VENOUS GANGRENE

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Gangrene of vascular origin results when the blood flow through the capillaries is brought to a standstill, either permanently or for a period long enough to cause cellular death. It may be due to a lesion of the arteries to the part, of the capillary bed itself, as in frostbite, or to occlusion of the veins preventing the outflow of blood from the area.

The term 'venous gangrene' is reserved for those cases of gangrene of a limb which occur in the presence of a venous occlusion but in which the arterial tree remains patent. The diagnosis can be made with certainty, therefore, only if the arteries are shown to be patent by examination of the vessels after amputation or death, or by arteriography, but increasing recognition of this rare condition in the past decade has led to the description of clinical features on which the diagnosis should be made. It is important to recognize the condition, for, if unsuspected, the appearance of the limb may lead to an unnecessary amputation.

The essential lesion in venous gangrene is a massive thrombosis of the veins of the limb (phlegmasia coerulea dolens), though gangrene does not invariably follow such an event, for even a few patent channels suffice to maintain a sluggish circulation. The lower limb is the one commonly affected and the lesion is bilateral in one-third of the cases. Only rarely is the upper limb involved but it may be so, alone or in addition to one or both lower limbs. The condition may occur in patients who appear to be healthy, but in nearly every case there is present some disease well recognized to predispose to thrombophlebitis. Of these, the most common are present or past local phlebitis, ulcerative colitis, pelvic infections and carcinoma of abdominal viscera, particularly of the pancreas, stomach or pelvic organs, but cases have followed retrograde injection of varicose veins at the time of internal saphenous vein ligation, ligation of the iliac veins or inferior vena cava, blood diseases and even intravenous infusion.

Massive thrombophlebitis may occur as a single dramatic episode in a previously healthy limb, the so-called pseudo embolic type of onset from its

supposed resemblance to arterial embolism. However, the clinical features of massive thrombophlebitis and of arterial embolism are very different, and, in my opinion, this nomenclature has given rise to much confusion, for the two conditions resemble each other only in the sudden onset of pain and weakness in a limb. Those cases which have been recorded as resembling arterial embolism appear, from the descriptions, to have been examples of a relatively localized femoral vein thrombosis with associated arterial spasm, as shown by the pale cool limb, the moderate oedema, and the rapid improvement following lumbar sympathetic block, removal of clot from the femoral vein or other antispasmodic measures. The more usual mode of onset, however, is of one or more episodes of thrombophlebitis over a period of a few days before the final widespread occlusion takes place.

The initial feature is sudden pain which in most cases begins at the root of the limb and radiates distally. Of a burning or bursting character, it is very severe and is only partially relieved by morphine. Almost simultaneously the limb takes on a cyanotic hue which deepens steadily until within an hour of the onset the distal parts appear deep violet or black. The colour persists on elevation and even firm pressure may fail to produce blanching which, if it occurs, is only momentary. Before long, petechial haemorrhages can be seen in the skin and later, heavily blood-stained blisters may appear.

The limb swells rapidly and, if the lower extremity is affected, gross oedema may extend to the buttocks and lower abdominal wall, for the thrombosis usually involves the iliac veins. The rapid loss of fluid into the limb leads to oligæmic shock, and circulatory failure has caused death more often than subsequent pulmonary embolism. Initially the limb is warm and the distal pulses, though weaker than in the contralateral limb, are palpable, but as the circulation slows the limb cools and pulsation can no longer be felt. Tenderness develops over the main veins and in the muscles, and there may be changes in skin sensation varying from hyperaesthesia to anaesthesia

which, as one would expect, are most marked distally.

Moist gangrene, if it is going to occur, is apparent by the third or fourth day and is usually limited to the digits. Rarely it reaches the elbow or knee. Thereafter, as the circulation becomes gradually re-established, pain is relieved, the cyanosis fades, swelling diminishes, the limb warms and gangrenous areas demarcate. Dilated collateral venous channels may become visible on the abdominal wall or, in the case of the upper limbs, around the shoulder girdle. Most patients subsequently require permanent support of the limb to prevent gravitational oedema, but occasionally, as in the case described below, the limb returns apparently to normal.

The clinical pathology of the condition is of practical importance, for misconception of the lesion has led to the advocacy of ineffective and possibly dangerous methods of treatment. The conditions, known severally as phlegmasia alba dolens, phlegmasia coerulea dolens and venous gangrene, are increasingly severe degrees of venous thrombosis with or without coincident venous and arterial constriction. The role of vascular spasm is disputed and, undoubtedly, is too readily suggested as the explanation of circulatory problems but that it can and does occur occasionally is shown by the following evidence.

