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P390 -Self-limiting nephrotic syndrome, acute kidney injury, and red cell aplasia – a case report of parvovirus associated glomerulonephritis

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Objectives and Methods
We report the case of a 31-year-old female who presented with self-limiting nephrotic syndrome, acute kidney injury associated and normocytic anaemia, thrombocytopenia and joint pain.

The patient attended her local hospital with a week-long history of fever, generalised joint and back pain and swelling to her hands, feet and abdomen associated with a 6kg increase in weight.

Her past medical history included gestational proteinuria without hypertension. She took no regular medications, lived with her husband and 1-year-old son and worked as a nurse in an emergency department. She had no history of recent travel or diarrhoeal illness. Both her maternal great aunt and great uncle required renal transplants, her great aunt for diabetic nephropathy and her uncle for unknow reasons.

On examination she had facial swelling and non-pitting subjective oedema to her arms and legs. Her JVP was not raised and her chest was clear with normal heart sounds. Her abdomen was mildly distended but soft and she had bilateral renal-angle tenderness. There was no erythema or heat to her joints and she had no skin changes.

Differential diagnosis included TMA or a transient GN.

3 days post admission her blood abnormalities began to self-resolve with full resolution both biochemically and clinically after 2.5 weeks.

We reviewed the literature to clarify the likely diagnosis.

Results
Parvovirus B19 is a small single stranded DNA virus with a pronounced tropism for erythroid precursor cells. It is common worldwide being mostly accumulated in childhood.
Infection with parvovirus B19 can cause several clinical syndromes. Fifths disease, transient aplastic crisis and pure red blood cell aplasia are among the most well established. Though a causal link is still to be proven B19 has also been implicated in glomerular disease, based on the temporal relationship of viral infection with onset of renal pathology and identification of viral proteins in the glomeruli on PCR following renal biopsy.
Clinical presentations have been varied but the most common is acute nephritic syndrome with hypocomplementemia, following a prodrome of fever, rash and arthritis. Nephrotic syndrome and thrombotic microangiopathy have also been described.
Spontaneous resolution is common though renal dysfunction and proteinuria can persist.

Blood was sent for parvovirus serology and was positive for both IgM and IgG indicating recent infection. On direct questioning, exposure to a local parvovirus outbreak was confirmed.

This case further demonstrates the likely link between parvovirus B19 and transient self-limiting glomerulonephritis with nephrotic syndrome in immunocompetent individuals. This case is unusual as thrombocytopenia was also present, as well as the more typical aplastic anaemia, clouding the clinical picture initially.

Conclusion

Parvovirus B19 has been linked to a spectrum of glomerular diseases including glomerulonephritis with nephrotic syndrome and can seemingly contribute to a TMA like picture with reduced platelets, high LDH and anaemia.

Serological testing could be considered in individuals with unexplained self-resolving GN, epically in those who also have feature of aplastic anaemia.

Further study is needed however to establish a definite causal link between parvovirus infection and GN.