MEDICAL MEMORANDA

Fat Embolism and Cerebral Infarction after use of Methylmethacrylic Cement

J. HUME ADAMS, D. I. GRAHAM, E. MILLS, T. G. SPRUNT

British Medical Journal, 1972, 3, 740–741

In view of the current interest in the cardiovascular effects of methylmethacrylic cement (Powell *et al.*, 1970; Peebles *et al.*, 1972) and its implication in the genesis of fat embolism (Hyland and Robins, 1970; Gresham *et al.*, 1971), we report the case of a patient who failed to recover consciousness after Schier's arthroplasty in which methylmethacrylic bone cement was used. Both cerebral fat embolism and infarction within cerebral arterial boundary zones were found at necropsy.

Case Report

A 67-year-old woman who had suffered from rheumatoid arthritis for several years was admitted to hospital for total joint replacement of the left knee. Her preoperative general condition was good: pulse 74/min, B.P. 135/80 mm Hg, E.S.R. 24 mm in the first hour. After routine premedication a Schier's arthroplasty using methylmethacrylic bone cement to secure the prosthesis was carried out. The operation, including the induction time, lasted one hour. No anaesthetic difficulties arose and there was no undue blood loss.

Two and a half hours after operation she had failed to regain consciousness. The blood pressure was 70/50 mm Hg but returned to the preoperative level within 48 hours. During the postoperative period measures were taken to correct a metabolic acidosis and a degree of hypoxaemia (Pa_{O_2} 44 mm Hg, Pa_{CO_2} 21 mm Hg). In spite of this intervention the neurological state failed to improve and she remained unconscious with bilateral extensor plantar responses until she died on the fifth postoperative day. The possibility of systemic fat embolism was considered but there were no petechiae, fat lobules were not found in the urine, and there were no retinal emboli.

At necropsy, apart from oedema and partial collapse of the basal segments of each lung, there were no appreciable macroscopic abnormalities. Histological examination showed extensive pulmonary and visceral fat embolism.

The brain was fixed in 10% formol saline for three weeks before dissection. The cerebral hemispheres were cut into 1 cm slices in the coronal plane after transection of the midbrain. Large blocks of the cerebral and cerebellar hemispheres and the brain stem were embedded in celloidin, and sections cut at 30μ were stained by the method of Nissl using cresyl violet, and by Woelke's modification of Heidenhain's method for myelin. Frozen sections from numerous representative areas of the brain were stained by oil red-O for fat. In both the cerebral and cerebellar hemispheres there were essentially wedge-shaped areas of established infarction affecting both cortex and white matter within the arterial boundary zones (Figs. 1 and 2). In addition frozen sections showed many fat emboli in the brain. There was no evidence of either extracranial or intracranial occlusive arterial disease.

Institute of Neurological Sciences, Glasgow S.W.1
J. HUME ADAMS, M.B., M.R.C.PATH., Professor of Neuropathology
D. I. GRAHAM, M.B., M.R.C.PATH., Lecturer in Neuropathology

Falkirk and District Royal Infirmary, Stirlingshire
E. MILLS, M.B., CH.B., Registrar in Pathology (Present address: University Department of Biochemistry, Royal Infirmary, Glasgow)
T. G. SPRUNT, M.B., F.R.C.S., Consultant Orthopaedic Surgeon

Comment

Gresham *et al.* (1971) described an association between Thompson's replacement arthroplasty using methylmethacrylic cement and a high incidence of fatal fat embolism in the immediate postoperative period, and suggested that fat embolism occurs more often than is recognized. Though the possibility of systemic fat embolism was considered in this case, evidence for it was not found in vivo. Pathological evidence, however, was readily obtained by microscopy.

