GANGRENE AND THROMBOSIS IN AN INFANT WITH CONGENITAL HEART DISEASE

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Only some fifty cases of gangrene in infants have been recorded in the literature, and the vast majority of these have occurred in the neonatal period (Dohan, 1934). The onset of gangrene after this period may be in association with severe infections (Watkins, 1938) or congenital heart disease (Gross, 1945). Current interest in cyanotic congenital heart disease warrants recording a case with this rare, but possibly preventable, complication.

Case Report

The patient, a male infant aged ten weeks, was admitted to hospital with a history of dark coloration of his left foot for four days, and of the right foot for one day.

He was born by breech delivery (birth weight 7 lb.) following a full-term normal pregnancy. Both parents and six siblings, including a twin sister, were healthy.

He had been cyanosed from birth, and was first seen in the out-patient department at the age of one month when it was noted that he had an enlarged heart and a harsh systolic murmur over the whole praecordium. The cyanosis was uniform and crying made the child breathless, but there was no evidence of congestive failure.

An accurate diagnosis of the congenital heart lesion was not attempted at this time, and he was seen at regular intervals up to the age of seven weeks. The general condition remained good but there was a failure to gain weight in spite of an adequate caloric intake.

For five days before the onset of the change in the feet the baby had been reluctant with his feeds. His mother stated that he was feverish during this time. He was, however, again feeding normally when admitted.

Examination. The weight was 6 lb. 12 oz., and the rectal temperature 97.4° F. The child was generally cyanosed and fretful but not dehydrated. Well demarcated gangrene of the feet (figs. 1 and 2) was present, and there was slight pitting oedema of the hands and of the legs below the knees. The arms were held in flexion, and there was a marked increase in muscular tone throughout the body.

Cardiovascular system. Pulsation could be felt in both femoral arteries but was not detected in the

popliteals. There was no distension of the veins in the neck. The pulse rate was 150 per minute and was regular. The heart showed enlargement clinically, and the systolic murmur had remained unchanged.

No abnormality was found in the lungs, abdomen, or central nervous system, and there was no evidence of infection.

Investigations. The red blood cells numbered 6,000,000 per c.mm. of blood (haemoglobin 14 g. per cent.), and the white cells 6,500 per c.mm. (polymorphs 47 per cent., lymphocytes 42 per cent., monocytes 1 per cent.).

The urine was normal and blood culture negative. A radiograph of the chest (Dr. Lodge) showed generalized enlargement of the heart. The vascular pedicle was small. The appearance was suggestive of transposition of the great vessels (fig. 3).

In view of the poor prognosis no specific therapy was undertaken. The child gradually became more apathetic and prone to dyspnoeic attacks. Oedema of the limbs spread to the trunk, but there was no extension of the gangrene. Death took place on the thirteenth day after admission.

Post-mortem report. There was oedema of the legs, sacrum and back, as high as the tenth rib, with gangrene of both feet as seen clinically.

The heart was enlarged, the right auricle being approximately twice the size of the left. There was transposition of the aorta and pulmonary artery. There was no endocarditis. The ductus arteriosus and foramen ovale were patent. There was thrombosis of the inferior vena-cava as high as the renal veins, in the internal iliac veins, and down to below the knees into the gangrenous areas. Several small vessels over the surface of the brain were thrombosed and the choroid plexus on both sides was grossly distended and thrombosed. The internal iliac and femoral arteries were patent and no arterial thrombosis was noted other than that in the popliteal arteries.

The respiratory and alimentary tracts, liver, spleen, pancreas, kidneys, adrenals, thymus, thyroid, lymph glands, and bones were normal.

Histology. In the lung some alveoli were collapsed, and many cells contained fat globules. The brain showed degeneration with many Hortega cells, and thrombosis in the vessels of the choroid

plexus. The blood vessels showed no change in structure; the intima appearing to be normal. Thrombus in some of the veins of the leg showed early organization.

Histology of the liver, kidneys, pancreas, and umbilicus showed no remarkable changes.

Discussion

Embolism and local disease of the blood vessels are unlikely causes of the gangrene in this case in view of the findings. Peripheral vasoconstriction, which may occur in states of severe heart failure (Fishberg, 1938), is a possible mechanism, but there is no evidence in favour of this.

The main feature at necropsy was the thrombosis, which affected (1) the vessels of the lower limbs extending in the case of the veins as high as the inferior vena cava, and (2) the choroid plexus and vessels over the surface of the brain. This wide distribution suggests that some general factor was responsible.

Thrombosis is a complication of cyanotic congenital heart disease and it is particularly prone to occur when polycythaemia becomes extreme, as in dehydration (Taussig, 1947).

The onset of gangrene here coincided with a period when fluid intake had been low for several days, and it seems probable that thrombus formation at this time occluded the blood supply to the feet. This would support the contention that every effort should be made to avoid dehydration in children with cyanotic congenital heart disease.

Summary

A case of gangrene of the feet in an infant aged ten weeks with cyanotic congenital heart disease is described. The possible causes leading to this complication are discussed.

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FIGS. 1 and 2.—Photographs showing gangrene of the feet.

FIG. 3.—Chest radiograph.