Postphlebitic syndrome after hip arthroplasty

43 patients followed at least 5 years

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We reviewed 43 patients using clinical scoring for evidence of postphlebitic syndrome at least 5 years after hip replacement. All had had a venogram as part of a screening study at the time of surgery. The postphlebitic syndrome was identified in 13 patients and was disabling in 6. The syndrome was present in 9 of the 11 patients with a venographically proven deep vein thrombosis (DVT) and in 4 of the 32 without DVT. Postphlebitic sequelae had developed despite anticoagulant therapy for the acute DVT.

The postphlebitic syndrome following asymptomatic deep vein thrombosis is an important long-term complication of total hip replacement.

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Submitted 93-11-21. Accepted 94-07-12

Patients with postoperative venous thrombi after hip replacement are mostly asymptomatic and are unlikely to be diagnosed or treated prior to discharge from hospital (Hoek et al. 1992, McNally and Mollan 1993). The postphlebitic syndrome is a serious condition causing disability in those with end-stage disease (Immelman and Jeffery 1984). Most patients with proven symptomatic DVT will develop the syndrome 5–10 years after thrombosis (O'Donnell et al. 1977, Browse et al. 1980, Strandness et al. 1983, Lindner et al. 1986). These patients consume considerable resources from a wide range of health care providers (O'Donnell et al. 1977, Skene et al. 1992).

The late effects of asymptomatic DVT are largely unknown. Clinical and hemodynamic assessment of patients who had asymptomatic DVT after general surgery showed early evidence of postphlebitic syndrome after only 5 years (Andersen and Wille-Jørgensen 1991). Little is known of the incidence of postphlebitic syndrome after hip arthroplasty.

This study was designed to investigate the occurrence and severity of the syndrome in patients who have had total hip replacement at least 5 years previously.

Patients and methods

A group of 82 patients who had primary cemented Charnley hip replacement in 1986 and 1987 were investigated with ascending venography of their operated leg 10 days after surgery as part of a screening study for deep vein thrombosis (Brown 1991). This investigation and the clinical data from the original study provided an objective assessment of the individual's venous system at the time of leaving hospital after hip surgery.

All 82 patients were sent a questionnaire on symptoms and signs of venous insufficiency and were invited to attend a follow-up examination in Musgrave Park Hospital. 13 patients had died since surgery and 9 did not reply. Of the 60 who replied, 24 women and 19 men agreed to attend for assessment, while 17 patients were unwilling or unable to attend. All 43 attending patients were studied at least 5 years after the operation and had a mean age of 73 years. On the questionnaires, there was no difference in age or in reported venous symptoms between those who attended and those who did not attend the clinic.

Venographic diagnosis

Venography identified new deep vein thrombosis in 8 of the 43 patients (2 proximal thrombi, 2 proximal and calf thrombi, 3 major calf thrombi and 1 minor calf thrombus) with old thrombosis in 3. These 3 patients gave histories of DVT after pregnancy in 2 cases and a pulmonary embolism 17 years before surgery in the third. 32 patients had no venographic evidence of thrombosis after the hip operation. Patients with new proximal or major calf thrombosis had been treated initially with intravenous heparin

Table 1. Frequency of symptoms and signs in operated legs in 43 patients 5 years after hip replacement

Symptoms and signs	Clinical score[a]	Number of patients
Secondary varicose veins	1	10
Swelling above the ankle	2	9
Skin pigmentation	2	14
Ankle flare	2	11
Venous claudication	3	7
Lipodermato-sclerosis	4	3
Venous ulceration	5	4

[a] After Browse et al. (1980)

Table 2. Incidence and severity of postphlebitic syndrome 5 years after hip replacement related to venographic diagnosis 10 days postoperatively

	Clinical score[a]			
Venographic diagnosis	0	1–3	4–9	10–19
No DVT	18	9	4	1
DVT before surgery	1	0	1	1
New DVT	1	0	5	2

[a] After Browse et al. (1980), 0 no symptoms or signs; 1–3 non-specific symptoms (mild ankle swelling, skin pigmentation, minimal varicose veins); 4–9 moderate postphlebitic limb; 10–19 severe postphlebitic limb.

and then oral warfarin for at least 3 months. All but 2 patients were asymptomatic for acute DVT and no patient had signs or symptoms of postphlebitic syndrome at discharge from hospital.

Clinical assessment

Clinical findings were scored (MMcN and GMcA) using a modification of the system described by Browse et al. (1980) (Table 1). A history of general aches and pains was given 1 point in the original scoring system. In our study, many patients had vague musculoskeletal pain attributable to degenerative joint disease and we did not accept this as evidence of postphlebitic syndrome. Our score was therefore based on a maximum of 19 points.

Postphlebitic syndrome was diagnosed when a total clinical score of at least 4 was found and when this included 1 or more specific signs of venous dysfunction (ulcers, claudication or lipodermato-sclerosis). It was graded as moderate (4–9) or severe (>10). Neither investigator was aware of the original venographic diagnosis or of the results of the non-invasive investigations performed prior to scoring.