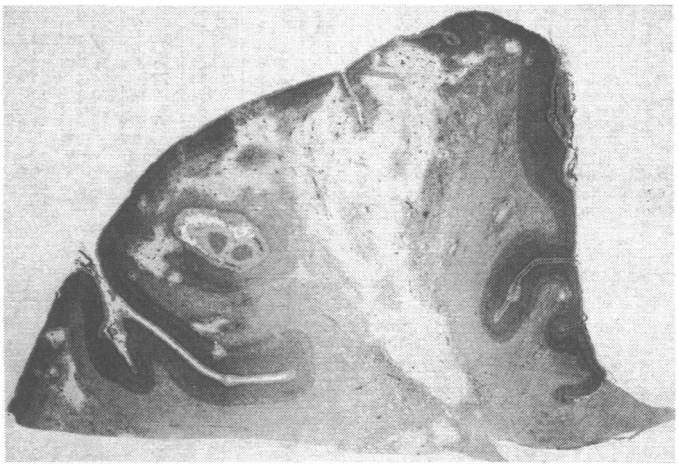

FIG. 1—Left parietal region. Note infarct in boundary zone between anterior and middle cerebral arterial territories. The smaller ischaemic lesions are due to fat embolism. (Cresyl violet. × 1·8.)

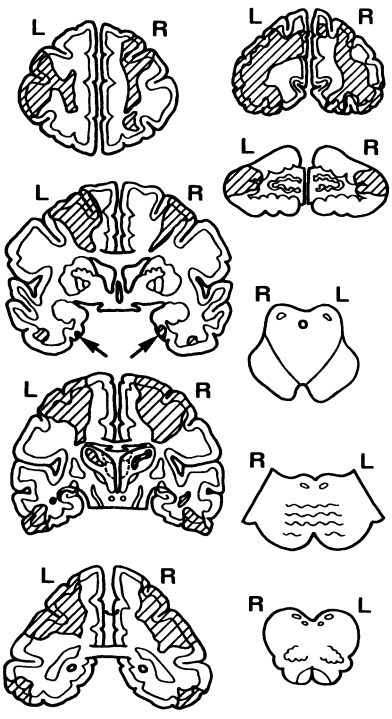

FIG. 2—Distribution of arterial boundary zone infarcts (shaded). There is bilateral infarction between territories of anterior and middle cerebral arteries, between middle and posterior cerebral arteries, and between superior and posterior inferior cerebellar arteries. Arrows indicate pressure necrosis at sites of tentorial herniation.

The cardiovascular effects of methylmethacrylic bone cement include both cardiac arrest (Powell *et al.*, 1970) and hypotension (Peebles *et al.*, 1972). The former tends to produce diffuse neuronal necrosis (Brierley *et al.*, 1971), while the latter is associated with focal ischaemic damage in cerebral arterial boundary zones in both man (Adams *et al.*, 1966) and primates (Meldrum and Brierley, 1971). The neuropathological findings in the present case—circumscribed infarcts affecting all of the major arterial boundary zones in the cerebral and cerebellar hemispheres in the absence of diffuse neuronal necrosis—are characteristic of a severe and precipitate fall in systemic blood pressure. From the observations that have been made previously on the cardiovascular effects of methylmethacrylic bone cement (Peebles *et al.*, 1972), and as the patient failed to recover consciousness postoperatively, there seems little doubt that an abrupt fall in blood pressure occurred at the time of insertion of the prosthesis. The focal nature and distribution of the infarction indicated that the postoperative hypoxaemia, which can be ascribed to pulmonary fat embolism, was not the primary cause of brain damage.

There is an increasing awareness of the significance of arterial boundary zone infarcts, as these lesions have been described not only in patients with systemic hypotension, but also in fatal head injuries (Graham and Adams, 1971). The significance of this case is that it highlights possible damage attendant on the use of methylmethacrylic bone cement in various orthopaedic procedures in the genesis of permanent brain damage.

Addendum

Since this paper was submitted for publication Sevitt (1972) has reported a detailed account of the occurrence of fat embolism in patients with fractured hips, some of whom were treated by arthroplasty utilizing acrylic cement.

References

Adams, J. H., Brierley, J. B., Connor, R. C. R., and Treip, C. S. (1966). *Brain*, **89**, 235.

Brierley, J. B., Adams, J. H., Graham, D. I., and Simpson, J. A. (1971). *Lancet*, **2**, 560.

Graham, D. I., and Adams, J. H. (1971). *Lancet*, **1**, 265.

