Intraosseous infusion for resuscitation

I G Ryder, H M Munro, I J M Doull

Abstract

An 11 week old infant who had a cardiac arrest secondary to gastrointestinal haemorrhage and was successfully treated using intraosseous infusion is reported. The child was discharged with no apparent neurological deficit.

Intraosseous infusion was widely practised in the 1940s.[1] There is renewed interest in its use in North America for paediatric resuscitation.[2] This route is successful because the marrow cavity cannot collapse, allowing rapid access to the circulation despite profound hypovolaemia.

Case report

An 11 week old infant was admitted with a history of diarrhoea, vomiting, and weight loss of 0·7 kg (6·5 to 5·8 kg). Viral gastroenteritis was suspected and intravenous rehydration begun. Within 48 hours the child was apyrexial, had resumed feeding, and the intravenous line was removed.

On the third night after admission the child deteriorated and became unresponsive, cold, and cyanosed. Intravenous access proved impossible despite attempts by experienced paediatric staff. The child deteriorated further and became asystolic, necessitating tracheal intubation and cardiac massage; 200 μg of adrenaline was instilled into the endotracheal tube. As further attempts to gain intravenous access were unsuccessful a 19 gauge hypodermic needle was inserted into the right tibia just below the tibial tuberosity, and 250 ml of 4·5% human albumin solution (HAS) was infused rapidly using a 20 ml syringe, followed by 5 ml of 4·2% sodium bicarbonate. A further 200 μg of adrenaline was given via the endotracheal tube.

The colour and peripheral perfusion of the child improved and irregular complexes appeared on the electrocardiogram. At this stage, when approximately 50% of the child's estimated circulating volume had been infused, it was possible to site an internal jugular line. Sinus rhythm was restored, carotid and femoral pulses became palpable, and he began to make respiratory effort.

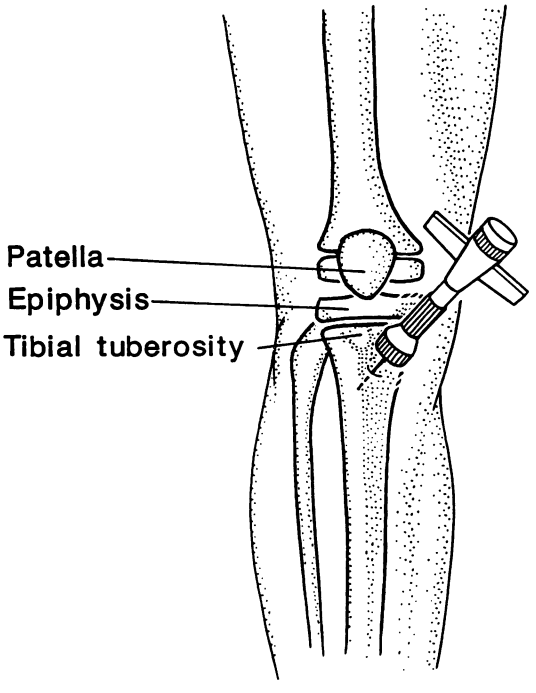
During further assessment rectal examination revealed bloodstained stool. At laparotomy the bowel was found to contain a large volume of blood. Although no cause for the bleeding was identified, intermittent volvulus was suspected. Eleven days after the cardiac arrest the child was discharged home with no apparent neurological deficit.

Discussion

Establishing intravenous access in critically ill children may be difficult. Rosetti *et al* found that it took more than 10 minutes to establish vascular access in 24% of paediatric circulatory arrests and in 6% of cases no vascular access was established.[3] In contrast, Smith *et al* recorded an 80% success rate for intraosseous infusion by paramedical staff during the journey to hospital.[4]

The technique of intraosseous infusion consists of sterilising the skin and inserting a 13 to 18 gauge needle into the tibia 1–2 cm below the tibial tuberosity on the medial, flat surface. A boring or screwing action may be required, directing the needle caudally, away from the growth plate (figure). Intraosseous placement is confirmed by loss of resistance, aspiration of bloodstained marrow, and free infusion of fluid.[2] Ideally, a bone marrow infusion needle should be used. In our case the largest needle available was a 19 gauge hypodermic needle.

Other sites for infusion include the distal tibia proximal to the medial malleolus, the distal femur, the iliac crest, and the sternum. In



Insertion site for intraosseous needle.

