THE POSTPHLEBITIC SYNDROME

By PAUL T. DECAMP, M.D., and ALTON OCHSNER, M.D.

From the Department of Surgery, School of Medicine, Tulane University, and the Ochsner Clinic, New Orleans, Louisiana

The postphlebitic syndrome is a chronic disabling condition of the lower legs which occurs following thrombophlebitis of the deep veins and which may be associated with edema, pigmentation, secondary superficial varicosities, dermatitis, cellulitis, fibrosis and recurrent ulceration.

Etiology

By definition, the initiating etiologic factor is extensive thrombophlebitis which may occur spontaneously or following a precipitating episode such as operation, parturition, systemic infection or trauma. Classically the acute attack is accompanied by severe pain, swelling and fever. Actually the initial symptoms are frequently mild and the onset may be marked only by the insidious appearance of edema. In diagnosis caution must be exercised because studies in our clinics indicate that occasionally chronic edema and all the complicating features usually associated with the postphlebitic syndrome may occur spontaneously or following injury in the absence of any anatomic or physiologic evidence of venous thrombosis. In view of this, it is difficult to assess accurately the relative importance of venous thrombosis and recanalization, as against secondary factors such as infection or trauma in the development of the subsequent postphlebitic syndrome.

Predisposing Factors

The fact that phlebitis, or any of the other precipitating factors, rarely gives rise to a similar chronic disabling condition elsewhere in the body suggests the importance of anatomic or physiologic characteristics of the leg. It has been suggested that the arterial circulation is poor in this area, but we know of no anatomic studies to support this contention. Certainly in all cases of known arterial insufficiency of the lower extremity, nutritional difficulties are first encountered in the toes and distal portions of the feet rather than in the lower leg. In the postphlebitic leg extensive fibrosis may lead to a secondary arterial insufficiency.

In the erect position the hydrostatic pressure in the veins of the lower extremity is very high,

on the order of 120 cm. of water at the ankle in a man of average height, whether or not the valves are competent.¹² Pressures of this order are unique to this area and must be complemented by pressures of similar magnitude in the extravascular spaces, within cells themselves, and actually in all tissues in the area. Healing of any injury in this area is slow. The importance of the pressure factor becomes more apparent when it is realized that most of the difficulties in healing in this area can be obviated by the sole expedient of assuming the horizontal rather than the vertical position. On the other hand, that pressure alone is not all important is evident, because it has been demonstrated that ligation of the proximal vein (such as the inferior vena cava for septic pelvic thrombophlebitis) in the absence of venous disease in the leg itself may result in persistent high recumbent venous pressure¹⁴ and high erect and ambulatory venous pressures⁴ for periods of six to eight years with minimal edema as the only complication.

Pathogenesis

The acute attack of thrombophlebitis is associated with inflammation of the vein wall and the perivenous tissues including the lymphatics. The more extensive the thrombotic process and the more severe the inflammation the greater the danger of subsequent complications. Sympathetic nervous overactivity results in severe arteriolar spasm which further increases the tendency to edema.^{3, 13} Prompt effective treatment in relieving vasospasm, arresting the spread of the thrombotic process, and the direct control of edema favourably influences the subsequent course.

Persistent edema sets the stage for complications. Infection plays a major role in postphlebitic and related syndromes. Streptococci^{8, 11} find fertile soil in the edematous tissues and cause repeated cellulitis spontaneously or following injury. Bacteria may enter the area through breaks in the overlying skin or through interdigital lesions of epidermophytosis, which is so commonly present. Staphylococcal organisms may cause stubborn pyoderma. With frank ulceration all types of

bacteria gain entrance to the tissues. The infection may involve skin, subcutaneous tissues, the lymphatics, and the veins and perivenous tissues. This leads to pigmentation and eczematoid changes in the skin, and to induration, fixation and contraction of the subcutaneous tissues. Because the inflammatory reaction is commonly chronic, the presence of infection is frequently not appreciated on clinical examination.

