Background/Aims
Acquired hypocomplementemia occurs through immune complex-mediated diseases, as seen in systemic lupus erythematosus (SLE), idiopathic membranoproliferative glomerulonephritis (MPGN) and cryoglobulinemia. Infective causes are less common, although chronic bacterial and viral infections causing glomerulonephritis (GN) and vasculitis have been reported. Bacterial infections causing chronic osteomyelitis, endocarditis and visceral abscesses have all been implicated, whilst hepatitis B is the only virus known to cause severe GN/vasculitis leading to low complement levels. Severe meningococcal disease is an established cause of acquired hypocomplementemia, typically associated with Nisseria Meningitidis. Parvovirus B19 is an infection exclusive to humans, usually manifesting as fifth disease, a mild illness characterised by a rash, fever and runny nose. Less common systemic effects include hydrops fetalis, thrombocytopenia, neutropenia, neurological sequelae (including encephalopathy, aseptic meningitis, neuropathy and neuralgic amyotrophy), myocarditis and hepatitis. Arthropathy is by far the most common manifestation in adults, affecting 80% of females and 30% of males.

Methods
The literature on Parvovirus B19 and hypocomplementemia is sparse, limited to case reports and comparative studies, the pathogenicity of which stems from glomerulonephritis, consequently resulting in low complement levels. We present a case series of 2 patients with Parvovirus B19 infection and acquired hypocomplementemia between 2016-2020.

Results
Patient A, a 26-year-old female, presented with a vasculitic rash on her legs and arthralgia affecting her hands and knees, refractory to oral prednisolone and hydroxychloroquine. She was found to have low complement levels (C3 0.61 g/l and C4 0.03 g/l), whilst ANA, ENA and ANCA testing was negative. She also had a negative rheumatoid factor and anti-CCP antibody, and a normal serum protein: creatinine (6 mg/mmol). Immunoglobulins were within normal range (lgG 8.7 g/l, lgA 0.80 g/l, lgM 2.41 g/l) and a serum protein electrophoresis was normal. Hepatitis and HIV serology was also negative. Patient B, a 34-year-old female, 9 weeks pregnant, presented with polyarthralgia, fever and a maculopapular rash, with normal inflammatory markers and a normal serum protein: creatinine (9 mg/mmol). Her complement was low (C3 0.57 g/l and C4 <0.02 g/l), ANA weakly positive, ENA negative and her ANCA screen showed a pANCA pattern with positive anti-PR3 antibodies (4.6 U/ml). She also had weakly positive dsDNA antibodies (37 IU/ml), and smooth muscle antibodies were weakly positive on tissue biopsy. Anti-C1q antibodies were normal. lgM was raised (2.56 g/l), whilst lgG and lgA were normal (10.8 g/l and 1.37 g/l, respectively). Serum protein electrophoresis was normal. HIV, hepatitis B and syphilis serology was all negative. In both cases, Parvovirus B19 IgM antibody was detected.

Conclusion
We recommend that clinicians consider acute viral illness in patients presenting with vasculitis and hypocomplementemia. If diagnosis and treatment can be established early, patients can avoid the need for long term immunosuppression.

Disclosure
A. Gharatya: None. C. Nelson: None. S. Melath: None.