Journal of Clinical and Medical Images

Clinical Image

ISSN: 2640-9615 | Volume 8

Sickle Cell Disease Nephropathy Kidney Biopsy Images

Liam Qi[1,2*], Michael Burke[1,2] and Boomi Kwon[3]

[1]Mater Kidney Health Service, Brisbane, Queensland, Australia

[2]Faculty of Medicine, The University of Queensland, Brisbane, Queensland, Australia

[3]Mater Anatomical Pathology, Brisbane, Queensland, Australia

[*]Corresponding author:

Liam Qi,
Mater Kidney Health Service, Brisbane, Queensland, Australia,
Faculty of Medicine, The University of Queensland, Brisbane, Queensland, Australia

Received: 11 Feb 2025

Accepted: 21 Feb 2025

Published: 26 Feb 2025

J Short Name: JCMI

Keywords:

Sickle Cell Disease; Nephropathy; Kidney Biopsy

Citation:

Liam Qi, Sickle Cell Disease Nephropathy Kidney Biopsy Images. J Clin Med Img. 2025; V8(7): 1-2

1. Introduction

Sickle-cell disease (SCD) is one of the most common severe monogenic disorders in the world with haemoglobin polymerisation resulting in multiple systemic complications including nephropathy [1]. The pathophysiology of SCD-related nephropathy is complex with vaso-occlusion and subsequent ischaemia-reperfusion injury, haemolysis, oxidative stress, and hyperfiltration [2]. Limited evidence exists for treating SCD-nephropathy [3]. This case provides insight into the pathophysiology of SCD-related nephropathy.

2. Clinical Case

A 20-year-old male of African ethnicity was diagnosed with new fluid overload in the setting of COVID-19 infection in April 2024 on a background of Homozygous HbS Sickle Cell anaemia, iron overload from multiple transfusions, left humeral head avascular necrosis and severe obstructive sleep apnoea. His renal function was normal (creatinine 47µmol/L and eGFR >90ml/min/1.7m2), urine protein-creatinine ratio (PCR) was 1870mg/mmol and urine albumin-creatinine ratio was 1375.8mg/mmol. His serum glomerulonephritis screen was unremarkable, and furosemide was commenced for fluid overload. A kidney biopsy via ultrasound guidance was performed in May 2024 and all pictures were taken with a 20x magnification lens. Proteinuria was treated with irbesartan and his urine PCR improved to 925mg/mmol with stable renal function 8 months later.Light microscopy showed focal segmental glomerulosclerosis (FSGS) with glomerular loops engorged with sickled red blood cells (Figure 1). Prussian blue iron stain showed haemosiderin deposition in the tubular lumen (Figure 2).

Immunofluorescence was unremarkable. Electron microscopy revealed features of sickle-shaped red blood cells, duplication of glomerular basement membranes, and numerous haemosiderin granules within the tubular cytoplasm which would be in keeping with the light microscopic impression of SCD-nephropathy (Figure 3).

3. Conclusions

This case demonstrates the vaso-occlusive nature of SCD in the glomeruli and tubules resulting in FSGS lesion and duplication of the glomerular basement membranes resulting in nephrotic range proteinuria as well as improvement of proteinuria in SCD-nephropathy with an angiotensin-receptor blocker.

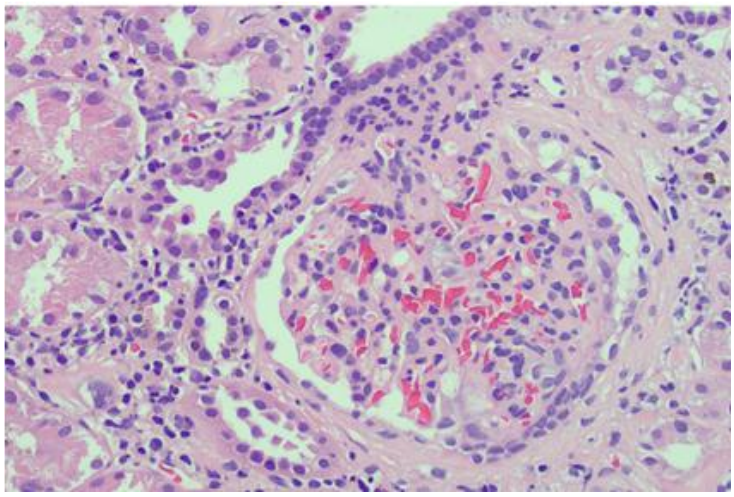

Figure 1: Hematoxylin and Eosin stain 200x.

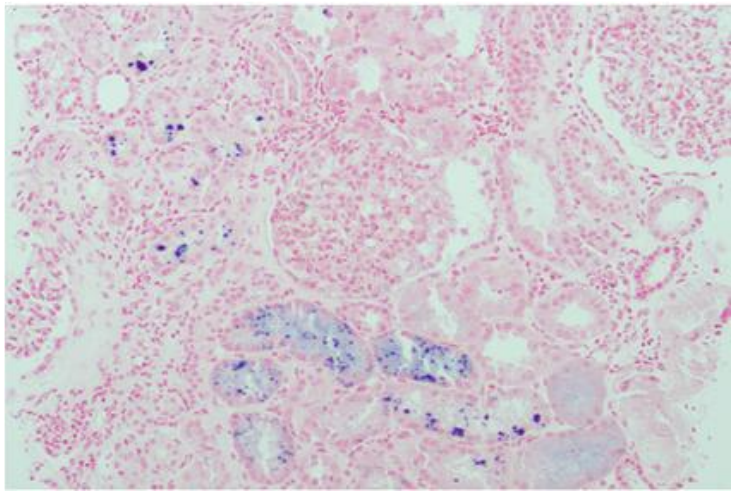

Figure 2: Prussian blue iron stain 100x.

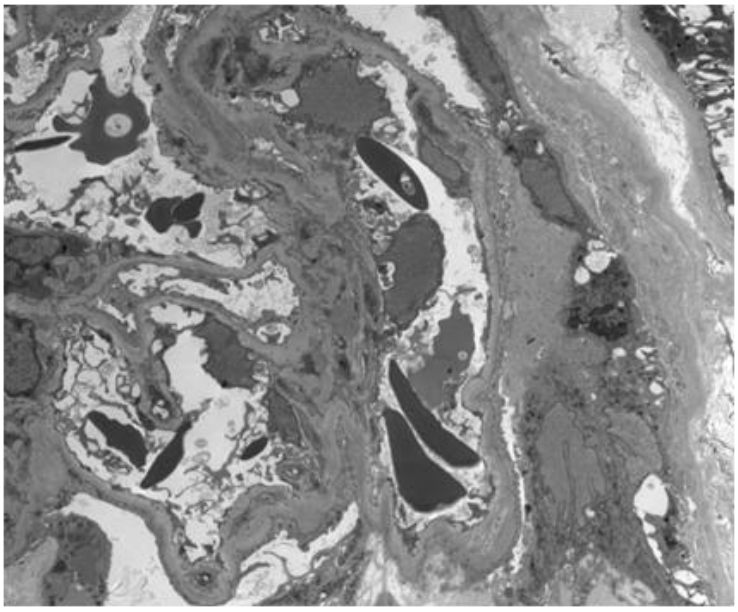

Figure 3: Electron microscopy.