Recanalization of the thrombosed veins may occur, although valvular function is irreparably lost.⁷ Subcutaneous veins of the leg become dilated, sclerotic and tortuous and are associated with incompetence of the valves of the superficial and communicating veins as well as of the deep veins. That these varicosities assume pathological significance is attested by the fact that when ulcers occur they usually appear over or near them. Since injuries in a normal lower leg heal slowly, it is small wonder that healing of an ulcer developing in such chronically diseased tissues is slow and difficult. With each recurrent episode of infection or ulceration further damage occurs in the tissues with increased deposition of scar tissue.

Symptoms

On careful questioning an accurate picture of the development of the disorder may be elicited from the patient. An overt thrombophlebitis may have occurred spontaneously or following an injury, operation or systemic infection. With the acute attack there is rapid development of symmetrical edema of the extremity. Pain and fever are frequent but not universal accompaniments. Sometimes a thrombophlebitis will be manifested solely by the appearance of edema of a leg over the course of a few days. One cannot, however, assume under such circumstances that the edema is invariably due to phlebitis. The edema often persists or it may disappear completely. With the passage of time pigmentation may appear insidiously or recurrent attacks of pain, tenderness, redness and heat may occur in the leg. A dermatitis of the leg may cause considerable oozing and great discomfort from itching, cracking and peeling. Scaling and itching between the toes herald the presence of epidermophytosis which is aggravated by excessive sweating that accompanies sympathetic overactivity. Varicose veins may develop and be the site of recurrent attacks of superficial phlebitis.

As the subcutaneous tissues contract a feeling of fixation and tightness develops and edema may become less apparent. It is at this time that ulceration may develop spontaneously or, more commonly, following minor trauma or infection. Pain occurs as the result of associated infection. In

our experience 'bursting pain,' allegedly due to a high venous pressure¹, is uncommon. Occasionally, pain is associated with vasospasm and responds to a procaine block of the sympathetic nerves.

Physical Findings

On visual examination the chief lesions are noted in the lower half of the leg. Edema is variable in amount and extent. It may be severe and involve the entire extremity or it may be minimal and involve only the lower leg and foot. The skin of the lower leg and often the dorsum of the foot is frequently dark brown, dry and scaling, or may reveal diffuse eczematoid lesions weeping thin serous fluid. There may be cyanosis of the foot and excessive perspiration due to sympathetic overactivity. The nutrition of the toes is usually adequate and arterial pulsations are usually present. Scars of healed ulcers and fresh open ulcers may be present, usually above the malleoli.

Varicose veins commonly develop. They are most severe in the leg, often leading down to an ulcer. They may also be prominent in the thigh and abdomen following extensive phlebitis. Frequently there is redness, tenderness and induration of acute superficial thrombophlebitis present over segments of these veins. The edematous tissues may be soft or indurated particularly in the lower portion of the leg. In the latter areas varicosities may be invisible but may be palpated as soft channels in the hard tissues.

Diagnosis

Only the history of the acute attack of thrombophlebitis at the onset identifies such a case clinically as a postphlebotic syndrome. The same clinical picture may follow any one of a number of other primary etiologic factors such as trauma, infection, ankylosis, obesity or various neurotrophic disorders. When the history of acute phlebitis is characteristic there need be little doubt as to the diagnosis. Considerable uncertainty must be entertained if edema develops spontaneously or follows major trauma, such as a fracture. Under these circumstances phlebitis may or may not have occurred. The diagnosis may be clarified by peripheral or retrograde phlebography¹⁰ or by measurement of the venous pressure during ambulation.⁵ At the present time a differential diagnosis is not too important from the clinical point of view as treatment is directed empirically at the various complicating features regardless of the initial etiologic episode.

