Clinical Article

Ischaemia of the foot in infants

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Abstract

Ischaemia of the foot in infants is a cause for concern leading to gangrene, amputation and medicolegal inquiry. The causes of gangrene are usually complicated and multifactorial.[1] The gangrene usually develops following a severe bacterial or viral infection. Septicaemia is usually accompanied by dehydration, shock and severe metabolic derangements. Mechanical causes include invasive vascular procedures and venipuncture especially in newborns.

The gangrenous change may imply surgical error especially when the infections occur following surgery or plaster cast immobilisation.

Children who survive these infections are at a higher risk for complex orthopaedic problems later with growth. Involvement of the physeal circulation due especially to bacterial septicaemia may take several years to manifest resulting in longitudinal and transverse growth problems.[2] Ischaemic insults to the developing skeleton result in gangrene, skin necrosis and irregularities of epiphysis, metaphysis and physis with premature physeal closure. This may require skin grafting (and later release of contractures), amputation (and later revision of stump) and orthopaedic procedures to address deformity and leg length discrepancy.

It is important for the orthopaedic surgeon to become involved early in the treatment team to decide on fasciotomy, amputation level, prosthetic fitting and anticipated long-term growth problems.

Aim

The aim of this paper is to highlight the various presentations and causes of gangrene that we encountered in four infants in the orthopaedic paediatric unit between 1996 and 2008.

Children who survive these infections are at a higher risk for complex orthopaedic problems later with growth

Case reports

Case 1

A 10-month-old baby with arthrogryposis and severe club feet underwent posteromedial releases of the feet. The child was immobilised in plaster slabs with crepe bandages, with the feet in the under-corrected position postoperatively with a view to plaster change in 2 weeks to obtain full correction. The circulation was good for 2 days. On the third day the child had bouts of severe diarrhoea and became dehydrated overnight; the right foot turned blue and the left showed ischaemia around the incision site superficially. The child was found to have a high fever, raised white cell count, low albumen and low haemoglobin. A paediatrician diagnosed chicken pox as the cause. On the fifth day the child underwent right below knee amputation; the wound healed well, the left foot healed with scarring. Medicolegal inquiry did not prove negligence or surgical error. The child wears a below-knee prosthesis at 8 yrs follow-up.

Case 2

A one-week-old baby was treated for bilateral club feet with a first stage Ponseti-type plaster cast to correct the cavus deformity only. The foot was in equinus and the knee flexed 90° in the cast. The circulation remained good for 3 days. The grandmother applied herbal medicine to the umbilicus because the child was restless. On the fourth day the feet developed a bluish discoloration. The plaster was removed at a nearby hospital. The feet became gangrenous up to the ankle and the child was referred for admission (*Figure 1*). On examination the child had fever, dehydration and septicaemic shock. The white cell count, K^+, Na^+, urea and blood sugar were high. The haemoglobin and platelets were low. Following paediatric intensive unit care, antibiotics and rehydration, the condition improved. *Escherichia coli* was cultured from blood specimens. The child underwent bilateral Symes amputations with healing of the skin and was lost to follow-up after 1 month. Local inquiry from hospital management did not prove negligence or poor plaster application as the cause of the gangrene. Septicaemia with severe metabolic derangement was concluded as the causative factor.

Case 3

A two-year-old baby was admitted to an intensive care unit and treated for fever for 3 days. The child developed gangrene of both feet (*Figure 2*). Examination revealed high fever, septicaemia and dehydration. The white cell count was high and haemoglobin was low. Lumbar puncture was negative. The blood culture revealed *Neisseria meningitidis*. The child underwent bilateral midfoot amputations four weeks later following demarcation, with healing of the skin. Follow-up is 10 months.

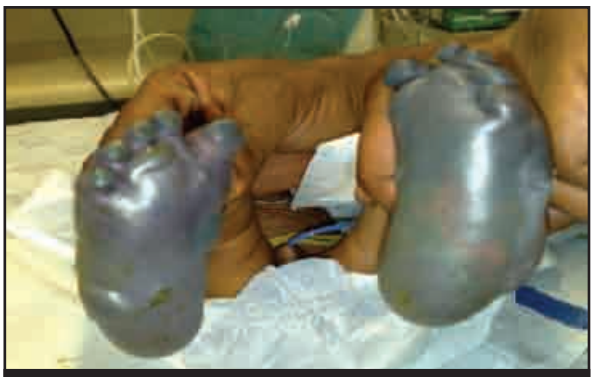

Figure 1: Bilateral symmetrical gangrene of both feet following *E. coli* septicaemia