Information was also collected on the range of treatments obtained by patients specifically for venous-related symptoms since their hip operations.

A noninvasive vascular assessment was also performed, using transcutaneous oxygen measurement ($tcPO_2$), liquid crystal thermography and venous occlusion plethysmography.

Statistical analysis was performed using a chi-square text.

Results

Clinical assessment

13 patients were considered to have sufficient evidence for a diagnosis of postphlebitic syndrome. The mean score in this group was 8.5 vs. 0.63 in patients without postphlebitic syndrome. There was no difference in age or length of follow-up between the groups.

Of the 32 patients without DVT at the time of surgery, 4 had developed a postphlebitic limb. In the 8 patients with new DVT after total hip replacement, 7 had developed postphlebitic syndrome. Only the single case of minor calf DVT had not produced venous compromise. 2 of the 3 patients with DVT present before total hip replacement developed postphlebitic syndrome (Table 2).

41 of the 43 patients were asymptomatic after surgery and of these 11 had thus developed postphlebitic syndrome at five years after operation.

6 patients with postphlebitic syndrome were disabled by the condition with reduced exercise tolerance and activity. This was invariably due to venous claudication and/or painful ulcers. 2 of these patients stated that they had gained no benefit from total hip replacement, having simply exchanged coxarthrosis for deep vein thrombosis and postphlebitic syndrome.

There was no difference in venous blood flow, thermographic profiles or tissue oxygenation between patients with or without post-phlebitic syndrome.

Treatment

11 of the 13 patients with postphlebitic syndrome had sought treatment since total hip replacement (Table 3). Treatment was largely provided in the community, although 4 patients had been admitted to hospital for management of intractable ulceration or painful superficial phlebitis. Prolonged use of graduated compression stockings was the preferred treatment for venous claudication. 1 patient had been maintained on warfarin therapy for 7 years because of recurrent thrombosis on stopping anticoagulation at 3 months.

Table 3. Frequency and range of treatments required by patients for postphlebitic syndrome

Treatment	Number of patients	Frequency of treatment
Graduated compression	5	4–7 years
Ulcer dressings	4	16–36 visits
Drug therapy[a]	4	2 weeks–7 years
Hospital admissions	4	1–4 weeks
GP visit	3	1–10 visits
District nurse visit	3	12–36 visits
Hospital clinic appointment	2	2–96 visits

[a]Antibiotics, non-steroidal anti-inflammatory agents, warfarin

Discussion

The majority of postphlebitic limbs occurred in patients who had asymptomatic proximal vein thrombosis as an acute complication of hip replacement. This high incidence in elective orthopedic patients was unexpected, but it has been shown that 39–50 percent of those with a tibial fracture will develop the syndrome between 5 and 17 years after injury (Willén et al. 1982, Aiken et al. 1987). Our incidence at 5 years compares with the 30.1 percent found by Lindhagen et al. (1984) in patients who were studied 3–5 years after general surgery.

There was a small incidence of postphlebitic limb in patients without venographic DVT and it has been shown that not all postphlebitic limbs follow deep vein thrombosis (Browse et al. 1980, Kistner 1980). These 4 patients may, of course, have developed DVT after discharge from hospital. However, the difference in incidence between the 2 groups was highly significant ($P < 0.001$), suggesting that asymptomatic postoperative DVT does produce postphlebitic syndrome. The correlation of the syndrome with proximal vein involvement was in agreement with the large study by Widmer et al. (1985).

The clinical scoring system was useful and correlated well with the patients' view of the severity of symptoms.

Patients with major thrombi in this series were treated with anticoagulant therapy. This treatment did not prevent long-term venous compromise and a more aggressive approach to the management of postoperative DVT may be required. Thrombolytic therapy for proximal thrombosis does produce thrombolysis and at follow-up prevents the development of postphlebitic limb (Arnesen et al. 1978, 1982, Elliot et al. 1979). In an extensive review of trials, Comerota and Aldridge (1992) showed that modern thrombolysis gave better long-term results than anticoagulation. Side-effects are now comparable to those experienced with heparin therapy for DVT after total hip replacement (Patterson et al. 1989).

The development of disabling postphlebitic syndrome after silent DVT highlights the need for effective thromboprophylaxis in total hip replacement. Asymptomatic proximal thrombosis is not a benign condition and must be identified and adequately treated, not only to prevent pulmonary embolism but also the later postphlebitic syndrome.

Acknowledgements

The authors would like to thank M Broome, and SA Croal and all medical and nursing staff of the Musgrave Park Hospital for their help and support during the study. We are indebted to Mr A A B Barros D'Sa MD FRCS, Consultant Vascular Surgeon, Royal Victoria Hospital, for his help in the study design. M A McNally acknowledges the financial support of the British Orthopaedic Association Wishbone Trust.

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