Prevention

At best the postphlebotic syndrome is a chronic disabling disorder. Treatment usually can relieve

the major symptoms and permit a return to normal activity although eternal vigilance is essential. Every effort must be made to prevent the initial thrombophlebitic episode by controlling infection rapidly, maintaining adequate hydration, minimizing or avoiding trauma and keeping patients as active as is consonant with the medical or surgical condition present. If acute thrombophlebitis does develop, every effort should be made to limit its extent by relief of vasospasm by prompt regional sympathetic nerve block with procaine. If thrombosis progresses anticoagulant therapy is indicated. If embolism occurs, proximal vein ligation is performed. Mechanical support is provided by elastic bandages snugly wrapped from the toes to the groin and ambulation is begun promptly. Edema usually can be controlled within a few days by this regime. With any tendency for persistent edema, elastic support to the extremity is continued as long as necessary, and the patient is followed carefully for a number of years to detect early signs of the postphlebitic syndrome. Incipient epidermophytosis is controlled by an appropriate fungicide, such as Desenex powder, applied between the toes once or twice a day.

Treatment

The treatment of the developed postphlebitic syndrome is basically empiric and consists in the appropriate treatment of the complications present in the individual case. Contributing systemic disease is sought for and treated if present. Obesity, so commonly present, *must* be energetically treated and controlled. Edema is minimized by rest and elevation and/or by appropriate elastic support. Rest periods during the day, with elevation of the extremity, will help the ambulatory patient. Exercise of the muscles during prolonged periods of standing will reduce the edema. If the edema is soft it may be readily controlled by elastic bandages or an elastic stocking. If considerable induration is present the use of elastic adhesive bandages is more effective (*vide infra*). Whenever possible in severe cases, prolonged bed rest and elevation should be employed as it is the most effective means of controlling edema. It is followed later by elastic support.

Occasionally persistent vasospasm contributes to the edema. It is evidenced by coldness and cyanosis of the foot with excessive sweating and by blanching on elevation. The diagnosis is confirmed with relief of symptoms by a lumbar sympathetic block with 1 per cent. procaine. Tobacco in any form is interdicted. If a degree of permanent benefit follows one block, a series of blocks is performed. If relief from the initial block is definite but only temporary, sympathectomy is indicated. Sympathectomy is also in-

dicated, particularly in older individuals, if obliterative arterial disease is present. Cellulitis or an infected ulcer is treated by hot moist applications of physiologic saline solution or mild boric acid solution. Systemic anti-bacterial treatment is prescribed. The vigour and duration of the initial course of therapy depends entirely on the severity of the infection and its response to treatment. It is extremely difficult to eradicate infection completely from edematous and damaged tissues. For this reason appropriate prolonged antibacterial treatment is carried out. This includes, finally, intermittent courses one week out of each month for six months. The specific agent is chosen on the basis of bacterial culture and sensitivity studies if open lesions are present. Otherwise empiricism is used. With staphylococcal infections, the sulphonamides are frequently ineffective and one of the newer antibiotics may be necessary. The necessity for sustained antibacterial therapy cannot be over-emphasized. It will prevent recurrent erysipeloid infections which are so prone to occur.

Epidermophytosis of the toes and feet is treated by an appropriate agent such as Desenex powder or ointment. Acute eczematoid reactions of the lower leg are common and are treated by boric acid soaks. Associated infection is treated by antibiotic agents. If the condition is complicated or resistant to treatment, a dermatologic consultation is obtained. Not infrequently an absorptive 'id' reaction appears on the hands or elsewhere in the body. Treatment of the primary dermatitis of the leg is the best treatment for this secondary condition. Excessive dryness of the skin is prevented by periodic application of a bland ointment such as lanolin or zinc oxide. If excessive sweating persists in spite of conservative treatment and aggravates the epidermophytosis or dermatitis, a sympathectomy is sometimes necessary to dry the part.

