invasion, often with spreading to the contralateral kidney, which may cause bilateral renal vein occlusion.

The nephrotic syndrome is associated with a risk for venous thrombosis throughout the circulation, including RVT. The RVT risk in patients with nephrotic syndrome correlates with severity of proteinuria and hypoalbuminemia; patients with a serum albumin concentration of less than 2 g/dL are at particular risk. Some studies have documented an incidence of RVT as high as 30% among patients with nephrotic syndrome, but most cases are not clinically apparent. Patients with membranous nephropathy seem to be at greatest risk for RVT for reasons that are not known, but RVT can also occur with focal segmental glomerular sclerosis, membranoproliferative glomerulonephritis, minimal change disease, and diabetic kidney disease. Hypercoagulability is thought to result from loss of the antithrombotic protein antithrombin III in urine, although other factors such as increased procoagulant factors and platelet activation may also be involved.

RVT may manifest with symptoms attributable to renal cell carcinoma, such as flank pain, gross hematuria, nausea, anorexia, or lower extremity swelling. In male patents, left renal vein occlusion may cause a left varicocele, a result of the venous drainage of the left gonadal vein. In patients without a malignancy, symptoms of RVT depend on the acuity of the thrombosis. Acute, complete thrombosis may manifest with hematuria, flank pain, abdominal distention, and acute renal failure. RVT in adults usually occurs gradually because of collateral venous drainage return; in this setting, symptoms of AKI are uncommon, although proteinuria and creatinine levels may be mildly elevated/

Because patients often do not have symptoms, RVT is likely more common than reported in the literature. Some have suggested CT screening of asymptomatic, high-risk patients, particularly those with membranous nephropathy and severe proteinuria and hypoalbuminemia.

The standard method for diagnosis is renal venography, but because it has a risk of clot dislodgment, bleeding, and iodinated contrast, less invasive methods are commonly used. Contrast-enhanced CT venography appears to have a relatively high sensitivity and specificity, although it carries some risk for contrast nephropathy. MRI using gadolinium-based contrast or time-of-flight sequencing without contrast may also be useful. Renal Doppler ultrasound is useful, but it is operator dependent and has lower sensitivity than CT venography.

Treatment with systemic anticoagulation is recommended in the absence of contraindications. Most clinicians maintain anticoagulation for 6 to 9 months, similar to the approach for non-renal deep vein thrombosis and pulmonary embolism. The long-term recurrence risk is low if the underlying predisposition is successfully treated, and patients are unlikely to require indefinite anticoagulation. Direct intravenous thrombolysis or operative thrombectomy may be considered in severe cases, particularly if the RVT is a source of pulmonary emboli or is causing AKI. Prophylactic anticoagulation in high-risk patients, such as those with severe membranous nephropathy (serum albumin concentration <2.5 g/dL) should be considered for appropriate candidates.

For a deeper discussion on this topic, please see Chapter 125, "Vascular Disorders of the Kidney," in Goldman-Cecil Medicine, *25th Edition.*

SUGGESTED READINGS

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