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FATAL PULMONARY EMBOLISM AFTER TOTAL HIP REPLACEMENT

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A retrospective study of fatal pulmonary embolism (FPE) was carried out in 1,324 cases of total hip replacement (THR), performed during 1969 to 1978. Dextran 70 (Macrodex® 6 per cent in saline, Pharmacia AB, Sweden) was given as thromboembolic prophylaxis. Sixteen patients died within 3 months. Autopsy was performed in 14 cases. Nine died from embolism, which makes an incidence of 0.7 per cent. Autopsy was performed in 8 of these cases. Seven patients died during the second and third week. Five patients had complained of acute chest pain and 4 of them had chest radiograms taken, which were normal. Only one patient had clinical symptoms of deep vein thromboses. Perfusion lung scan was performed as a screening procedure in 3 cases, all of them showing defects typical of pulmonary embolism. Four patients died from FPE, despite heparin therapy for 3–5 days. A comparison between patients with FPE and a control group showed that premonitory attacks of acute chest pain and previous operations for orthopaedic reasons were significantly more common in patients with FPE ($P < 0.001$ and $P < 0.05$), respectively). No difference could be found between the groups concerning blood loss, amount of transfusion, sex, operated side, type of prosthesis and weight.

Key words: hip joint (surgery); pulmonary embolism

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There is mounting evidence that the frequency of venous thromboembolic complications in the postoperative period is increasing in the Western world (Hume et al. 1970). The most plausible reason is the improvement of anaesthetic and operative techniques which have made it possible to perform successful major surgery in elderly people. This applies especially to total hip replacement (Cavendish & Charnley 1972).

Approximately 80 per cent of pulmonary emboli arise without premonitory symptoms of peripheral venous thromboses (Kakkar 1977). Studies of autopsies have shown that most cases of pulmonary embolism are not diagnosed during life and therefore not treated (Fitts et al. 1964, Freiman et al. 1965). Death from attacks of major pulmonary embolism occurs in two-thirds of the cases within 30 minutes of the embolic event, a period too short for successful therapy (Donaldson et al. 1963). Thromboprophylaxis has thus become a necessity in the prevention of fatal embolism in high-risk patients. The incidence of FPE after THR without thromboprophylaxis varies between 1.8 and 3.4 per cent (Salzman & Harris 1976).

The purpose of this study was to determine the incidence of FPE within 3 months after THR with dextran prophylaxis. Furthermore, we wanted to determine possible operative risk factors for pulmonary embolism as well as the significance of acute chest pain as a premonitory symptom.

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PATIENTS AND METHODS

During 1969 to 1978 a total of 1,324 hips were operated on and THR performed by several senior surgeons at the Department of Orthopaedic Surgery, Malmö General Hospital. The sex distribution was 831 women (63 per cent) and 493 men (37 per cent). The average age of the women was 65.6 ± 9.3 and of the men 63.4 ± 9.8. Seven hundred and ten right hips (54 per cent) were operated upon and 614 (46 per cent) on the left side. The types of prostheses used were in 695 cases (53 per cent) the Charnley, in 608 cases (46 per cent) the Lubinus or Brunswik, and in 21 cases (1 per cent) various other types. One hundred and six hips (8 per cent) were operated on for rheumatoid arthritis and 1,218 hips because of osteoarthritis and other reasons.

Ever since studies in our department showed that administration of dextran 70 led to a significant decrease in deep vein thrombosis following hip fractures (Ahlberg et al. 1968, Ahlberg 1969), this anti-thrombotic agent has been used consistently in every THR patient.

During the period 1969 to 1978, 500 ml of dextran 70 (Macrodex® 6 per cent in saline, Pharmacia AB, Sweden) was given intravenously during the operation and 500 ml on the second postoperative day. There has been no primary contraindication to this prevention as patients submitted to surgery have had their cardio-vascular status judged preoperatively.

Blood loss was substituted peroperatively and to a certain extent postoperatively. Postoperative management, including physiotherapy and full weight-bearing, was allowed on the first to third postoperative day.

Screening of deep vein thromboses by phlebography and pulmonary embolism by perfusion lung scan was performed during 1975–1977 in 135 patients.

Heparin given as treatment of thromboembolism was administered i.v. in a dose of 30,000 IU per 24 hours on four or six occasions. No regular tests were made of the effect upon the coagulation system.

In order to calculate the postoperative mortality we investigated every patient withdrawn from the population register within 3 months after the operation.

The actual case books, including autopsy charts and certificates of death, were picked out and scrutinized.

FPE was defined as pulmonary embolism which was the main cause of death.

One hundred patients, out of the 1,315 not exposed to FPE, were chosen at random to serve as a control group. Documents could be found in 94 per cent. Sex, operated side, weight, type of operating position, amount of bleeding and transfusion were recorded as well as the presence of acute chest pain in the postoperative period.

