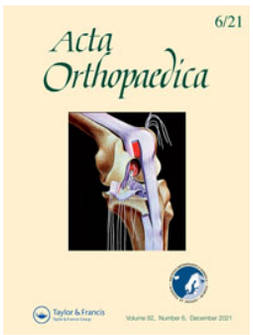

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FAT AND THROMBOEMBOLISM AFTER TOTAL HIP REPLACEMENT

JERZY M. SIKORSKI[1] & JOHN W. BRADFIELD[2]

[1]Department of Surgery (Orthopaedic Surgery), The University of Western Australia, Nedlands, Western Australia; [2]Pathology, University of Bristol, Bristol, England

Three patients who died after total hip replacement were subjected to a necropsy examination. The object of this was to determine whether there was any evidence of a relationship between fat emboli produced by the surgery and thrombotic complications.

All three patients showed extensive fat embolisation in the lungs and antemortem thrombosis. In two of these the morphological relationship between these two phenomena was such that it seems likely that the fat emboli preceded and caused the thrombosis.

Key words: fat; hip; thromboembolism

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The operation of total hip replacement (THR) is occasionally complicated by embolic phenomena in the postoperative period. Two quite distinct forms have been recognised. Thromboembolism occurs in about 6 per cent of all cases (Dorr et al. 1979) and this may be fatal in about 2 per cent (Crawford et al. 1968). Fat embolism may also occur. When a prosthesis is inserted into the femoral shaft, through methylacrylic cement, very high pressures are generated in the medullary cavity (Breed 1974) which force marrow fat into local draining veins (Pelling & Butterworth 1973, Harris et al. 1975) and from there into the pulmonary circulation (Modig et al. 1975). These changes are unique to replacement arthroplasty involving a long bone and do not occur if the cavity of the bone is not sealed with cement (Breed 1974). Clinically a full-blown and potentially-fatal fat embolism syndrome may result (Harris 1975, Letournell et al. 1971, Adams et al. 1972, Hyland & Robins 1970, Burgess 1970, Cohen & Smith 1971, Phillips et al. 1971, Kepes et al. 1972, Dandy 1971, Gresham et al. 1971, Sevitt 1972).

While there is no doubt that the two forms of embolic syndrome may present in quite distinct ways and that they may occur quite independently of one another, there is a possible connection between the two. Fat if introduced into the venous circulation is thrombogenic (Connor et al. 1963) and in the experimental situation will produce pulmonary thromboemboli with enmeshed fat globules (Sikorski et al. 1977). A limited necropsy study has been undertaken to see whether a similar relationship could be demonstrated in man. Patients who died after hip replacement were examined to determine whether there was any demonstrable morphological relationship between embolic fat and thrombosis.

PATIENTS AND METHODS

Patients admitted to Winford Orthopaedic Hospital in the years 1972–79 were included in a study on postoperative deep vein thrombosis which has been reported previously (Sikorski et al. 1981). Three patients died in the postoperative period and their next-of-kin consented to a postmortem examination. In these patients an attempt has been made to define the distribution of thrombosis and embolic fat in the venous system, the heart and the lungs.

All thrombi and lung specimens were fixed in 10 per cent buffered formol saline. Sections were cut at 5 μm and stained with haematoxylin and eosin (Gill et al. 1974). Fibrin was demonstrated by the Martius Scarlet Blue method (Lendrum et al. 1962). Neutral triacylglycerol was demonstrated using oil-red-0 (Sikorski et al. 1981).

Case 1

A female aged 72 years had a THR through the posterior approach and 7 days later developed left-sided pleuritic chest pain and dyspnoea. At this stage her ^{125}I-labelled fibrinogen leg scan did not show any evidence of a peripheral vanous thrombosis. In spite of this she was started on full dose intravenous heparin. Her condition deteriorated and she died the following day.

At necropsy she was found to have massive pulmonary emboli in both pulmonary arteries and an area of gross congestion in the right upper lobe. There was no thrombus found in any peripheral vein of either leg, nor in the central veins of the chest, nor in the heart chambers. There was no evidence of intimal damage in the femoral vein of the operated thigh.