Varicose veins secondary to phlebitis are commonly more prominent in the leg than in the thigh. They are vigorously treated surgically as they often appear to contribute directly to ulceration. If recurrent deep phlebitis has occurred and the leg appears congested, caution is exercised in dividing the internal saphenous vein in the thigh as it may be an important outflow channel for venous blood. Increasing discomfort in the tightly wrapped leg on walking (a positive Perthes' test) has not proved sufficiently sensitive in our hands. If in doubt, an ambulatory venous pressure test may be performed.^{4, 5} If the ambulatory pressures are higher when the superficial veins are obstructed by a tourniquet, the internal saphenous vein in the upper thigh is preserved. Surgery alone is used to obliterate the superficial veins and the

technics of high excision, stripping, excision of leg veins and subfascial ligation are all employed as indicated. All incompetent perforating veins are divided. Particular care is taken to obliterate veins in the region of an ulcer.

Acute or subacute ulcers are treated conservatively by warm moist dressings, systemic antibacterial therapy, bed rest and vigorous compression therapy when the patient is up. The ulcer itself is dressed with zinc oxide ointment. Kay⁹ has suggested that the effectiveness of zinc oxide ointment is due to the action of ionized zinc inactivating a tryptic enzyme of bacterial origin frequently present in the ulcer. The persistent empiric use of zinc oxide in ointments, boots and dressings over many years is interesting.

Direct compression of the ulcer is helpful and may be obtained by placing a thick sponge rubber over the dressing and under the snugly wrapped elastic bandage. If induration is present, an elastic adhesive bandage is a more effective means of compression. Irritation of the skin is prevented by first wrapping the leg with a gauze bandage impregnated with a gelatin, zinc oxide mixture. Depending on the amount of discharge from the ulcer, and the sensibilities of the patient, such a bandage may be left on for three or four weeks. We have found it more effective than the inelastic gelatin boot.

If the ulcer is chronic and associated with permanent changes in the skin and subcutaneous tissues, a more active treatment regime is employed. It includes bed rest and control of infection followed by a lumbar sympathectomy to increase the blood flow in the fibrotic tissues and to facilitate wound healing. A few days later, as a one-stage procedure, appropriate obliteration of the superficial varicose veins is performed (*vide supra*), the ulcer and the underlying and surrounding indurated tissues are excised down through the deep fascia and a split thickness skin graft is applied. In these cases the tissues are irreparably damaged and local ablation and grafting are the best means of therapy. These patients require careful postoperative care and prolonged elastic compression of the leg. If radical treatment is contraindicated the conservative programme outlined above can be carried out. With conservative treatment the ulcers will heal, but the treatment programme is prolonged and the end result is less secure.

We now seriously question the advisability of ligation of the deep veins (superficial femoral or popliteal) for the postphlebotic syndrome even if valvular incompetence and reflux venous blood flow is demonstrated. Benefit has been claimed on the basis of alleged reduction in venous pres-

ures during ambulation. To our knowledge no one has demonstrated any consistent lowering of the ambulatory venous pressure by such a measure and studies in our clinic⁴ indicated that a collateral venous system with competent valves never develops following ligation of the major deep vein of an extremity. In our experience, ligation of a major deep vein tends to elevate rather than lower venous pressure.⁴ As the clinical results have also been equivocal,^{2, 4, 8} we no longer perform deep vein ligation for the postphlebotic syndrome unless pulmonary embolism has occurred.

Whatever the treatment required to control the manifestations of the postphlebotic syndrome, the patient is never discharged from the care of a physician. Except in the mildest cases, irreparable changes have occurred in the leg and trouble may recur at any time. Infection must be controlled by prolonged treatment. Edema must be overcome by bed rest and/or appropriate elastic support as long as it is present. Elastic stockings are frequently helpful at this stage, but they must be renewed as elasticity diminishes. Another important feature of the unending patient-doctor relationship is that it is the only means of adequately impressing the patient with the need for everlasting care and vigilance. He is instructed that any injury to the leg or any evidence of inflammation or irritation demands an immediate consultation.

With this programme the majority of the patients are made comfortable and can return to reasonable activity. In the severe cases the sooner the patient can adjust to the idea that he must be careful with his leg indefinitely the better. Actually the majority of those who have had severe symptoms and recurrent ulcers are happy enough to be up and about and comfortable.

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