Data were compared using the multiple regression analysis and the chi-square method with Yate's correction.

RESULTS

Sixteen patients (1.2 per cent) died within 3 months postoperatively, the autopsy rate being 14/16. Nine patients had the diagnosis of FPE, which was verified by autopsy in 8 cases. This implies an incidence of 0.7 per cent. The mean time of postoperative survival for patients with FPE was 25 days. The other verified causes of death were bronchopneumonia in 3 cases, cardiac infarction in 2 and cardiac insufficiency in one case.

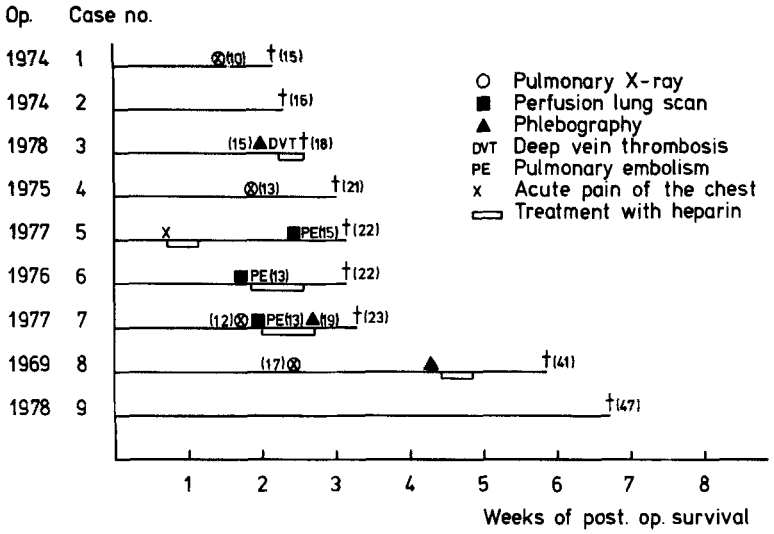

Figure 1. Patients with fatal pulmonary embolism.

Orthopaedic operations had previously been performed in 7 patients of the FPE group and in 32 of the control group. The difference is significant ($chi^2_{(1)} = 4.83, P < 0.05$). Two out of seven patients in the FPE group had had a previous THR. Five patients with FPE had a postoperative attack of acute chest pain compared with 8 in the control group ($chi^2_{(1)} = 10.92, P < 0.001$).

The patients with the diagnosis of FPE are listed in Figure 1. Seven patients died during the second and third week. Five patients had an attack of acute chest pain. Four of these patients had acute chest radiograms taken with negative results. Only one patient with chest pain (no. 5) received the clinical diagnosis of pulmonary embolism and was treated with heparin, which had to be stopped after 3 days because of a complicating haemorrhage. Pulmonary embolism was diagnosed in 3 patients (nos. 5, 6 and 7), who had been screened by perfusion lung scan. This test was positive for pulmonary embolism in every case. Two of these patients were treated for 5 days with intravenous injection of heparin. Patient no. 3 died during heparin treatment. The screening phlebography had shown bilateral thromboses of the vena profunda femoris. In patient no. 7, bilateral phlebography proved to be negative, but autopsy 5 days later showed thromboses on the non-operated side. Only one patient (no. 8) had clinical symptoms of deep vein thrombosis, which was verified by phlebography and found to be situated in the ilio-femoral vein on the operated side. This patient suffered from a hemiplegia, after 2 days of treatment, as a result of which heparin was abandoned. Autopsy was never performed. Patient no. 9 died in her home 5 days after leaving the hospital. No symptoms of thromboembolism were recorded.

Sex, operated side, type of prosthesis, bleeding per- or postoperatively, amount of blood transfusion, age, weight and number of operations in general, or gynaecological surgery in particular, did not differ significantly between the two groups.

There was no case of serious anaphylactic reaction (grade III–IV) or cardiac insufficiency recorded as a result of the administration of dextran 70.

Every patient with FPE had a completed prophylaxis with dextran.

DISCUSSION

Incidence figures of FPE after THR are relatively few. In a controlled study of 900 replacements of the hip, Crawford et al. (1968) found the incidence of FPE to be 1.8 per cent in the control series. This was to be compared with 0.9 per cent in the group with phenindione prophylaxis. However, the benefit was offset by three deaths from gastro-intestinal bleeding in the anticoagulation group. The autopsy rate was 87 per cent. Later, Charnley (1972) reported 1.4 per cent of FPE among early complications during the stay in hospital. In 1973, Coventry et al. reported two cases of FPE verified by autopsy in 58 patients without thromboembolic prevention (3.4 per cent). Johnson et al. (1977) have presented a fatality rate from pulmonary embolism of 2.3 per cent in 1,174 cases of THR without thromboembolic prophylaxis. Phenindione, intravenous heparin and dextran, all reduced the rate of FPE to 1.04 per cent in 7,959 hip arthroplasties. The autopsy rate was 88 per cent. Gruber et al. (1980) found in an international multicentre study, comparing dextran 70 and low-dose heparin, an overall frequency of 1.9 per cent with no statistical difference between the two groups. In a prospective and randomized study, Atik et al. (1970) reported one FPE in patients treated with dextran prophylaxis and 8 in the control group.