Gresham, G. A., Kuczynski, A., and Rosborough, D. (1971). *British Medical Journal*, **2**, 617.

Hyland, J., and Robins, R. H. C. (1970). *British Medical Journal*, **4**, 176.

Meldrum, B. S., and Brierley, J. B. (editors). (1971). In *Proceedings of International Symposium on Brain Hypoxia (Carshalton, 1970)*, p. 20. London. Clinics in Developmental Medicine, Spastics International Medical Publications.

Peebles, D. J., Ellis, R. H., Stride, S. D. K., and Simpson, B. R. J. (1972). *British Medical Journal*, **1**, 349.

Powell, J. N., McGrath, P. J., Lahiri, S. K., and Hill, P. (1970). *British Medical Journal*, 3, 326.

Sevitt, S. (1972). *British Medical Journal*, **2**, 257.

Erythropoietic Protoporphyria: First Report in an Indian

L. K. BHUTANI, S. G. DESHPANDE, S. K. SOOD

British Medical Journal, 1972, 3, 741–742

Erythropoietic protoporphyria, a recently recognized disorder of porphyrin metabolism, was first described by Magnus *et al.* in 1961. It is characterized by mild to moderate photosensitivity and an excess of protoporphyrins in the erythrocytes and the stools. The condition is alleged to be inherited as an autosomal dominant character (Schmid, 1966). A large number of cases have by now been reported from different parts of the world. To our knowledge, however, there has been no previous report of erythropoietic protoporphyria from India. In reporting such a case we also wish to draw attention to certain unusual features which were present.

Case Report

A 7-year-old Sikh boy, the product of a consanguinous marriage, presented with a history of recurrent asymptomatic vesiculobullous lesions on the arms, face, and uncovered portions of both legs since the age of 1 year. The lesions healed within two or three days, leaving atrophic and hyperpigmented spots. There was no history of preceding erythema, oedema, or any particular relation to exposure to sunlight. "Peeling-off" of the skin was reported to occur on minor mechanical trauma, though vesicular lesions would appear spontaneously on exposed parts. The urine was reported to be of normal colour. No other member of the family was reported as suffering from a similar condition.

On examination the most striking clinical features were hyperpigmented macular and atrophic hyperpigmented scarred lesions confined to the face, ears, hands, arms, and feet (Fig. 1). The fingers were stunted and showed thickening of the skin over the knuckles (Fig. 2). There was a moderately dense crop of lanugo on the upper lip and chin and on the forehead, where it covered almost the entire area from the eyebrows to the hairline. The forearms and legs were also covered by dense hair. The spleen and liver were not palpable and systemic examination did not show anything abnormal. The teeth, which were normal in colour, showed fluorescence under Wood's light.

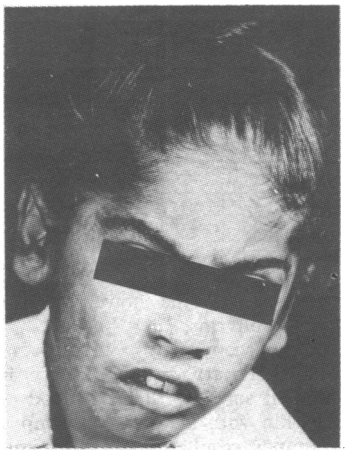

FIG. 1—Hypertrichosis and macular hypopigmented and hyperpigmented lesions.

The haemoglobin level was 10·8 g/100 ml and the packed cell volume 35%. A peripheral blood smear showed normal erythrocyte morphology with little anisocytosis. The total white cell count was 11,400/mm³ (polymorphs 46%, lymphocytes 52%, eosinophils 2%) and the reticulocyte count 0·1%. The serum proteins were 6·4 g/100 ml, serum cholesterol was 200 mg/100

All-India Institute of Medical Sciences, New Delhi-16, India

L. K. BHUTANI, M.D., Associate Professor of Dermatology and Venereology

S. G. DESHPANDE, M.B., B.S., Clinical Resident, Dermatology and Venereology

S. K. SOOD, M.D., Associate Professor of Pathology