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children under 3 years of age the sternal marrow space is inadequate and there is the risk of mediastinal infusion.[2 5]

The contraindications are osteogenesis imperfecta, osteoporosis, osteomyelitis, ipselateral fracture of the extremity, or local skin infection.[2]

The commonest complication is subcutaneous infusion after failure to enter the marrow cavity. Skin infection has been reported in 0·7% (five out of 694), which is less than that reported with intravascular catheters (3·7%). Osteomyelitis has been reported in 0·6% (27 out of 4270), with an increased risk if the needle is left in situ for a prolonged period. Despite concern about effects on bone growth, no long term side effects have been reported.[5] The deaths attributed to intraosseous infusion have all followed sternal puncture.[2 3]

Blood products, fluids, vasoconstrictors, inotropes, and anticonvulsants have all been investigated using animal models and in clinical practice.[2 6] Although we were reluctant to infuse adrenaline because of fears about the vasoconstrictor effects on the marrow, studies have shown that adrenaline exerts a systemic effect within 20 seconds and atropine exerts its effect more rapidly via the intraosseous route than via the intratracheal route.[6] Sodium bicarbonate can be infused safely, although an increased risk of osteomyelitis has been reported with hypertonic solutions.[2 4]

Fluid flow rates under gravity infusion vary between 1 and 25 ml/minute, but at 40 kPa (300 mm Hg) pressure flow can be increased to 40 ml/minute via a 13 gauge needle.[6]

In the UK intraosseous infusion is seldom used despite the extensive literature on the subject. Even for those with experience of paediatric resuscitation intravascular access may be impossible to achieve, and for the trainee called to resuscitate a child the prospect of placing in intravenous cannula rapidly can be daunting. Intraosseous infusion provides reliable access to the circulation even in inexperienced hands and more than justifies the small risk of complications if intravasular access is difficult. However, this route should only be used in extreme emergencies, in cases where no venous access has been achieved, and after adequate ventilation has failed to improve the child's condition. In this case we are in no doubt that the child would have died if we had not used an intraosseous infusion. We believe that bone marrow infusion needles should be stocked on all paediatric resuscitation trolleys.

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Meningitis caused by human herpesvirus-6

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Abstract

Since the discovery of human herpesvirus-6 (HHV-6) the illnesses associated with it have increased steadily. Two infants with meningitis are reported: both suffered a mild meningitis and serological studies confirmed an acute HHV-6 infection. This report supports a role of HHV-6 in nervous system disease.

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Human herpesvirus-6 (HHV-6) was first reported by Salahuddin *et al* in 1986.[1] After its discovery illnesses linked to HHV-6 have been reported and include roseola infantum, hepatitis, lymphadenitis, mononucleosis, atypical polyclonal lymphoproliferative disorder, and haemophagocytic syndrome.[2–4] Here we report two cases of roseola infantum with meningitis, which to the best of our knowledge has not been reported before.

Case reports

CASE 1

A 7 month old girl presented with fever of three days' duration and a mild cough. Physical examination showed only pharyngitis, an appreciably bulging anterior fontanelle, and mild hepatomegaly (3 cm palpable below right costal margin). Lumbar puncture showed 18 mononuclear and 2 polymorphonuclear cells/mm^3. Her cerebrospinal fluid protein concentration was 0·35 g/l and glucose 3·05 mmol/l. During her stay in hospital the peripheral white cell counts ranged from 6·9 and $12{\cdot}0\times10^9$/l and lymphocytes accounted for 60 to 90% of them. Atypical lymphocytes represented 12% of all white cells initially and reached a peak of 25%.

Serological studies showed no evidence of infection of Epstein-Barr virus, cytomegalovirus, or *Toxoplasma gondii*. Virus culture of cerebrospinal fluid was negative. Antibody to HHV-6 was determined by indirect immunofluorescence assay using HHV-6 (U1102 strain) infected J Jhan cells as antigens. The IgG anti-HHV-6 was tested as reported by Salahuddin[1] and IgM anti-HHV-6 as the method of Niederman *et al*.[3] The first serum taken on the fourth day of illness was negative for IgG anti-HHV-6 but positive for IgM anti-HHV-6 (titre 1:10). The second serum taken 11 days later was