Veins have an afferent and efferent sympathetic nerve supply, and de Sousa Pereira demonstrated that a chemically-induced phlebitis causes pain and may lead to venous spasm extending far beyond the affected segment of vein. Both pain and spasm are relieved by block or section of the sympathetic fibres to the limb: de Bakey *et al.* found a marked reduction in pulse volume in a limb when the venous pressure was increased significantly by isolating a segment of the main vein, and were able to prove that this was due to reduction in the elastic properties of vessels caused by mechanical distension. Extending the scope of the experiment, they showed that if the isolated segment were injected with sclerosant, the pulse volume was still further reduced, but returned to the level following simple ligation when sympathetic denervation of the limb was effected.

Clearly, then, a degree of spasm which will reduce the arterial supply to a limb can be produced experimentally and there is ample evidence that similar phenomena occur clinically, though by no means regularly. First, the peripheral veins of patients suffering from oligæmic shock are sometimes constricted to such an extent that an intravenous infusion will not run. That this is due to spasm and not solely to reduced blood flow is shown by the immediate dilatation of the vein and rapid flow of the infusion when 2 cc. of 2 per

cent. procaine are introduced into the vein through the infusion needle. Secondly, surgical ligation of the femoral or iliac vein produces moderate oedema which is usually transient and in no way compares with the oedema of phlegmasia alba dolens. Direct comparison in this way, however, is misleading, for, in the former condition the occlusion affects only a millimetre of vein and allows full use of collateral veins, whereas in the latter a long length of vein is occluded. Nevertheless, there is good evidence of reduced arterial input with adequate venous channels in that the limb is cold and white in a feverish patient, and arterial pulses are impalpable or greatly reduced in volume, yet following sympathetic block the limb becomes warm and pink, the pulses improve, and though the thrombotic occlusion remains, oedema rapidly subsides.

Finally, there is visual evidence. Sutton has described a case of ischaemia of the forearm following intravenous infusion into the median basilic vein. Exploration showed the brachial artery to be in intense spasm, relieved at once by the application of papaverine, but no evidence of arterial injury or of extravenous infusion fluid was found. Several cases of femoral vein thrombosis on exploration have shown the femoral artery to be narrow and pulsating only feebly, but returning to normal when the clot was removed from the vein. Most dramatic of all is the case of Clain and Nussbaum. This patient was admitted with a line of demarcation already present and amputation was performed at midthigh level. The femoral artery was seen to be small and pulseless, but as soon as it was ligated and divided, the proximal end pulsed vigorously.

Pulses in the limb may be reduced or impalpable because of previous arterial occlusion, the peripheral circulatory failure present, the pressure of oedema, arterial spasm, or because of filling of the venous bed. The skin temperature is proportional to the blood flow and, if the limbs are compared under identical conditions, cooling merely confirms reduced arterial inflow to the part. Arteriography (in the absence of previous arterial occlusion) demonstrates patent arteries but this does not exclude spasm, since the contrast medium is a vasodilator and the injection of contrast intra-arterially in a normal limb is followed by flushing of the skin of the limb. Examination of limbs, in cases of venous gangrene after death or amputation, confirms patency of the arteries and shows complete occlusion of all the veins leaving the gangrenous area. Experimentally, Fontaine and de Sousa Pereira have demonstrated that it is possible to produce venous gangrene but that to do so, because of the vast number of venous connections, all the veins leaving a limb must be interrupted.

What then is the sequence of events? It would

seem from the evidence described that thrombosis of the main vein leads to the typical features of a phlebothrombosis with a warm, moderately oedematous and perhaps cyanosed limb; thrombophlebitis with spasm to phlegmasia alba dolens; massive thrombophlebitis sparing a few veins to phlegmasia coerulea dolens; and occlusion of all veins to venous gangrene. So long as a venous channel, however tortuous, remains, gangrene does not ensue, and this explains why gangrene of this type is nearly always restricted to the digits where the arterial inflow is least and is most easily arrested by occlusion of the veins. Thus, too, is explained the high incidence of previous attacks of thrombophlebitis in these cases, for fewer patent veins remain to be occluded in the final episode.