The relatively low incidence of FPE in our material (0.7 per cent) is possibly the result of our interest in diagnostic as well as screening procedures, including lung scan and phlebography, which may have contributed to the anticoagulative treatment of some presumptive cases of FPE.

Previous operations for orthopaedic reasons were more common in the group of FPE than in the controls. The reason might be that patients with previous deep vein thrombi have an increased risk of developing new thrombi and emboli. It must not be overlooked that the significant difference might be a result of repeated statistical evaluations. Orthopaedic operations

might also have been better documented than others in the orthopaedic case book.

According to Johnson et al. (1977), premonitory symptoms are very rare in FPE after THR. However, Bell et al. (1977) found in The Urokinase Pulmonary Embolism Trial that chest pain was present in 89 per cent of patients with clinically suspected pulmonary embolism verified by angiography. By scrutinizing the bedside reports in all cases, we found that acute attacks of chest pain had been noted in 5/9 cases. By comparing this rate with that of the control group we found that this premonitory symptom was significantly more common in FPE.

Thus, an acute attack of chest pain in the postoperative period must be taken seriously as being an indication of pulmonary embolism. A perfusion and ventilation lung scan should then be utilized as an objective, diagnostic method of excluding pulmonary embolism.

In 4 cases with acute chest pain, a chest radiogram was performed. The result was consistently negative and no further measures were taken. However, a negative chest radiogram should give rise to an even greater suspicion of pulmonary embolism (Harris et al. 1967, Evarts & Feil 1971). A perfusion and ventilation lung scan is then the only logical course of action, apart from an ECG, which is of immediate interest to exclude cardiac reasons. Stein's findings that there do not seem to be any changes in the ECG specific for pulmonary embolism (Stein et al. 1975) are in agreement with ours. We did not find any consistent changes in the ECG of the 9 patients with FPE.

Minor pulmonary embolism is often asymptomatic. In 3 cases with the diagnosis of pulmonary embolism a perfusion lung scan was performed as a screening procedure. As there are no data regarding the minimum size and number of emboli sufficient to cause death, every case of pulmonary embolism should be considered and treated (Gruber et al. 1977).

The time of onset of FPE in our material corresponds well with the noted maximal onset during the second and third week reported by Johnson et al. (1977) and Sevitt & Gallagher (1961).

Autopsy studies of FPE have shown that between half (Hume et al. 1970) and two-thirds of the cases (Havig 1977) show evidence of previous embolism.

A small pulmonary embolism can be the precursor of an eventual massive embolism, which has been reported to account for 80 per cent of FPE (Sevitt & Gallagher 1961).

Heparin treatment for 3–5 days did not prevent FPE in 3 patients. It is to be assumed that anticoagulative prevention must be extended for a longer period of time to allow the fibrinolytic system to resolve thrombi and emboli. The heparin treatment was not standardized in our study which explains the irregular duration of the treatment. We have now decided to extend the anticoagulative treatment to a period of 6 weeks. However, FPE can occur soon after the diagnosis of pulmonary embolism and the initiation of heparin treatment (patient no. 3).

Several reports have documented proximal thrombi in the ilio-femoral veins to be the main source of pulmonary embolism (Sevitt & Gallagher 1961, Havig 1977). Seventy-five per cent of FPE have been found to originate in the caval and ilio-femoral segment (Havig 1977). The two positive phlebographies in our material both had thrombi in the femoral vein. This location of deep vein thrombosis is common in patients after THR in contrast to patients undergoing general surgery (Nillius & Nylander 1979). To prevent, diagnose and treat these thrombi seems to be an all-important goal of thromboprophylaxis in THR.

In our study, FPE was the most common cause of death within 3 months after THR. Greater efforts seem to be necessary in order to assess the premonitory symptom of acute chest pain. A lung scan, as soon as possible, is recommended as the most simple and objective way of excluding pulmonary embolism. Treatment of pulmonary embolism should include anticoagulants for an adequate period of time.

CONCLUSIONS

1. The incidence of FPE was 0.7 per cent in 1,324 patients, all treated with dextran prophylaxis.

2. FPE was the most common cause of death within 3 months after THR.
3. Previous orthopaedic operations are a point to be noted concerning the possibility of developing FPE after THR.
4. Acute attacks of chest pain in the postoperative period are an important symptom, which should be followed by a lung scan in order to rule out pulmonary embolism.
5. Chest radiogram alone is inadequate for the evaluation of acute chest pain after THR.
6. Heparin therapy for a short period of time did not prevent FPE in cases with a scintigraphic diagnosis of pulmonary embolism.

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