Renal hemosiderosis secondary to intravascular hemolysis after mitral valve repair

In Hee Lee¹, Gun Woo Kang¹, Chang Yeon Kim², Sun-Jae Lee³, Min-Kyung Kim⁴
¹Department of Internal Medicine-Nephrology, Daegu Catholic University School of Medicine, Korea, Republic of
²Department of Internal Medicine-Cardiology, Daegu Catholic University School of Medicine, Korea, Republic of
³Department of Pathology, Daegu Catholic University School of Medicine, Korea, Republic of
⁴Department of Pathology, Dongguk University College of Medicine, Korea, Republic of

Case Study: Renal hemosiderosis is a disease in which hemosiderin deposits in the renal cortex as a form of iron overload. However, cases of renal hemosiderosis due to intravascular hemolysis following mitral valve repair have been rarely reported. The authors report a case of renal hemosiderosis caused by chronic intravascular hemolysis following mitral valve repair. A 62-year-old woman who had undergone mitral valve repair with artificial chordae and an annuloplasty ring presented to the hospital two years later with microscopic hematuria and low-grade proteinuria that had persisted for a year. Laboratory findings revealed normal renal function (serum creatinine, 0.8 mg/dL); however, blood examination revealed normocytic normochromic anemia with a hemoglobin level of 8.9 g/dL, decreased haptoglobin (<10 mg/dL) and elevated level of lactate dehydrogenase (1,506 U/L), and the presence of schistocytes in the peripheral blood smear. Paroxysmal nocturnal hemoglobinuria was excluded based on negative results for percentages of CD59+ cells and CD55+ cells. On the basis of a suspicion of an asymptomatic urinary abnormality, a percutaneous renal biopsy was performed, which revealed no specific glomerular abnormality, tubular atrophy, or interstitial fibrosis but extensive deposition of hemosiderin in the proximal tubule. On the follow-up echocardiography, moderate mitral regurgitation with regurgitant blood striking against annuloplasty ring was confirmed. The patient refused another surgery and hence was administered oral iron preparations, N-acetylcysteine, and a β-receptor blocker. During the 24-month follow-up period, hemolytic anemia persisted, but there was no significant decline of renal function. Our case indicates that timely hematologic evaluation and follow-up echocardiography for mechanical hemolysis in cases of persistent anemia following mitral valve repair is important. Moreover, for cases of chronic intravascular hemolysis with asymptomatic urinary abnormalities, a renal biopsy is required to exclude underlying kidney pathology and predict potential renal insufficiency.