On microscopy a recent pulmonary embolus was seen impacted in a branch of the pulmonary arterial system. In the head and tail of the thrombus there were aggregated vacuoles typical of fat vacuoles lost during processing (Figure 1). The head of the thrombus showed organisation by invasion with endothelial cells and fibroblasts (Figure 2).

Case 2

A male aged 64 years had a THR through the posterior approach. He was also given prophylactic subcutaneous heparin, 5000 i.u. before surgery and three times daily. Four days postoperatively he developed an extensive deep vein thrombosis diagnosed by ^{125}I-labelled fibrinogen leg scanning. He had a bilateral ascending phlebogram which confirmed the radio-isotope findings and he was started on full dose intravenous heparin. Two days later he collapsed suddenly and died.

At necropsy there were large pulmonary emboli occluding both pulmonary arteries and the femoral vein of the operated thigh was virtually occluded by a long and poorly-attached antemortem clot. There was extensive thrombosis in the calf veins.

On microscopy a branch of the pulmonary artery showed impacted, recent thromboembolism and inti-

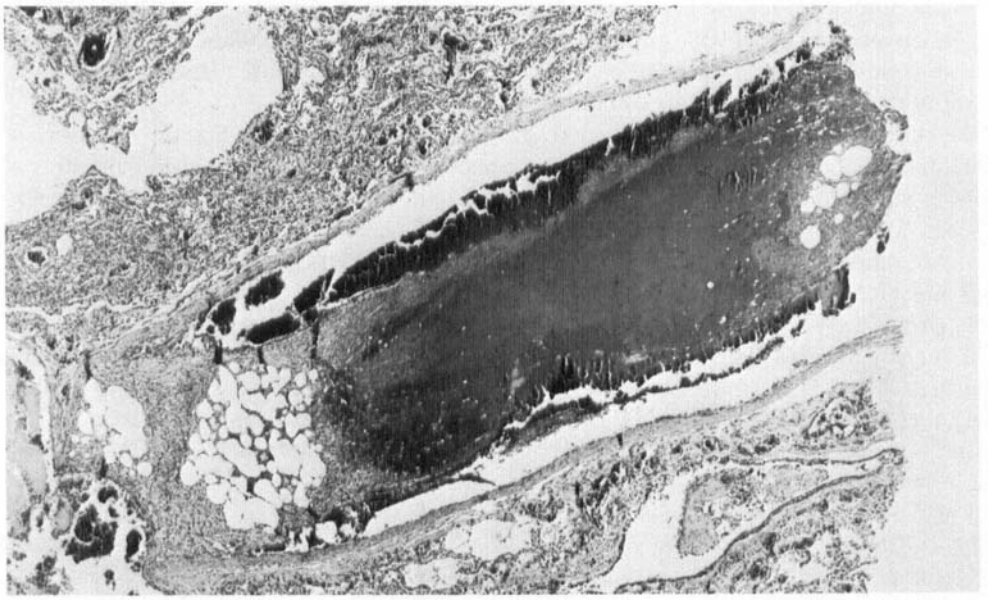

Figure 1. Postmortem appearance of pulmonary thromboembolism in a patient who died 8 days after total hip arthroplasty. The embolic mass is impacted in a pulmonary artery and there is evidence of fat in both the head and tail. (Magnification ×40).

