CASE REPORT

Prolonged hypothermia following respiratory syncytial viral infection in infancy

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Following proven respiratory syncytial viral infection, a previously healthy 2 year old boy displayed notable persistent hypothermia—the lowest temperature being 34.2°C. No obvious ill effects were observed.

A rise in central body temperature is a common feature of infections in infants, often highest during the prodromal or preclinical phase,[1] returning to normality with convalescence. A reduction in body temperature with infection is less common, but may occur during overwhelming sepsis.[2] The respiratory syncytial virus (RSV) causes respiratory illness in winter epidemics in infants, who may exhibit a variety of extrapulmonary complications, of which hypothermia is one.[3] We report on profound hypothermia in a young child, several weeks after recovering from a proven RSV infection.

CASE HISTORY

A normally developed, well grown 2 year old boy, born at term, with birth weight 3580 g and Apgar scores 9 and 10, was found on antenatal ultrasound scan to have hydronephrosis, hydroureter, and severe vesicoureteric reflux. He was treated with prophylactic trimethoprim from birth. At age 11 months, he spent four days in hospital with a chest infection. His highest temperature was 37.6°C.

At the age of 2 years, he was admitted with pyrexia, coughing, and poor appetite. Bronchiolitis was diagnosed, caused by RSV infection (antibody titre ⩾1/256 on admission; 1/32, seven weeks later). Tests for antibodies for influenza A and B, adenovirus, and *Mycoplasma pneumoniae* were all negative. Initial skin temperature was 39.1°C, rising to 39.7°C. He was treated with intravenous cefuroxime, although not appearing to be clinically unwell. Within two days of admission he developed episodes when he appeared "grey" in colour and felt cold and clammy. At this point his rectal temperature was 35.8°C, respiratory rate 32/min, and capillary blood glucose 6.2 mmol/l. He was slowly rewarmed and appeared to recover. Rectal temperature was measured at intervals on successive nights. Figure 1 shows the lowest temperatures recorded; hypothermia persisted for several nights, the minimum temperature recorded being 34.5°C on the 12th night after admission. At this time he appeared to be improving clinically. Blood glucose, and heart and respiratory rates were maintained: serum cortisol showed a normal stress response of <7.0 for the cortisol/creatinine ratio (normal 5–15).

While in hospital, on the 14th night after admission, his rectal temperature was monitored continuously for 14 hours overnight while he was asleep, using a rectal probe attached to a Grant-Squirrel data logger. This procedure was repeated at home two weeks later, after discharge. Figure 2 compares the records. Both show the typical pattern of deep body temperature seen in younger infants—a fall in temperature for 2–3 hours after bedtime, usually followed by a period of a few hours when the temperature remains at the same level, then rises slowly prior to waking. However, at 12 days of the illness, the maximum fall in temperature is reached at 34.5°C; two weeks later the lowest temperature reached is 35.5°C, and he has therefore recovered from the levels of hypothermia seen during the illness.

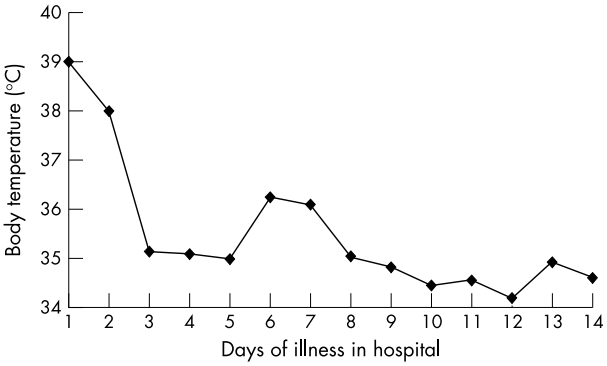

Figure 1 Body temperatures taken during night time recordings in hospital.

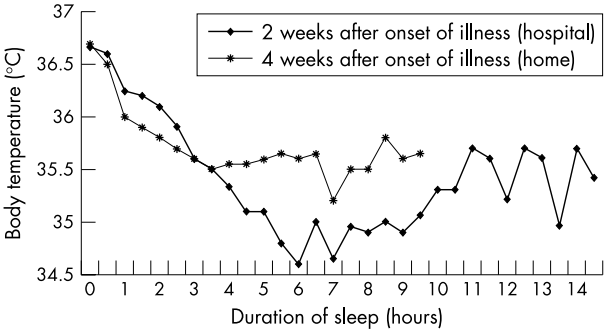

Figure 2 Comparison of continuous rectal temperature recordings, two and four weeks after onset of illness.

DISCUSSION

Hypothermia has rarely been reported as a complication of RSV infection in young children,[3 4] recurring at the peak of the illness, not persisting after apparent clinical recovery, as appears to be the case here.

Neurological complications are not a feature of RSV infections, and the impermeability of the blood-brain barrier to antigen-antibody complexes rules out any direct affect on the central control mechanism of body temperature. Molecular mimicking by way of a hypersensitivity type of reaction is a possibility. Epitopes shared between infectious agents and human nervous tissues[5] could explain why antibodies generated against a certain type of infectious agent, may

become autoantibodies to a homologous epitope in the host, causing tissue dysfunction, even after the clearance of the pathogen. RSV is one of the only respiratory pathogens that produces its most devastating ill effects at the time of abundant antibody production,[6] which lends support to the idea of being involved in the causation of hypothermia in this child; although the fact that it occurred so early in the illness is an argument against this.

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