ANSWER TO PHOTO QUIZ (PAGE 365)

A FATTY CAUSE OF ACUTE RENAL FAILURE

DIAGNOSIS

Renal biopsy showed a chronically injured kidney with severe arteriosclerosis but also evidence of extensive and recent atheroembolism, with cholesterol emboli surrounded by a cellular infiltrate but no fibrosis. It was assumed that atheroemboli had dislodged from atherosclerotic lesions in the aorta and/or renal arteries and had caused acute renal failure. Renal atheroembolism is iatrogenic in the majority of cases. Angiographic procedures and cardiovascular surgery are well-known triggering events. Long-term anticoagulation and/or fibrinolytics as triggers for atheroma rupture are less known, but were also present in 76% of patients in one case series.[1,2] Oral anticoagulants, heparin, low-molecular-weight heparin, and fibrinolytics such as streptokinase may dissolve clots that stabilise atherosclerotic plaques.

Renal complications of atheroemboli may be acute, subacute, or chronic,[3] but may also be a coincidental finding in renal biopsies or autopsies.[4] Most of the time, renal involvement is accompanied by other signs and symptoms of systemic embolisation. These may vary, but may include fever, wasting, laboratory evidence of inflammation, decreased C4 levels, and high eosinophil counts. Cutaneous signs are virtually always present. Fundoscopy demonstrates retinal cholesterol crystals in 22% of cases.[1] In the vast majority of cases, non-renal biopsies (e.g. skin biopsies) are diagnostic of atheroembolism and renal biopsy can be avoided. In the current case, the C-reactive protein was minimally elevated at 18 mg/l, the eosinophil count was normal, as were the C4 levels. Fundoscopy was not performed due to a low clinical suspicion for atheroemboli.

After establishing the diagnosis, acenocoumarol was substituted for acetylsalicylic acid, six months after the pulmonary embolism. Intensive hypertension control, including an ACE inhibitor and statin therapy, were initiated. Six months after kidney biopsy, the eGFR had stabilised at 39 ml/min/1.73 m^2. Therefore, additional glucocorticoid therapy was not initiated.

CONCLUSION

Anticoagulation and/or fibrinolytics can be triggers for atheroma rupture and renal atheroemboli. In patients with atherosclerotic plaques or aortic abdominal aneurysms the risks and benefits of anticoagulation should be weighed.

DISCLOSURES

The authors declare no conflicts of interest.

REFERENCES

1. Belenfant X, Meyrier A, Jacquot C. Supportive treatment improves survival in multivisceral cholesterol crystal embolism. Am J Kidney Dis. 1999;33:840-50.
2. Glassock RJ, Ritz E, Bommer J, et al. Acute renal failure, hypertension and skin necrosis in a patient with streptokinase therapy. Am J Nephrol. 1984;4:193-200.
3. Meyrier A. Cholesterol crystal embolism: Diagnosis and treatment. Kidney Int. 2006;69:1308-12.
4. Moolenaar W, Lammers CB. Cholesterol crystal embolization in the Netherlands. Arch Intern Med. 1996;156:653-7.