



2. Higher-power photomicrograph of Fig 1 to show histiocytic macrophages with "foamy" cytoplasm in intima and media. Note also prominence of plasma cells (arrows) in leukocytic exudate in artery wall (hematoxylin and eosin, $\times 400$).

Comment

Microscopic abnormalities occurring in the renal vessels of rejected human transplanted kidneys have been well described.¹⁻³ These changes seem to be confined to the interlobular, arcuate, and interlobar arteries and arterioles, and vary with the length of survival and with vessel size. In the early stages, these lesions consist of a diffuse inflammatory cellular infiltrate made up chiefly of plasma cells and lymphocytes, necrosis of the media, and occasional fibrinoid necrosis of the vessel wall with fibrinous deposit on the intima.³ In patients surviving a longer period, 25 to 28 days, the vascular damage is more pronounced and more vessels are involved, with the main renal artery spared. At this time the changes consist of medial necrosis, fibrinoid necrosis, and intimal thickening. In addition, the intima contains large vacuolated phagocytic cells which are distinctive, possibly plasma-cell derivatives, and peculiar to this type of rejection phenomenon.²

In the ureteral arteries of the two patients herein reported, severe vascular abnormalities were noted. These lesions are similar to those previously described in renal arteries in transplant rejection.¹⁻³ Rejection of the ureter may contribute to the physiological failure of the renal transplant.

The ureteral vascular changes followed closely after transplantation and consisted of focal round-cell infiltration of the vessel wall, fibrinoid degeneration of the intima, and focal medial necrosis (case 1). In the second patient, who survived for a longer period of time (20 days, case 2), the vascular abnormalities in the ureter consisted of in-

timal proliferation and degeneration, focal histiocytic infiltration of the media and intima, and medial necrosis. Infiltration of the artery wall by histiocytes with foamy cytoplasm (Fig 1) appears to be unique to the human renal transplantation rejection mechanism.² These cells are large, have vacuolated cytoplasm which does not stain with the PAS reagent, and have small, ovoid, eccentrically placed nuclei. Their origin may be from plasma cells.²

Summary

Degenerative changes were present in the ureteral arteries in two patients who died after having undergone renal transplantation. These abnormalities are similar to those present in arteries in the kidney. The renal vascular rejection process has been previously described, whereas vascular rejection in the human ureter has not. Round-cell infiltration and focal necrosis of the media is present in ureteral arteries in early rejection. Later findings include medial necrosis and infiltration of the media and intima with large vacuolated histiocytes and leukocytes. The histiocytes appear to be peculiar to the homotransplant rejection phenomenon.

Generic and Trade Names of Drugs

Azathioprine—Imuran.

Prednisone—Deltasone, Deltra, Meticorten, Paracort.

Chloramphenicol—Chloromycetin.

References

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Benign Asbestos Pleurisy

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THE classical picture of asbestosis has been divided into several stages.¹ At first, there is a latent period of ten or more years during which asbestos bodies are formed around the asbestos fibers. During this time the patient is completely asymptomatic, and results of his chest x-ray are normal. This stage is followed by the gradual appearance of respiratory symptoms in the form of dyspnea, cough, expectoration, and wheezing; the roentgenologist finds bilater-

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al pulmonary fibrosis. As the disease advances, systemic complaints are added, such as fatigue, malaise, weakness, anorexia, and weight loss. Final-

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