Aspirin Pretreatment Prevents Post-cannulation Radial-artery Thrombosis

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Aspirin inhibits platelet aggregation,¹ and recent studies have shown it to be effective in lessening mortality and morbidity resulting from a variety of thromboembolic disorders, including transient ischemic episodes² and thromboembolism.³ The role of aspirin in reducing thrombosis related to vascular cannulation is controversial, however, since it does not appear to reduce the incidence of arterial occlusion associated with cardiac catheterization⁴ but is effective in reducing thrombosis in arteriovenous fistulas.⁵ Since radial arterial thrombosis is a frequent occurrence after prolonged percutaneous cannulation for monitoring,⁶ we undertook a prospective evaluation of the efficacy of aspirin pretreatment in reducing the incidence of post-cannulation radial-artery occlusion.

MATERIALS AND METHODS

One hundred patients (mean age 64 years ± 16 SD) scheduled for major elective surgical procedures were interviewed on the day prior to operation, and informed consent for the study was obtained.‡ Allen’s test³ and Doppler ultrasonic examination at that time demonstrated patent radial and ulnar arteries. Identical capsules containing either aspirin (600 mg) or placebo (lactose) were given orally in randomized double-blind fashion with the evening meal. Exceptions to this protocol included 17 patients scheduled for total hip replacement who were taking aspirin up to the time of operation and nine patients scheduled for prosthetic aortic-raft operation who did not receive aspirin preoperatively due to the possibility of hemorrhagic complications associated with combining aspirin and heparin treatment.⁸

Immediately prior to the induction of general anesthesia, percutaneous radial arterial cannulation was performed with 18-gauge teflon⁹ catheters as previously described.⁹ Catheters were continuously flushed with heparinized 0.9 per cent saline solution (2 units/ml) at a rate of 3 ml/h from an Intraflow¹ system.

All cannulas were removed on the first postoperative day (mean duration of cannulation: 26.2 hours ± 0.7 SD). Immediately before decannulation, an arterial blood sample was drawn for platelet aggregation studies and an arteriogram of the cannulated radial artery was obtained by rapidly injecting 3 ml of contrast solution through the cannula while an x-ray of the hand and wrist was obtained. Vessel diameter was measured to the nearest .25 mm, and the amount of thrombus in the lumen was quantitated on a 0–3+ scale by two radiologists who did not know the patients' clinical histories.

Platelet aggregation was studied at the time of decannulation in a light-transmission aggregometer** by adding 0.02 ml of 1:1,000 epinephrine solution to 0.4 ml of the patient's platelet-rich plasma. Twelve patients were found to have either inhibited platelet aggregation without having been given aspirin, or normal aggregation after being scheduled to receive aspirin. Data from these patients were not included in the results. Radial arterial patency was examined daily after decannulation using Allen's test and Doppler ultrasonic technique until the patients were discharged from the hospital (mean follow-up period: 14 days ± 10 SD).

Statistical comparisons were made using the Student t test for nonpaired data and Yates’ correction for chi-squared analysis. P < 0.05 was regarded as significant.

RESULTS

Among the 62 patients in the double-blind portion of the study, 31 received aspirin pretreatment and

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‡ The protocol for this study was reviewed and approved by the Committee for the Protection of Human Subjects in Research of the Mary Imogene Bassett Hospital.

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§ Cathlon IV Jelco Laboratories, Raritan, New Jersey.
¶ Sorensen Research, Salt Lake City, Utah.
** Chronoalog Corp., Broomall, Pennsylvania.

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showed inhibited platelet aggregation. Four of these patients (13 per cent) sustained post-cannulation radial-artery occlusion. Among the other 31 patients who received placebo and had normal platelet aggregation, 12 (39 per cent) experienced arterial occlusion ($P < 0.05$ versus aspirin-treated group). The mean thrombus scores assigned after arteriography were $1.3 \pm 0.2$ SE in the aspirin-treated group and $1.9 \pm 0.3$ SE in the placebo group ($P < 0.05$).

An unexpected finding was that women appeared to benefit more from aspirin pretreatment than did the men. Among all women in the study the incidence of arterial occlusion was 63 per cent (12 of 19) for those who did not receive aspirin, compared with 27 per cent (7 of 26) for men in the control group. This discrepancy is probably related to the smaller size of women's arteries ($2.0 \, \text{mm} \pm 0.1 \, \text{SEM}$ versus $2.3 \, \text{mm} \pm 0.1 \, \text{SEM}$, $P < 0.05$). Aspirin therapy reduced the incidences of arterial thrombosis to 12 per cent for men (3 of 25) and to 22 per cent (4 of 18) for women. Although the reductions in occlusion caused by aspirin were proportionally similar for men and women (56 and 65 per cent, respectively) the difference was not significant for men ($\chi^2 = 0.9, P > 0.05$), but was significant for women ($\chi^2 = 4.75, P < 0.05$).

None of the patients in this study showed evidence of distal vascular ischemia, skin slough over the cannula site, or local infection. Despite initial concern that aspirin pretreatment might lead to an increased incidence of bleeding complications in these surgical patients, there was no instance of unexplained or excessive postoperative hemorrhage. Similarly, none of the patients in either group had a significant hematoma at the site of arterial puncture after the cannula had been removed.

**DISCUSSION**

Aspirin is thought to inhibit platelet aggregation by irreversibly acetyling the enzyme cyclo-oxygenase. This enzyme facilitates the production of prostaglandins (PG) G-2 and PGH-2 from free arachidonic acid, which, in turn, is generated from platelet phospholipid when platelets adhere to collagen, as occurs when intimal damage is induced. Both PGG-2 and PGH-2 cause platelets to release their granule contents and to aggregate. In addition, PGH-2 is converted to thromboxane A-2, which is an even more potent stimulus for platelet aggregation.

Radial arterial thrombosis occurs most frequently when catheters occupy a large proportion of the vessel lumen, resulting in impaired blood flow and damage to vascular endothelium. Patients who have small ($\leq 2 \, \text{mm}$ diameter) arteries thus sustain a higher incidence of occlusion than do patients with larger arteries, and 18-gauge catheters induce more thrombi than do 20-gauge catheters. We used 18-gauge catheters in this study because we felt it was necessary to examine the efficacy of aspirin pretreatment specifically in the situation where cannulas occupy a high proportion of the vessel lumen. Figure 1 shows that aspirin was most effective in reducing the incidence of arterial occlusion in vessels where cannulas occupied more than 30 per cent of the vessel lumen. Since the women in this study had smaller radial arteries than the men did, they likewise sustained greater reduction in post-cannulation radial artery occlusion than the men did.

Recent clinical reports indicate that aspirin may afford greater protection from stroke and venous thromboembolism in men than in women. It is thought that the mechanism of these disorders, however, probably depends on sex-related blood and vascular factors other than platelet activation alone.
particularly since aspirin inhibits platelet aggregation equally in both sexes. Platelet aggregates comprise a major portion of radial arterial thrombi resulting from cannulation, and the present data suggest that inhibition of platelet aggregation is an effective means of reducing the likelihood of development of these occlusive lesions.

Radial arterial occlusion induced by monitoring catheters is a potential source of considerable morbidity. Since we found no evidence suggesting bleeding complications associated with preoperative administration of 600 mg aspirin, we conclude that this treatment is a safe and effective means of reducing the risk of post-cannulation radial arterial thrombosis.

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References


