Further Section


Quiz of the Month

Answer to Question 1
The renal biopsy showed normal glomeruli, interstitial edema with an intense inflammatory infiltrate consisting predominately of mononuclear cells and plasma cells with about 5% eosinophils, and no vascular lesions. The biopsy was consistent with acute interstitial nephritis (AIN). The combination of fever, skin rash, and acute deterioration of renal function strongly suggests AIN. Rapid deterioration of renal function due to progressive systemic sclerosis is usually associated with significant hypertension and retinopathy, neither were present in this patient. The negative history for toxins is against diagnosis of acute tubular necrosis. The renal biopsy was done to determine the continuing need for steroids which were started for her pulmonary problem. Both hydrochlorothiazide and fenoprofen were held at admission and were not restarted. The steroids were continued and the BUN and creatinine returned to normal in 4 weeks.

Both hydrochlorothiazide and fenoprofen have been associated with AIN, the latter drug also having been associated with the nephrotic syndrome, papillary necrosis, and acute renal failure secondary to underperfusion. The reactions seen with both drugs can occur with variable times of exposure and five days to five weeks after initial exposure, even if the drug is stopped. Only a minority of patients with this type of AIN present with the classic triad of fever, rash, and eosinophilia. Eosino-philuria is said to be pathognomonic of AIN. The mechanism of AIN is unclear but a recent report demonstrating that the cells in the interstitial infiltrate of AIN secondary to fenoprofen stain exclusively for T lymphocyte markers suggests an immunologic role. The prognosis for recovery of renal function is good and there is suggestive evidence that steroid hasten recovery of renal function.

References

Answer to Question 2
The clinical impression of the physicians caring for this patient was exacerbation of congestive heart failure with probable thromboembolism of the renal arteries from a mural thrombus. This should not have been a high diagnostic consideration because such patients develop sudden deterioration of renal function without significant proteinuria, often with hematuria (usually
microscopic) and frequently have evidence of thromboembolic disease at other sites. The first procedure done was a renal arteriogram which showed normal renal arteriolar vasculature but slow venous drainage. For this reason, an inferior vena cava study was done which showed bilateral renal vein thrombosis. The following day a renal biopsy was done showing membranous glomerulonephritis. It should be noted that this patient, with renal disease, proteinuria, congestive heart failure, and effective arterial blood volume contraction, was at considerable risk of developing dye-induced acute renal failure. Fortunately, he did not develop this complication.

This is a case of membranous nephropathy with nephrotic syndrome developing in a patient with underlying congestive heart failure. The patient’s proteinuria was initially ascribed to his congestive heart failure. Though proteinuria can occur with congestive heart failure, it is almost always mild and rarely occurs to the degree seen in

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this patient. Membranous nephropathy developing in a patient of this age has a significant association with underlying malignancy. Chronic hepatitis B infection and underlying systemic lupus erythematosus must be ruled out before labelling the disease as being idiopathic, the assigned diagnosis in our patient. These patients have a variable course with about equal numbers having spontaneous remissions, progressing to end stage renal failure and chronic proteinuria. Patients with membranous nephropathy and the nephrotic syndrome have a hypercoagulable state; renal vein thrombosis (in addition to deep leg vein thrombosis) is a well-described complication. Renal vein thrombosis should be in the differential when working up a nephrotic patient with sudden deterioration of renal function, especially when this is associated with an increase in proteinuria and/or sterile pyuria. Effective arterial blood volume contraction, present in this patient, may predispose to this complication in adults as it does in children. The recent literature suggests that long-term anticoagulants can improve renal function in patients with acute onset renal vein thrombosis but not in those of chronic duration. Our patient received heparin then coumadin therapy with return of his renal function to baseline and resolution of his renal vein thrombosis when the inferior vena cava dye study was repeated 4 weeks later. The coumadin was continued for 6 months and stopped empirically after 6 months of negative clinical follow-up. His proteinuria resolved with 3 months of steroid therapy, after which the steroids were tapered and stopped.

References
L. Llach
F.; Arieff
A.J.; Massry
Llach
F.; Papper
S.; Massry