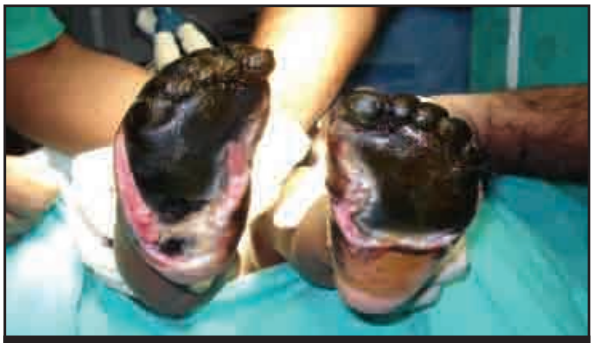

Figure 2: Bilateral; symmetrical gangrene of both forefeet following *N. meningitidis* septicaemia

Case 4

A three-month-old baby was treated at a rural hospital for 1 week for gastroenteritis, meningitis and bronchopneumonia with antibiotics and intravenous fluids. The child was then referred to the paediatric surgical unit with gangrenous toes and fingertips. Blood culture revealed *Neisseria meningitidis*. The white cell count, urea and K^+ were raised. The haemoglobin, albumen and platelets were low. The gangrene spread up both legs into the thighs. The surgical unit performed fasciotomies of both lower limbs. Two weeks later the child underwent below knee amputation on the left tibia and metatarso-phalangeal amputations on the right foot. Several skin grafts were done to the left thigh. He was referred 5 years later to orthopaedics with 'skeletal dysplasia'. Examination revealed stunting of growth, short stature, short lower limbs and severe flexion contracture of the left knee and bilateral genu varus deformities. Radiographs revealed old healed changes in the metaphyses and epiphyses of the left distal radius, left proximal femur, and both distal femoral and proximal tibial regions (*Figure 3*). He underwent several procedures to facilitate weight bearing including soft tissue releases of the knees, tibial osteotomies and finally left through knee amputation. He is ambulant with a prosthesis at 15 years of age.

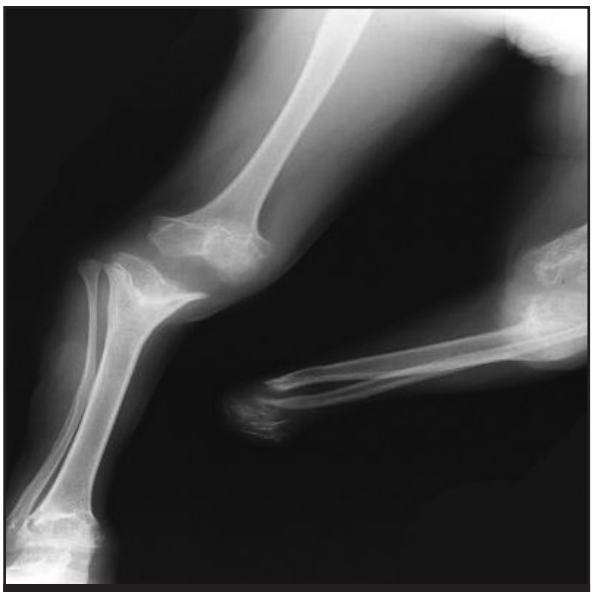

Figure 3: Radiograph showing epiphyseal, metaphyseal and physeal changes around both knees 5 years later following *N. meningitidis* septicaemia

Discussion

Peripheral limb ischaemia resulting in gangrene is rare in children. It has been observed in neonates and infants with birth asphyxia, invasive vascular access, venipuncture, gastroenteritis, and severe bacterial and viral infections. The causes resulting in gangrene are multifactorial and complicated.[1]

In this study all patients had evidence of acute infection as probably the initial cause of irreversible changes in the lower limbs. Such infants develop dehydration and shock resulting in increased blood viscosity, sludging of erythrocytes in the capillaries, poor tissue perfusion, cellular hypoxia, disseminated intravascular coagulation and thrombosis. Biochemical derangements resulting in vasculitis include hypocalcaemia, hypernatraemia, hyperkalaemia, and raised blood sugar and urea levels. Chemical changes occur in the blood indicating metabolic acidosis and endocrine derangements.[2] The ischaemia is usually symmetrical. Thrombocytopaenic purpura, purpura fulminans or post-infectious thrombosis with gangrene has been reported in infants following infectious diseases.[3,4] Children with bacterial septicaemia, especially due to meningococcus, are at high risk for complex orthopaedic problems with acute or chronic manifestations.

