INTRODUCTION

Lupus nephritis (LN) is present in about 50% of patients with systemic lupus erythematosus. Pathologic mechanisms contributing to acute kidney injury (AKI) are broad when observed in LN. Rarely fibrin deposition is found in conjunction with AKI in LN. We present a case of glomerular fibrin deposition and add to the literature mechanisms of AKI in LN.

CASE SUMMARY

An 18-year-old female presented to the hospital with cutaneous lesions refractory to prior management. She presented with an AKI with an initial creatinine of 3.7mg/dL (baseline 0.7-0.9) with new pancytopenia also identified. Urine analysis showed 2+ Hgb (hemoglobin) and 10 RBC (red blood cells). No additional contributions to AKI were identified and renal ultrasound was unremarkable. Urine microscopy demonstrated dysmorphic RBCs and empiric pulse-dosed steroids were completed while serologies were pending given the high suspicion for underlying nephritic etiology. LDH elevated to 309, haptoglobin elevated to 238, and fibrinogen normal at 232. Hypocomplementemia was also present with C3 16 and C4 <3. ANA was positive along with DsDNA. Lupus anticoagulant was negative. A renal biopsy was subsequently performed to guide further immunosuppressive therapies. Light microscopy showed an increased mesangial matrix along with glomerular fibrin thrombus at the vascular pole. Electron microscopy showed frequent subendothelial deposits and mesangial immune complex deposits. With biopsy results confirming the suspicion of proliferative lupus nephritis, mycophenolate mofetil was started and the patient’s creatinine, pancytopenia, and cutaneous lesions subsequently improved.

REFERENCES


CONCLUSION

Our case demonstrates fibrin deposition in LN without a background hemolytic process. Glomerular fibrin deposition can lead to crescent formation and is thought to play a role in the development of glomerular sclerosis. It is thought to be secondary to PAI-1 (plasminogen activator inhibitor 1). The major physiological role of PAI-1 is to block the conversion of plasminogen to plasmin by tissue-type plasminogen activator (tPA) and urokinase-type plasminogen activator (uPA). Immunosuppression has long been the standard of care for LN but we hypothesize that these findings of fibrin deposition highlight the importance of gaining further clarity and understanding of the fibrinolytic system as a potential contributor to the pathogenesis of AKI in LN. Further correlation studies of the fibrinolytic system are required and could provide additional molecular targets in early management of proliferative LN.