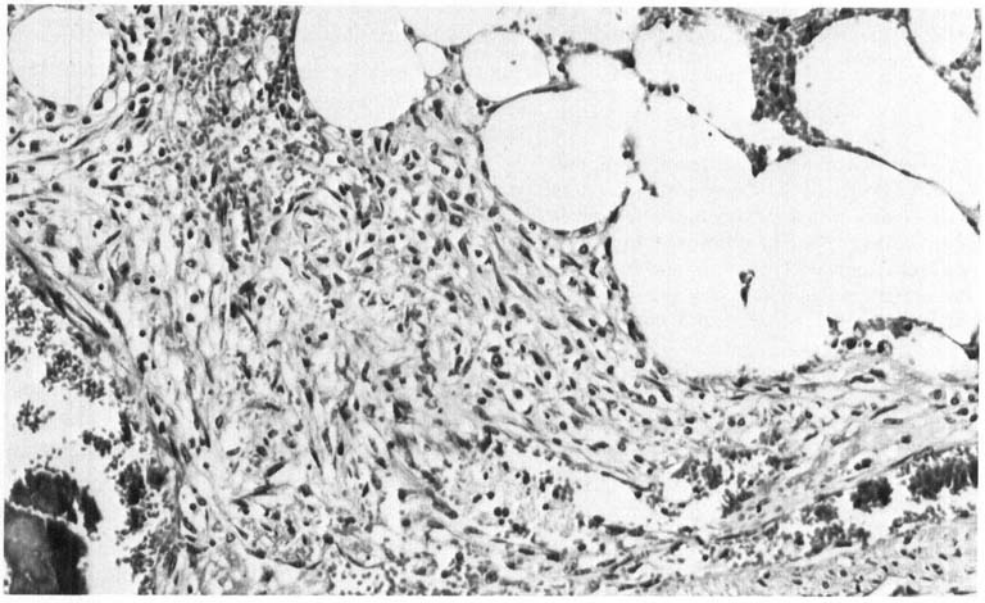

Figure 2. High power view of the head of the thromboembolic mass shown in Figure 1. There is organisation around the head of the fat/thrombus. (Magnification ×450).

Figure 3. Microscopic appearance of the lung shown of a man who died 6 days after total hip replacement. The section of a small pulmonary artery shows impacted thrombus. There are small spaces within the thrombus which could either be left by fat or could represent artefacts. Within the wall of the artery there is extensive infiltration by neutral fat, stained red with oil-red-0 and shown here as black dots. (Magnification ×65).

mal thickening at the point of impaction. There was lipid accumulation in the wall of the artery which gave a positive (red) staining reaction to oil-red-0 (Figure 3).

Case 3

A female aged 65 was mildly hypertensive preoperatively. Following a posterior THR she developed a deep vein thrombosis on the fourth postoperative day, which was diagnosed on the ^{125}I-labelled fibrinogen leg scan. She was not anticoagulated because of the hypertension. On the seventh postoperative day she suffered a left-sided cerebrovascular accident and died on the following day.

At necropsy there was extensive subarachnoid and intracerebral bleeding and a ruptured saccular aneurysm at the trifurcation of the left middle cerebral artery. Examination of the venous system showed a small area of residual thrombosis in the femoral vein and a clot in one of the major soleal veins, all on the side of the hip arthroplasty. There was antemortem thrombus, 1.5 cm × 1.0 cm in the left side of the heart. In the right lung there was a large thrombus occluding the main branch of the middle lobe.

Microscopically there was fat in the small vessels in all areas of both lungs and within alveolar macrophages. No fat was found enmeshed in either the leg thrombus or within the pulmonary embolus.

DISCUSSION

All three of the patients presented here showed both fat globules in the lung and coincident thrombo-embolism. In two of the patients fat and thrombosis were found in close proximity, within the same high power field. In one of these patients a thromboembolus contained fat in its substance and there was good evidence that the fat embolism had occurred at least 1 week prior to death. The degree of organisation seen surrounding the fat vacuoles could not have occurred in less than that time. When the thrombus formed is more contentious, but there are two main possibilities. The whole fat/thromboembolus could have arrived as one embolic mass. This would explain the presence of fat in both head and tail of the embolus. Alternatively the fat embolus could have arrived first and provided a site for local thrombus formation. Either way fat was acting as a thrombogenic influence at a site of intravascular stasis.

The histological appearance of the fat spaces in this patient gives no clue to the chemical nature of the fat. Stains specific for triacylglycerols did not show anything and this suggests that the fat spaces seen in the haematoxylin and eosin preparations may have been left by non-esterified fatty acids.

Triacylglycerol, stained red by oil-red-0, has been demonstrated in the other two patients. In one of these the fat was in close proximity to the thrombo-embolus embedded in large amounts within the wall of a pulmonary artery. This mass of fat occurred at the point of impaction of a thromboembolism. It is impossible to be sure of the exact relationship between this fat and thrombus from the sections produced. The most likely explanation is that a fat and thrombus similar to the one in Figure 1 impacted against the artery wall. The fat was thus dispersed and taken up by the wall of the artery.

In all three cases fat and thromboembolism co-existed and the appearances were very similar to those produced experimentally by the intravenous injection of triacylglycerol (Sikorski et al. 1981).

