6.10 Systemic Diseases and the Kidney

**FIGURE 6-18**
Cystoscopic appearances of different bladder lesions associated with Schistosoma haematobium infection. **A**, Bilharzial (schistosomal) pseudotubercles. **B**, Bilharzial submucous mass covered by pseudotubercles. **C**, Bilharzial ulcer surrounded by pseudotubercles. **D**, Bilharzial ulcer surrounded by sandy patches. (Courtesy of N. Makar, M.D.)

**FIGURE 6-19**
Postmortem specimen showing advanced bilharzial involvement of the urinary tract. Note the dirty bladder mucosa, fibrosed muscle layer, and neoplastic growth (histologically a squamous cell carcinoma) cut through transversely. The ureters are dilated, with a clear stricture at the lower end of the right ureter. Also seen in this patient are bilateral hydroureters with submucous cystic lesions (bilharzial ureteritis cystica). The kidneys show considerable scarring, with the right kidney also showing chronic back pressure changes.

**FIGURE 6-20**
Filariosis of the abdominal lymphatics. Lymphangiogram shows the dilated retroperitoneal lymphatics in a patient with filarial chyluria.
FIGURE 6-21
The pathogenesis of falciparum malarial renal complications. Note the infection triggers two initially independent pathways: red cell parasitization and monocyte activation. These subsequently interact, as the infected red cells express abnormal proteins that induce an immune reaction by their own right, in addition to providing sticky points (knobs) for clumping and adherence to platelets and capillary endothelium. TNF-α released from the activated monocytes shares in the endothelial activation. As both pathways proceed and interact, a variety of renal complications develop, including acute tubular necrosis, acute interstitial nephritis and acute glomerulonephritis. B—B-lymphocyte; CD8—cytotoxic T cell; CIC—circulating immune complexes; TH—T-helper cells (1 and 2); TNF-α—tumor necrosis factor-α.

FIGURE 6-22
Erythrocyte knobs in a patient with falciparum malaria [43]. These erythrocyte knobs contain novel proteins, mainly Plasmodium falciparum erythrocyte membrane protein (PFEMP), histidine-rich protein 1, and histidine-rich protein 2, that are synthesized under the influence of the DNA of the parasite [44–46]. These proteins constitute the sticky points (arrows) by which parasitized erythrocytes aggregate and adhere to blood platelets and endothelial cells [47,48]. EN—electron microphotograph. (Magnification × 12,000.)

FIGURE 6-23
Renal lesions in a patient with falciparum malaria. A, Proliferative and exudative glomerulonephritis, an immune-complex-mediated lesion that may lead to an acute nephritic syndrome, which usually is reversible by antimalarial treatment. (Hematoxylin-eosin stain × 175.) B, Acute tubular necrosis (ATN) associated with interstitial mononuclear cell infiltration. ATN is seen in 1% to 4% of patients with falciparum malaria and in up to 60% of those with malignant malaria. (Hematoxylin-eosin stain × 200.) (Continued on next page)
C. Subendothelial and mesangial malarial antigen deposits seen on immunofluorescence. Often, complement 3, immunoglobulins M and G, and fibrinogen also are seen. (Hematoxylin-eosin stain x 200.)

The broad lines of the immune response to parasitic infections. Note the pivotal role of the monocyte, activated by exposure to parasitic antigens, in stimulating both T-helper 1 (TH1) and T-helper 2 (TH2) cells. The different cytokine mediators and parasite elimination mechanisms are shown. B—B-lymphocyte; γ-IFN—γ-interferon; CIC—circulating immune complexes; GM-CSF—granulocyte-macrophage colony-stimulating factor; Ig—immunoglobulin; IL—interleukin.

The T-helper 1–T-helper 2 (TH1-TH2) cell balance that determines the clinical expression of different parasitic nephropathies. TH1 predominance leads to either reversible acute proliferative glomerulonephritis or acute interstitial nephritis. TH2 predominance tends to lessen the severity of the lesions and may lead to chronic glomerulonephritis in the presence of copathogenic factors such as concomitant infection (malaria, schistosomiasis), autoimmunity (malaria, filariasis, schistosomiasis), or immunoglobulin A (IgA) switching (Schistosoma mansoni) [7, 9, 49–52]. CD4—T-helper cells; CD8—cytotoxic cells; γ-INF—γ-interferon; IL—interleukin.