Neisseria meningitidis contains a lipopolysaccharide in its walls that acts as an endotoxin to elicit an acute inflammatory reaction.[3,4] This initiates a diffuse vasculitis and disseminated intravascular coagulation resulting in occlusion of small vessels, haemorrhagic lesions and gangrene. Secondly a discrete inflammatory response occurs in trabecular bone resulting in abscesses in the metaphyseal vessels similar to acute haematogenous osteomyelitis.[2,4,5] Chondro-osseous damage with resulting metaphyseal, physeal and epiphyseal growth disturbances may become evident much later and may resemble metaphyseal and epiphyseal dysplasia of bone.[6,7]

Several reports have documented radiological changes associated with vascular insults to the developing skeleton. These findings have included radiographic irregularities in the epiphyses, metaphyses and physes resulting in physeal arrest, destruction of ossification centres and chondro-osseous deformity with cup-shaped metaphyses. These changes can be mistaken for skeletal dysplasias, sickle cell anaemia, infantile pyogenic osteomyelitis and trauma.[7,8]

Patients can present with leg length discrepancy and angular deformity several years after the deformity, as in case 4.

Gangrene of the feet may also develop in association with viral infections such as chicken pox (varicella gangrenosa). The child may or may not become toxic and demonstrate thrombocytopaenia. Macroscopic and microscopic changes have been observed in the major vessels occluded by the thrombus.[9,10]

Several other risk factors must also be considered in our environment as contributing factors to the cause of gangrene. These include vasculitis from retroviral disease, and the use of herbal enemas and herbal medicines.

Epidemics of ergotism caused by the ingestion of bread or cereal made from rye contaminated by the fungus *Claviceps purpurea* or ergot have been described.[11] In some parts of Africa diseases are treated by oral herb preparations, enemas, scarification and inhalation. Inhalations used for respiratory disorders, made from herbs, were considered to be the causative factor in 12 infants with peripheral gangrene in a study by Noyez *et al*.[12] A substance causing severe vasospasm similar to the gangrenous extremities seen from ergotism was used in these children.

Invasive vascular access such as arterial catheterisation venipuncture and umbilical catheterisation has been reported as a direct cause of gangrene of the limbs.[13-15] One study found 2% of neonates developed gangrene during infusion through umbilical catheters.[16,17]

Mechanical and anatomical factors must be considered in infants undergoing orthopaedic treatment as these may be implicated in medicolegal cases. These include prolonged tourniquet time, compression from crepe bandages and plaster casts. Hootnick found absence of the dorsalis pedis and tibialis posterior vessels in vascular studies in club foot.[18] These abnormalities must be considered in cases especially where surgical error is implicated.[19] Patients with arthrogryposis may have poor vasculature and may be prone to vascular insults intra-operatively and postoperatively with plaster cast immobilisation.

Dehydration and infection may further compromise the peripheral circulation.

Tight plaster casts are well known to compromise the circulation in orthopaedic patients especially if the foot is forcefully dorsiflexed and the knee extended. However in the first stage of casting using the Ponseti method the foot is plantarflexed and supinated and the knee flexed thereby relaxing the posterior tibial artery and the popliteal artery. Immobilisation of the feet during septicaemia may have contributed to the gangrene in case 2.

It is important for the orthopaedic surgeon to be involved in the treatment and planning as soon as gangrene is observed in children. Early fasciotomy and the planning and selection of skin graft donor sites should be performed to avoid contractures and discomfort when prosthetic fitting is done. The level of amputation must be planned. Treatment should involve the paediatrician, haematologist, plastic surgeon and physiotherapist.

The children should be transferred to the paediatric intensive-care unit. Management should include aggressive fluid replacement, intubation, ventilation, antibiotics and ionotrope support. Digits should be allowed to demarcate and autoamputate.

It is important for the orthopaedic surgeon to be involved in the treatment and planning as soon as gangrene is observed in children

The role of fasciotomy is controversial. It has been suggested that fasciotomy should be performed within the first 24 hours from the onset of purpura fulminans, and only in patients without bleeding diathesis.[20] Davies *et al* recommend that fasciotomies should be made with as short as possible incisions in the skin, but the fascial release should be extensive.[20]

Bache and Torode[21] found that distal perfusion was not improved by fasciotomy. At surgery, the deep tissues were invariably non-viable, and multiple procedures may be required. The eventual level of amputation may be higher than if the limb segment had been allowed to demarcate prior to surgery. Fasciotomy should be reserved for patients in whom ischaemic changes are not evident.

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