She had altered behaviour with agitation, disorientation, fluctuating consciousness, hallucinations, and altered sleep. On examination, she had malar rash, icterus, and hepatosplenomegaly. She also had catatonia, mutism, would stare intermittently, had low speech output and psychomotor retardation with rigidity. There was no focal deficit. Investigations revealed pancytopenia, transaminitis, conjugated hyperbilirubinaemia, normal renal functions, antinuclear antibody (ANA) - positive (homogenous pattern), high anti-dsDNA with hypocomplementemia. Liver biopsy revealed steatosis with hepatitis. Screen for infections was negative, except CMV. Very high levels of CMV DNA in blood were noted on PCR. It was a clinical dilemma as to whether CMV was causative, co-infection or a re-activation due to immunosuppression. Magnetic resonance imaging (MRI) brain showed cortical atrophy. There was no evidence of any vascular involvement.

She was treated with intravenous (IV) methylprednisolone, IV cyclophosphamide pulses and oral valganciclovir.

**Results** A repeat CMV viral load done after six weeks of oral valganciclovir therapy was undetectable. She has been followed up for a period of 6 months. She has shown marked improvement in her neurological status and transaminases have normalised.

**Conclusions** CMV is an important pathogen in patients with SLE; however its exact pathogenesis needs more research.