Jung-Lin Chang is a 23-year-old graduate student in biology. He presents at the university health center, brown and foamy urine. The physician there admits him to the infirmary and orders a throat culture, ASO titer, CBC, BUN, serum creatinine, and urinalysis.

**ASSESSMENT**
Connie King, the nurse admitting Mr. Chang, notes that his history is essentially negative for past kidney or urinary problems. He relates having had a “pretty bad” sore throat a couple of weeks before admission. However, it was during midterms, so he took a few antibiotics he had from a previous bout of strep throat, increased his fluids, and did not see a doctor. The sore throat resolved, and he felt well until noticing the change in his urine. He admits that his eyes seemed a little puffy, but he thought this was due to lack of sleep and fatigue. He has eaten little the past 2 days, but was not alarmed because his food intake is irregular most of the time.

Physical assessment findings include: T 98.8° F (37.1° C) PO, P 98, R 18, and BP 136/90. Weight 165 pounds (75 kg), up from his normal of 160 (72.5 kg). Moderate periorbital edema and edema of hands and fingers noted.

Throat culture is negative, but the ASO titer is high. CBC essentially normal. BUN 42 mg/dL, serum creatinine 2.1 mg/dL. Urinalysis reveals the presence of protein, red blood cells, and RBC casts. A subsequent 24-hour urine protein analysis shows 1025 mg of protein (normal 30 to 150 mg/24 hours).

The physician diagnoses acute poststreptococcal glomerulonephritis and places Mr. Chang on bed rest with bathroom privileges. He orders fluid restriction (1200 mL/day) and a restricted sodium and protein diet.

**DIAGNOSIS**
Ms. King develops the following nursing diagnoses for Mr. Chang.

- Excess fluid volume related to plasma protein deficit and sodium and water retention
- Risk for imbalanced nutrition: Less than body requirements related to anorexia
- Anxiety related to prescribed activity restriction
- Risk for ineffective therapeutic regimen management related to lack of information about glomerulonephritis and treatment

**EXPECTED OUTCOMES**
The expected outcomes are that Mr. Chang will:

- Maintain blood pressure within normal limits.
- Return to usual weight with no evidence of edema.
- Consume adequate calories following prescribed dietary limitations.
- Verbalize reduced anxiety regarding ability to continue studies.
- Demonstrate an understanding of acute glomerulonephritis and prescribed treatment regimen.

**PRACTICE CONNECTIONS**
Provide instructions for the client and family, including the following topics.

- Information about the disease and the prognosis
- Prescribed treatment, including activity and diet restrictions; the use and potential effects, both beneficial and adverse, of all medications
- Risks, manifestations, prevention, and management of complications such as edema and infection
- Signs, symptoms, and implications of improving or declining renal function
- Measures to prevent further kidney damage, such as nephrotoxic drugs to avoid

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**Home Care**
Glomerular disorders may be self-limited or progressive. In either case, the course is lengthy, ranging from months to years. Self-management is essential. Provide instructions for the client and family, including the following topics.

- Information about the disease and the prognosis
- Prescribed treatment, including activity and diet restrictions; the use and potential effects, both beneficial and adverse, of all medications
- Risks, manifestations, prevention, and management of complications such as edema and infection
- Signs, symptoms, and implications of improving or declining renal function
- Measures to prevent further kidney damage, such as nephrotoxic drugs to avoid
THE CLIENT WITH A VASCULAR
KIDNEY DISORDER

Renal function is dependent on an adequate supply of blood. Blood supports renal cell metabolism and is vital to kidney function, the nephron in particular. The kidney can regulate fluid, electrolyte, and acid-base balance and serve as a major organ of excretion only when its blood supply is sufficient. Vascular disorders, therefore, can have a significant impact on renal function.

HYPERTENSION

*Hypertension,* sustained elevation of the systemic blood pressure, can result from or cause kidney disease.

Prolonged hypertension damages the walls of arterioles and accelerates the process of atherosclerosis. This damage primarily affects the heart, brain, kidneys, eyes, and major blood vessels. In the kidney, arteriosclerotic lesions develop in the *afferent* (leading into) and *efferent* (going out of) arterioles and the glomerular capillaries. The glomerular filtration rate declines and tubular function is affected, resulting in proteinuria and microscopic hematuria. Approximately 10% of deaths attributed to hypertension result from renal failure (Braunwald et al., 2001).

*Malignant hypertension* is a rapidly progressive form of hypertension that may develop in clients with untreated primary hypertension. The diastolic pressure is in excess of 120 mmHg and may be as high as 150 to 170 mmHg. Malignant hypertension affects less than 1% of hypertensive clients; it is more common in African Americans than in people of European ancestry. Untreated, malignant hypertension causes a rapid decline in renal function due to vessel changes, renal ischemia, and infarction.

Approximately 5% to 10% of hypertensive clients have *secondary hypertension,* which is actually a manifestation of an underlying disease. Renal vascular disease and diseases of the renal parenchyma, such as diabetic nephropathy, are commonly associated with secondary hypertension.

Management of hypertension to maintain the blood pressure within normal limits is vital to prevent kidney damage. When hypertension is secondary to kidney disease, adequate blood pressure control can slow the decline in renal function. Hypertension and its management is discussed in depth in Chapter 33.

RENAral ARtery OCCLUSION

Renal arteries can be occluded by either a primary process affecting the renal vessels or by emboli, clots, or other foreign material. Risk factors for *acute renal artery thrombosis* (formation of a blood clot in the renal artery) include severe abdominal trauma, vessel trauma from surgery or angiography, aortic or renal artery aneurysms, and severe aortic or renal artery atherosclerosis. Emboli from the left side of the heart can travel via the aorta to occlude the renal artery. Emboli may form as a result of atrial fibrillation (irregular and uncoordinated electrical activity of the atria), following myocardial infarction, as vegetative growths on heart valves associated with bacterial endocarditis, or from fatty plaque in the aorta.

Renal arterial occlusion may be asymptomatic when the occlusion develops slowly and the affected vessels are small. Acute occlusion leading to ischemia and infarction typically causes sudden, severe localized flank pain, nausea and vomiting, fever, and hypertension. Hematuria and oliguria may occur. In the older client, the new onset of hypertension or worsening of previously controlled hypertension may signal renal artery thrombosis.

Laboratory studies reveal leukocytosis (elevated WBC), and elevated renal enzyme levels, including aspartate transaminase (AST) and lactic dehydrogenase (LDH). These enzymes,