In a condition which carried a mortality approaching 30 per cent. from acute circulatory failure, the first essential is to restore the depleted blood volume and to correct the haemoconcentration which also occurs. Whole blood is lost to the circulation by clotting, but the principle loss, as in burns, is of water, electrolytes and plasma proteins into the oedematous tissues. Transfusion of blood will certainly correct these losses, reduce haemoconcentration, and is the measure usually adopted, but, theoretically, by so doing it is possible to produce polycythaemia with its attendant risk of inducing spread of the thrombotic process, and I believe it would be better, therefore, to infuse dextran and saline. Dextran is preferred to plasma because it has a slight anticoagulant effect, because none of the protein lost to the circulation is lost to the body, and because the risk of homologous serum jaundice is avoided.

The second object of treatment is to restore normal circulation to the limb as quickly as possible. The limb should be elevated to encourage drainage from it, yet not to such an extent that the arterial input is further embarrassed. An angle of 45° has been found satisfactory, and it is most important that no pressure falls on the buttocks, so that full advantage is taken of the anastomoses between the profunda and gluteal veins, and that there is no undue angulation at the femoro-iliac junction. To improve drainage further, it is recommended that passive movements be carried out frequently from the start of treatment, and active movements as soon as they become possible.

The limb should be exposed to room temperature and a fan arranged to play a stream of air over it. In this way, the oxygen requirements of the tissues are reduced, the limb is kept dry, thereby reducing the risk of infection, and to some extent, pain is relieved, though analgesics are always required. To reduce the tendency to further thrombosis, anticoagulant therapy is advocated, using

heparin for several days, until any gangrenous area is demarcated, and thereafter discontinue until the patient becomes ambulant.

Antibiotics cannot possibly reach the distal parts of the limb in sufficient concentration to influence infection in the early stages, but, nevertheless, should be given in order to maintain a blood level high enough to be effective when the circulation begins to improve, and so limit any loss of tissue to that destroyed by circulatory arrest. To prevent infection in the early days, reliance must be placed on keeping the limb dry and exposed to the air.

In most cases the tissue loss is small and superficial and, if the temptation to intervene early is resisted, it is surprising how much apparently devitalized tissue will recover. Only when no further spontaneous improvement is taking place is it justifiable to debride the area and replace skin loss.

The use of antispasmodic measures is not recommended since they cannot be expected to benefit organic occlusion of the veins and, in reported cases of undoubted venous gangrene, have not led to improvement. In addition, the abolition of arterial tone is potentially dangerous in such a limb. Operative removal of clot from the femoral vein is likewise, most unlikely to clear any of the distal veins and is not indicated.

Two cases of venous gangrene have been seen in this unit in the past two years, and the following illustrative case history is recorded by permission of Professor Rob, under whose care the patient was admitted.

Case History

A girl, then aged 12 years, had been admitted to hospital in August 1954 with a history of diarrhoea during the preceding two months. Sigmoidoscopy and barium enema confirmed the clinical diagnosis of ulcerative colitis. A course of medical treatment had little effect, but she was discharged as she was considered unsuited psychologically to prolonged stay in hospital. She was re-admitted on March 21, 1956, for pain and swelling of the left leg which had begun two days previously. She was still passing four to five motions daily. A diagnosis of venous thrombosis was made and discontinue therapy was commenced. On March 25, 1956, the oedema was more extensive and extended into the thigh, the foot was blue and no arterial pulsation was felt below the femoral. She was transferred to St. Mary's Hospital with a diagnosis of femoral artery thrombosis.

On admission she was found to be a very precocious child of 14 years, and most anxious about the condition of her limb. The pulse rate was 124 per minute and the blood pressure 105/55. The

whole left lower limb showed marked, pitting oedema. The foot was cyanosed and the remainder of the limb red. The capillary circulation in the foot and toes was quite satisfactory, although only the femoral pulse could be felt. It was concluded that this was a case of moderately extensive venous thrombosis and that the distal pulses were obscured by oedema. Anticoagulant therapy was continued and the foot of the bed raised. The following morning the patient complained of pain in her limb, which on examination was found to be cyanosed as high as the groin. The foot was almost black in colour, the oedema no longer pitting, and capillary circulation in the toes had ceased. The angle of elevation was increased to 45°, and massage of the limb carried out as the patient refused to permit passive movements: 24 hours later, the colour of the proximal half of the foot had improved and blisters were appearing distally. By April 11, 1956, all oedema had subsided, and the peripheral pulses were palpable, but there was skin loss over the distal half of the foot and the toes were mummified with dry gangrene. On May 7, 1956, demarcation was clear and the toes were removed. The foot was subsequently covered with split skin grafts and, when the

patient was last seen, in March 1957, the foot was soundly healed and the limb free of oedema, though the patient had worn no supporting bandages. The ulcerative colitis was still active.

The second case has already been reported elsewhere by Moore and Scott.

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