

Parvovirus infection in a family with wheeze in an adult

R. POSTLETHWAITE, BSc, MD, FRCPath
Professor of Virology, Department of Bacteriology,
University of Aberdeen

SUMMARY. In a family outbreak of human parvovirus infection, a 59-year-old doctor had a prodromal illness with wheeze. This was followed by a distinct acute episode with itch and arthropathy and a two-month phase characterized by intermittent cough and wheeze.

Introduction

AFTER the association of parvovirus¹ with human haematological disease² was first recognized, other clinical manifestations were described.³⁻⁵ However, although symptoms of upper respiratory tract infection occur in children with erythema infectiosum (fifth disease)³ and studies of volunteers have demonstrated the airborne transmission of human parvovirus,⁶ lower respiratory tract disease is seldom reported.⁷ It was prominent in a 59-year-old man in the following description of a family outbreak.

Family case report

On 17 June 1984, a 59-year-old doctor became ill with chills, malaise and a mouth temperature of 37.8 °C which kept him off work for two days. He then felt well for five days apart from a transient, but, for him, unusual wheeze. From 26 June until 3 July 1984 he was again ill in bed with intense itching, puffiness of hands, wrists and feet and generalized muscular pains. The itch was controlled by Anthisan cream (mepyramine maleate 2%) and Caladryl lotion (calamine 8%, diphenhydramine hydrochloride 1%, camphor 0.1%); aspirin relieved headaches and muscle pains. Both his knees were hot for a day or two and he suffered pain over the insertion of the Achilles tendon on walking. The doctor returned to work on 3 July but from 30 June until the end of August he complained of lassitude, intermittent expiratory wheeze and a mainly dry cough which disturbed his sleep from 2 to 5 August and was relieved by aniseed sweets and fruit drinks. The skin of the middle and distal phalanges peeled on 31 July. The respiratory symptoms did not fully resolve until 29 August when the results of physical examination, chest radiographs, pulmonary function tests and sputum culture were all normal.

There was no serological evidence for recent infection with the viruses of measles, rubella or influenza A or B, nor by adenovirus, Epstein Barr virus, respiratory syncytial virus, chlamydiae, *Coxiella burnetii*, *Mycoplasma pneumoniae* or group A β haemolytic streptococci. There was no abnormality of serum rheumatoid factor, C-reactive protein, immunoglobulins (IgG, IgM, IgA or IgE) or complement C3, but complement C4 while within normal limits (15–40 mg dl⁻¹) on 5 July and 14 August was low (11.7 mg dl⁻¹) on 27 June. Human parvovirus-specific antibodies⁸ were already present on day 10 of the illness and had increased markedly by day 18 but IgM fell rapidly and IgG fell slowly thereafter (Figure 1). Human parvovirus antigen was not detected in sera collected between 27 June and 19 October.

The doctor's wife was feverish (38 °C) with malaise for four days from 26 June and developed an itchy rash on her arms, legs and neck on 29 and 30 June. Her neck lymph nodes were swollen. Swollen feet and stiff hands and neck, which were noticed on 3 July, improved rapidly, but her knees remained stiff for two or three weeks. Serum IgG and IgM antibodies to human parvovirus were elevated on 14 August (> 100 and 11.0 radioimmunoassay units respectively).

The doctor's 17-year-old daughter was in bed with an upper respiratory tract illness and sore throat from 7 to 9 June. Serum IgG (but not IgM) antibody to human parvovirus was raised (54.0 radioimmunoassay units) on 31 August.

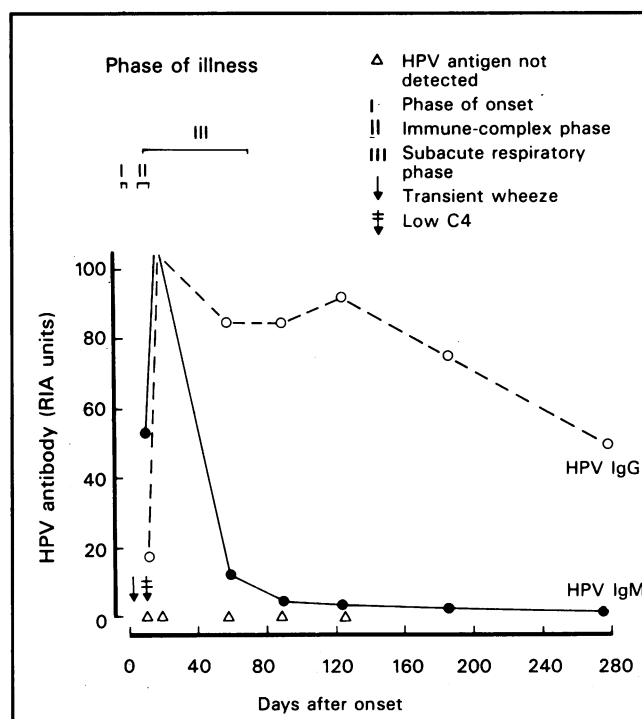


Figure 1. Correlation between phases of clinical illness in the doctor and serum antibody levels to human parvovirus (HPV).

Discussion

This episode occurred toward the end of an outbreak of human parvovirus infection in Grampian⁹ and the infection was probably introduced into the family by the daughter. This report is prompted by the father's lower respiratory symptoms. While other factors were not entirely excluded, evidence for human parvovirus infection was undoubted and immune complexes associated with rising antibody titres, absence of detectable antigenaemia and transient depression of complement C4 could have caused the symptoms during the second phase of illness. Monocyte factors may be important in the pathogenesis of asthma¹⁰ and a role for interferon and virus-specific IgE^{11,12} in the respiratory symptoms reported here is not excluded by the normal values for total serum IgE.

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Acknowledgements

It is a pleasure to acknowledge the help of Dr T. Brown, Dr T.M.S. Reid, Dr J.A.R. Friend, Dr B. Cohen, Dr P.P. Mortimer and Dr M.S. Pereira in the investigation of this family outbreak.

Address for correspondence

Professor R. Postlethwaite, Department of Bacteriology, University of Aberdeen, Medical School, Foresterhill, Aberdeen AB9 2ZD.

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