REFERENCES

Adams, J. H., Graham, D. I., Mills, E. & Sprunt, T. G. (1972) Fat embolism and cerebral infarction after use of methylmethacrylic cement. *Br. Med. J.* **3**, 740–741.

Breed, A. L. (1974) Experimental production of vascular hypotension and bone marrow and fat embolism with methylmethacrylate cement. *Clin. Orthop.* **102**, 227–244.

Burgess, D. M. (1970) Correspondence: Cardiac arrest and bone cement. *Br. Med. J.*, 588.

Cohen, C. A. & Smith, T. C. (1971) The intra-operative hazard of acrylic cement. *Anaesthesiology* **35**, 547–549.

Connor, W. E., Hoak, J. C. & Warner, E. D. (1963) Massive thrombosis produced by fatty acid infusion. *J. Clin. Invest.* **42**, 860–866.

Crawford, W. J., Hillman, F. & Charnley, J. (1968) A clinical trial of prophylactic anticoagulant therapy in elective hip surgery. Internal publication No. 14. Centre for Hip Surgery, Wrightington Hospital.

Dandy, D. J. (1971) Fat embolism following prosthetic replacement of the femoral head. *Injury* **3**, 85–86.

Dorr, L. D., Sakimura, I. & Mohler, J. G. (1979) Pulmonary emboli following total hip arthroplasty: Incidence study. *J. Bone Joint Surg.* **3**-A, 1083–1087.

Gill, G. W., Frost, J. K. & Miller, K. A. (1974) A new formula for a half-oxidised haematoxylin solution that neither overstains nor requires differentiation. *Acta Cytol.* **18**, 300–311.

Gresham, G. A., Kuczynski, A. & Rosborough, D. (1971) Fatal fat embolism following replacement arthroplasty for transcervical fractures of the femur. *Br. Med. J.* **2**, 617–619.

Harris, N. H. (1975) Correspondence: Cardiac arrest and bone cement. *Br. Med. J.* **3**, 523.

Harris, N. H., Miller, A. J., Bourne, R., Wilson, S. & Kind, P. (1975) Experimental investigation of fat embolism after the use of acrylic cement in orthopaedic surgery. *J. Bone Joint Surg.* **57**-B, 245–246.

Hyland, J. & Robins, R. H. (1970) Correspondence: Cardiac arrest and bone cement. *Br. Med. J.* **4**, 176–177.

Kepes, E. R., Underwood, P. S. & Becsey, L. (1972) Intra-operative death associated with bone cement. *JAMA* **222**, 576–577.

Lendrum, A. C., Fraser, D. S. & Slidders, W. (1962) Studies on the character and staining of fibrin. *J. Clin. Path.* **15**, 401–413.

Letournell, E., Vacher, D. & Temkine, J. (1971) L'emboli graisseuse, complication a redouter dans l'arthroplastie totale de genou. *Presse Med.* **79**, 2479–2481.

Modig, J., Olerud, S., Malmberg, P. & Busch, C. (1975) Medullary fat embolisation during total hip replacement arthroplasty. *Injury* **5**, 161–164.

Pelling, D. & Butterworth, K. R. (1973) Cardiovascular effects of acrylic bone cement in rabbits and cats. *Br. Med. J.* **2**, 638–641.

Phillips, H., Cole, P. V. & Lettin, A. W. (1971) Cardiovascular effects of implanted bone cement. *Br. Med. J.* **3**, 460–461.

Sevitt, S. (1972) Fat embolism in patients with fractured hips. *Br. Med. J.* **2**, 257–262.

Sikorski, J. M., Pardy, B. J., Bradfield, J. W. & Dudley, H. A. (1977) The pathophysiological changes of experimental fat embolism: early pre-hypoxemic changes. *Br. J. Surg.* **64**, 6–10.

Sikorski, J. M., Hampson, W. G. & Stadden, G. E. (1981) The natural history and aetiology of deep vein thrombosis after total hip replacement. *J. Bone Joint Surg.* **63**-B, 171–177.

Correspondence to: J. M. Sikorski, Department of Surgery (Orthopaedic Surgery),
The University of Western Australia, Queen Elizabeth II Medical Centre, Nedlands 6009, Western Australia.