac valves. Clinical data and autopsy protocols were scrutinised for symptoms like haematuria, azotemia, albuminuria and gross pathological findings in the kidneys. Histological sections stained with HE, PAS, fibrin, PASM were reviewed.

RESULTS

Fifty five cases of infective endocarditis were studied. There were 40 males and 15 females. Age range varied from 6 years to 50 years. M : F ratio was 2.6 : 1.

Clinical features pointing to renal disease were noted in only 10 cases. Haematuria was noted in 2 cases, azotemia in 5 cases and albuminuria in 3 cases.

Gross pathology

The kidneys appeared flea-bitten in 4 cases-2 showed multiple tiny abscesses. Fresh infarcts were noted in 7 cases while old infarcts in 4. In the remaining cases, the kidneys were unremarkable on gross examination.

Spectrum of histological lesions [Table 1]

Old and recent infarcts seen on gross (4 + 17 cases respectively) were confirmed on microscopy.

Microabscesses were seen in the interstitum in 5 cases and hyaline thrombi were noted in glomerular capillaries in 2 cases.

Focal proliferative glomerulonephritis was noted in 11 cases. (BE 10 and FE 1). In 7 cases, glomerulonephritis was associated with crescents. In 3 cases, there were segmental areas of sclerosis where a localised area of fibrous or hyaline material was seen at the periphery of the lobules which were fused together. There were adhesions of this material with the Bowman’s capsule. Diffuse mesangial proliferation of moderate degree with patchy basement membrane thickening was noted in 10 cases. (9 B.E., 1 F.E). Neutrophilic infiltration was not seen in any of these cases. In none of the cases of fungal endocarditis, fungi were noted in the kidneys. There was no lesion noted in 10 cases of B.E. and 5 of F.E.

Correlation of pathological features with azotemia

Five cases had raised BUN varying from 40 to 132 mg%. In one of these cases, multiple
DISCUSSION

It is generally agreed that 3 types of renal lesions may occur in the course of bacterial endocarditis; (1) renal infarcts, (2) focal 'embolic' glomerulonephritis (3) acute diffuse glomerulonephritis. Bell's[7] studies recorded 52% subacute endocarditis and 7% of acute endocarditis showing embolic glomerulonephritis, where as, diffuse lesions were seen in 64% of 108 patients with subacute endocarditis and 28% of 58 patients with acute endocarditis. Embolic lesions were thought to be due to small bacterial emboli that destroyed portions of glomerular capillary loops. However, Allen[1] summarised the reasons for rejecting the embolic theory of pathogenesis of the lesions and agreed with Dongoscope[15] who considered these lesions to be of allergic nature. Acute endocarditis with infective blood cultures giving rise to antigen excess led to diffuse proliferative glomerulonephritis.

Immunofluorescence and electron microscopic studies of Gutman et al[10] have shown subepithelial deposits of complexes. In the pre-antibiotic era, death from renal failure associated with bacterial endocarditis occurred in patients with sterile cultures, so called bacteria free stage. Such patients had chronic illness sometimes lasting decades, perhaps these patients had prolonged circulation of immune complexes formed in antibody excess which could account for sterile cultures[25] Gutman et al[10] have shown evidence of immune complexes both in focal and diffuse glomerulonephritis. Several observers[6],[10],[12],[18] have demonstrated low serum complement levels in cases with biopsy-confirmed renal abnormalities. Secondly, circulating immune complexes have been demonstrated in several patients with SBE.[5],[13],[17],[20] It was thus thought that depending on the type of lesion, acute or subacute endocarditis, the renal lesions resembled those in acute or chronic serum sickness and probably reflect the differences in the size and composition of immune complexes. However, Boulton-Jones et al[8] found that of 5 cases of SBE with renal involvement that they studied, 3 had focal while had diffuse proliferative glomerulonephritis.[4] Surprisingly in both these latter cases, no deposits were seen on immunofluorescence, though they were seen in all the 3 cases of focal endocarditis. Thus there is no agreement on the pathogenesis of these lesions and the inter relationship between them.

In our material, being a retrospective study, immunofluorescence was not possible. Hence on morphological grounds we have divided the lesions as (1) embolic eg. infarcts, abscesses, focal proliferative lesions, with or without crescents and focal segmental areas of sclerosis (the latter represents the healing stage of necrotic focus which may have been there) and (2) diffuse proliferative glomerulonephritis.

Bell[7] reported diffuse lesions in 60% of cases Christian et al[9] 80% and Heptinstall[11] in 17% of cases. In the present series, it was found in 16.66% of cases. Embolic lesions still formed the major bulk of lesions seen in our material [Table 1]. There were no renal involvement seen in 27.2% of cases.

In our material we had 10 cases of fungal endocarditis, 8 cases by aspergillus and the remaining 2 caused by Candida. Fungus was identified by its morphologic features in the vegetations on the valves. We found one case with focal and one with diffuse proliferative
nephritis in 2 cases of fungal endocarditis caused by aspergillus. In none of the cases, however, the fungus was identified in the kidney lesions.

Fungal endocarditis is a rare disease. There has been an increase in the incidence of this disease that has corresponded with open heart surgery, especially in prosthetic valve surgery.[24] Fungal endocarditis is also known in heroin addicts.[16] The commonest fungi encountered are candida and aspergillus. Renal involvement is usually embolic and has been noted in 55 to 70% of cases and most of these lesions have been reported in patients with endocarditis due to aspergillus or candida species.[21] No focal or diffuse renal lesions have been reported in endocarditis due to cryptococcosis, blastomycosis, dermatides, histoplasmosis or mucor mycosis, though several of these appear to be good antigens.[21]

Clinical picture of renal involvement in endocarditis is generally overshadowed by cardiac features and the effects of infection. Haematuria and proteinuria are common. Microscopic hematuria may occur with infarcts or 'embolic' lesions.

In our series, though infarcts were noted in totally 21 cases, haematuria was noted in only 2 cases, proteinuria in 3 cases. Uraemia is rare and was described in 25-30% of cases in the preantibiotic era, but with the advent of antibiotics there has been a decline and the incidence is now in the region of 10%. Bachr[5] felt that renal function was rarely altered in the presence of focal glomerular lesions because the uninvolved portions of the damaged glomeruli remained normal, leaving enough healthy glomerular tissue to maintain normal function. In our material raised BUN was noted only in 5 cases out of 55 cases giving an incidence of 9.9%. Surprisingly azotemia was not seen in any of the cases with diffuse glomerulonephritis.

In summary, 55 cases of infective endocarditis (45 BE, 10 FE) were studied. Renal symptoms were seen in only 5 cases and azotemia was noted in 5. None of the cases with diffuse proliferative lesions showed evidence of azotemia. Aspergillus endocarditis in 2 cases was associated with focal glomerulonephritis in one and diffuse glomerulonephritis in the other.

References