SCIATIC NERVE PARALYSIS FOLLOWING ANTICOAGULANT THERAPY

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A woman aged sixty-seven was admitted to Edinburgh Royal Infirmary in November 1970 with severe mitral incompetence following a myocardial infarction in March 1970. In January 1971 she underwent mitral valve replacement (Starr-Edwards) and as a routine post-operative measure she was given Warfarin. Her progress was uneventful for the first fourteen days after operation and the anticoagulation appeared well controlled. On the fifteenth day she had mild epistaxis and haematemesis; in spite of reducing the dosage of anticoagulants the prothrombin time still exceeded 60 seconds with a prothrombin ratio of >5:1 on the seventeenth day.

She complained of severe burning pain in the left buttock radiating down the back of the thigh, of inability to move the lower limb and of loss of sensation in the lower leg. Examination revealed a large tense fluctuant swelling in the buttock with complete motor paralysis of the sciatic nerve except for the semimembranosus and semitendinosus (power 2), and loss of sensation in the total nerve distribution.

Since this neuropathy had arisen when anticoagulant therapy was out of control it was thought justified to diagnose sciatic palsy from local haemorrhage.

Treatment and progress—The patient was treated conservatively. Anticoagulants were abruptly stopped but vitamin K1 was not administered. Passive exercises were begun and a drop-foot splint was applied. Progress was disappointing; pain persisted though the swelling in the buttock appeared to resolve. No return of motor power was noted, but ten days after the initial onset she had recovered normal sensation except for hypoaesthesia over the sole of the foot and a small area over the left lateral malleolus.

She was discharged from hospital after two months. She had recovered some power of the hip rotators (power 3–4), hip abductors (2) and tibialis anterior (1) and had retained power in the inner hamstrings. There had not been any further change in sensibility.

Three months later she was readmitted after a sudden collapse. She was found to have complete left hemiparesis. A diagnosis of cerebral embolus was made, the embolic source being attributed to the Starr-Edwards prosthesis. The hemiparesis has remained complete and it has been impossible to assess further the sciatic palsy.

DISCUSSION

Neuropathy caused by haemorrhage during anticoagulant therapy is an infrequently diagnosed and poorly understood condition. Early studies of the complications of anticoagulant therapy (Allen 1947; Peyman 1958; Riddick 1960; Pastor, Resnick and Rodman 1962) did not mention it. Groch, Hurwitz, McDevitt and Wright (1959) and Calverley and Mulder (1960) each described a case of femoral neuropathy thought to be due to haemorrhage into the lumbo-sacral plexus. Since 1965 sporadic reports have appeared in the literature describing a variety of neuropathies in patients on anticoagulant therapy (Prill 1965; Lange 1966; Hartwell and Kurtay 1966; Gallois, Dhers and Badarou 1967; Mehrotra 1967; Goodfellow, Fearn and Matthews 1967; Susens, Hendrickson and Mulder 1968; Kettlekamp and Powers 1969; Patten 1969; Parkes and Kidner 1970; Kubacz 1971). The cases reported include neuropathies affecting the femoral nerve (nine), sciatic nerve (seven) and median nerve (three). In one of three cases affecting the lumbo-sacral plexus there was combined femoral, obturator and sciatic palsy.

There appear to be distinctive clinical signs and symptoms which permit early recognition and it may be possible to prevent paralysis by adequate treatment (Patten 1969). The most striking features appear to be the occurrence of pain, the appearance of a palpable haematoma.
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and the rapid onset of complete motor and sensory loss coincidentally with a loss of control of the anticoagulation therapy. Less constant features include prodromal bleeding, such as haematemesis, epistaxis and generalised ecchymoses after previous surgical procedures.

Various authors (Goodfellow et al. 1967, Susens et al. 1968, Kettlekamp and Powers 1969) believe that femoral neuropathy is due to haematoma under the iliac fascia causing a compression syndrome at the pelvic outlet. The femoral nerve is trapped between the inguinal ligament, the iliac haematoma and the ilium, and the resulting neuropathy is explained by compression. Operative decompression appears to confirm this as well as relieving the neuropathy (Goodfellow et al. 1967, Kettlekamp and Powers 1969).

Evacuation of the haematoma and decompression of the nerve might be expected to be effective if the direct pressure and pressure-induced ischaemic effects are reversible. In most reported cases, however, the patients have been treated conservatively, with uniformly disappointing results so far as return of muscle power and disappearance of pain are concerned. Seemingly the correct management of these cases should include cessation of anticoagulant therapy, the administration of vitamin K1 and analgesics, followed by surgical decompression of the affected nerve as soon as the patient’s general condition is suitable.

SUMMARY

1. A case of complete sciatic palsy complicating anticoagulant therapy is presented.
2. A brief review of the possible pathogenesis is made and the importance of early recognition and treatment of the syndrome is emphasised.

I am indebted to Professor J. I. P. James for his encouragement and advice in the preparation of this paper.

REFERENCES


Patten, B. C. (1969): Neuropathy Induced by Hemorrhage. Archives of Neurology (Chicago), 21, 381.


Vol. 54 B, No. 1, February 1972