Case Report

CMV and EBV infection presenting with ascites and hypoalbuminemia in an immunocompetent child

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Abstract

A previously well 4 year old boy presented with an acute onset of ascites and Hypoalbuminemia. He clinically showed complete resolution of symptoms over a 3 day period without any clinical intervention. His blood serology was positive for CMV PCR and EBV PCR and no further treatment was needed. Ascites as a sign of underlying pathology is unusual in childhood and viral causes should be considered after exclusion of protein losing enteropathies and malignancy.

Case report

A previously well 4 year old boy was referred to a regional gastroenterology department with abdominal distension and hypoalbuminemia. He had been well prior to presentation but was treated with oral cephalexin by his General Practitioner (GP) for a suspected urinary tract infection (UTI) for 2 days. He was noted to have abdominal distension and swelling to the upper part of his thigh since the start of oral cephalexin and was thought to have had an allergic reaction to Cephalexin. Following a previous UTI he was noted to have left lower pole renal scarring for which he was taking prophylactic trimethoprim. Subsequent ultrasound scan and DMSA scan were reported as normal. He was treated for asthma with inhaled salbutamol and beclomethasone by his GP. There was no other significant past medical or family history. On examination he was noted to have ascites and shifting dullness. His liver was palpable 3 cms below the right costal margin and he had pitting oedema to his knees in both legs His urine dipstick test was normal and urine protein/creatinine ratio was 28 (normal range upto 50), protein 0.08 (normal <10mg/dL) and creatinine 2.9 (normal). Liver Function tests, thyroid function and serum urea and electrolytes were normal. Serum albumin was 17g/dl (20-45 g/dl), LDH 825 u/ml (range500-1000 u/ml). Immunoglobulins performed during the acute episode, IgG 2.53 (4.44-11.9), IgA 0.44 (0.30-0.65), IgM 0.26 (0.41-1.86). Full blood count was normal.

A Cytomegalovirus (CMV) PCR titre was positive at 4731 copies/ml and an Epstein Barr Virus (EBV) PCR titre was positive at 6253 copies/ml. ANA was positive at 1:80. Anti double stranded DNA, Anti e-ANCA, p-ANCA, and Epidermal BM antibody were negative. His stool alpha-1-antitrysin was 7.4 mg/dl (normal range 0.13-2.28).

There was a rapid improvement in clinical signs over three days with similarly rapid resolution of hypoalbuminemia without treatment and at three month clinic review there were no abnormal signs or symptoms.

Discussion

Cytomegalovirus virus and Epstein Barr Virus belong to the same Herpes family. They have similar characteristics to Herpes Simplex Virus (1&2), Varicella zoster, and Herpes Virus (6,7& 8) and all share the property of latency and reactivation. These viruses have double stranded linear DNA, icosahedral symmetry and a viral envelope.

Clinical manifestations may be mild in immunocompetent individuals, the most common manifestations being fever, fatigue, pharyngitis, lymphadenopathy, and hepatitis. Headache, abdominal pain with diarrhoea, arthralgias, and rash may also occur. Laboratory abnormalities may include lymphocytosis or lymphopenia with thrombocytopenia and elevated serum transaminases. However, the heterophile antibody titres or monospot test for EBV may be negative.

Unusual manifestations of acquired CMV infection in healthy individuals include rare reports of pneumonitis; myopericarditis, haemolytic anaemia, viral hemophagocytic syndrome, granulomatous hepatitis, Guillain-Barré syndrome, and meningoencephalitis. There are no previously reported sole gastrointestinal manifestations in immunocompetent individuals who do not require any treatment apart from
supportive care and usually make a full recovery. There are no clinical trials which strongly support evidence for treatment.

EBV has a variety of manifestation in children ranging from otitis media, diarrhoea, upper respiratory infection, and Infectious Mononucleosis. About 90-95% adults are seropositive for EBV indicating how common the infection is, and individuals remain lifelong carriers. Spontaneous splenic rupture (one and two cases per thousand), acute hepatitis, pancreatitis, nausea, vomiting, anorexia have all been reported as well recognised gastrointestinal manifestations of acute EBV infection. EBV infection in the immunocompromised and in children following organ transplant is a well recognised phenomenon. There are no case reports of ascites or hypoalbuminemia secondary to CMV, EBV nor of both infections occurring concurrently and treatment of infections in immunocompetent individuals is supportive. There are no studies supporting evidence for treatment of immunocompetent individuals with CMV or EBV infection, with either antiviral therapy or immune modulatory drugs.

Ascites as a sign in childhood disease is unusual. Hypoalbuminaemia as the cause related to protein losing enteropathy in the absence of overt hepatitis or renal protein loss is the usual cause, although malignancy should be considered.

Due to the rapid recovery of this child, no ascitic fluid was analysed and it was only after resolution of the illness had occurred, that evidence of presumed cause was available following reporting of the viral serology. Ascites as a sign of underlying pathology is unusual in childhood and viral causes should be a consideration.

References