P-25

Pathology of Chronic Allograft Arteriopathy

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Chronic rejection has been defined in renal function starting at 3 month after transplantation. Neither clinical nor pathologic feature individually are sufficient for a definite diagnosis. However, the arterial lesions particularly permit definite diagnosis of chronic rejection by morphology alone. As early as 1 month after transplantation, graft arteries of all size can develop severe intimal proliferation and narrowing, while are most prominent in the larger arteries, but extends from interlobular to main artery, characterized by pronounced intimal cencentric fibrous thickening with proliferation of spindle shaped smooth muscle cells, and subendothelial ring like mononuclear cell infiltrations, which are strikingly different from atherosclerosis. Also seen are infiltrations of foamy macrophages, characteristically along the internal elastica. Disruption and irregular duplication of the elastic lamina are present. The stage 1 lesion, endarteritis (TypeII acute rejection) lacks matrix formation, which is believed to be T-cell mediated and directed primarily at the endothelium. Stage 2 lesion have evidence of matrix production and accumulation of myocytes. Stage 3 lesion have fibrous intima with scanty inflammatory cells, due to humoral antibody. The stage 2 & 3 have been termed "chronic allograft arteriopathy"