Human serum parvovirus associated vasculitis

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Summary: A 48 year old man had a severe symmetrical arthritis of knee and ankle joints accompanied by an extensive purpuric rash of both legs and several areas of skin necrosis. No other system was clinically involved. Human serum parvovirus-specific IgM was present in a blood sample taken 2 weeks after the onset of the clinical illness indicating recent infection with this virus. The patient was treated with complete bed rest, and the application of saline soaks to both legs. He had a recurrence of the rash 5 weeks after onset, but otherwise made a complete recovery. Purpura with skin necrosis has not previously been reported in association with this virus.

Introduction

Paroviruses are small 18–26 nm non-enveloped, single-stranded DNA viruses. They depend for their replication on actively dividing cells (Siegl et al., 1985). Enjoying a world-wide distribution they have long been known as a source of considerable morbidity in the field of veterinary medicine, where their host range stretches from rodents, through domestic pets, to livestock (Siegl et al., 1985).

In 1975 the first human member of the parvovirus genus was discovered (Cossart et al., 1975). Subsequently this human serum parvovirus (HSPV) has been shown to be the cause of two clinical entities: the childhood exanthem-erythema infectiosum (Anderson et al., 1983) and aplastic crisis in various chronic haemolytic anaemias (Anderson & Pattison, 1983).

The various clinical manifestations of infection with HSPV in adults are also coming to light, particularly the predilection to cause a symmetrical arthropathy (Reid et al., 1985).

We report a case of HSPV infection in a 48 year old man who presented with arthritis and a florid vasculitic rash.

Case report

Two weeks before hospitalization a 48 year old man noticed 'tiny red spots' around both ankles. Two days before his admission the rash became much more prominent and extensive. The next day he developed severe pain in both ankles and knees.

On admission he had a purpuric rash on both lower legs, and the backs of both thighs, extending up to the buttocks. There were a few tiny areas of superficial skin necrosis on his lower legs, and at this stage his ankles and knees were so painful that he was unable to move them. Effusions were present in both knee joints. Laboratory investigations included the following: haemoglobin 13.2 g/dl; platelets 333 x 10³/l; white cell count 20.1 x 10³/l (neutrophils 64%, lymphocytes 30%, monocytes 1%). The white cell count later returned to normal; antistreptolysin 0 titre not significant; RA latex negative; Rose Waaler negative; total haemolytic complement (CH₅₀) 381 units/ml (normal range 250–702 units/ml); circulating immune complexes 45 µg aggregated human gammaglobulin equivalents (AHG)/ml (normal range 0–49 µg AHG eq/ml); C3 0.94 g/l (normal range 0.5–1.2 g/l); C4 0.34 g/l (normal range 0.2–0.5 g/l). An autoantibody screen was negative for anti-smooth muscle, anti-nuclear, anti-gludin, anti-mitochondrial, antithyroglobulin, and anti-gastric parietal cell antibodies.

Antibody tests were carried out on sera obtained at 2 weeks and 2 months into the patient’s illness and significant titres to the following viruses and agents were not found: rubella, measles, adenovirus, varicella-zoster, cytomegalovirus, herpes simplex, mumps, Mycoplasma pneumoniae, Q fever, and psittacosis-lymphogranuloma venereum.

HSPV-specific IgM was present in the serum sample taken 2 weeks after the onset of illness when tested by IgM antibody capture radioimmunooassay.

On complete bed rest and saline soaks to the legs the patient made a remarkable recovery. On the 7th day he

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was mobile again, his joint pain and effusions having settled completely. His purpuric rash had also faded very quickly but the tiny necrotic areas broke down to leave superficial ulceration. At this stage he was discharged at his own request, and at review 1 month later his skin ulcers were all healed and he had no further joint symptoms.

It is interesting to note that he had a recurrence of the rash 5 weeks after onset of illness, on his right lower leg, after driving his car.

Discussion

The facial rash seen in erythema infectiosum in children is characteristically described as having a 'slapped cheek' appearance. This is usually accompanied by a macular rash on the extremities, which fades to give a lacy reticulated pattern.

In adults this typical presentation is often not seen. Instead, workers have described a range of skin rashes from erythematous, to macular, to rubella-like (Plummer et al., 1985; Reid et al., 1985; White et al., 1985). Recently it has been reported that 2 patients with HSPV infection demonstrated purpura in their clinical presentation (Lefrere et al., 1985). To this range of dermatological manifestations we can now add purpura associated with skin ulceration.

The clinical spectrum of cutaneous vasculitis ranges from urticarial lesions to purpura, nodules and ulceration. In the assessment of a patient with cutaneous vasculitis one should enquire about the drug history, recent streptococcal pharyngitis, viral prodromata and also look for evidence of associated systemic diseases, particularly malignancy and connective tissue disorders. Despite full investigation, no aetiological factor can be identified in many cases. These are then classified as vasculitis of unknown aetiology. We would suggest that HSPV involvement should be looked for in such cases, particularly if accompanied by joint involvement.

In our patient the rash presented 2 weeks before the onset of the arthralgia. However this period can extend up to 3 months (White et al., 1985). In addition, the various clinical presentations that can be associated with this virus make it essential that clinical suspicions are confirmed by appropriate serological tests. In this context clinicians should be aware that HSPV-specific IgM remains detectable for 10–12 weeks from the onset of symptoms (Anderson et al., 1982).

References


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