Salicylate-Induced Bleeding Problem in Ophthalmic Plastic Surgery

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SUMMARY
A 54-year-old woman with clinically asymptomatic PCV underwent eyelid surgery. Twenty-four hours after an uncomplicated procedure she had a severe bleeding diathesis following the ingestion of a small amount of aspirin. Treatment of her bleeding disorder and a review of the effects of salicylates on platelet function are discussed.

INTRODUCTION
The ophthalmic plastic surgeon operates in one of the most vascular areas of the body. A bloody field can markedly prolong surgical procedures, and severe intraoperative and postoperative bleeding can lead to a poor functional and cosmetic result and even visual loss.1

In recent years physicians have become aware of coagulation problems caused by the excess use of aspirin (ASA).1 This commonly used drug can cause severe bleeding during surgery in a richly vascular area such as the orbit or ocular adnexa.

The purpose of this paper is to present a case report of severe postoperative bleeding following an oculoplastic surgical procedure on a patient exquisitely sensitive to ASA. In addition, the effects of salicylates on blood clotting will be discussed.

CASE HISTORY
A 54-year-old white female was referred for a blepharoplasty (Figure 1). Her past medical history was pertinent in that she had known polycythemia vera (PCV) for five years. The disease had run a benign course, and the patient had required only one previous phlebotomy to lower her hematocrit which usually ranged from 48 to 54. She had no previous history of excess bleeding following trauma or surgery, and her general health was otherwise unremarkable. A preoperative physical examination by her internist was normal. Pertinent laboratory data included a hemoglobin of 15.5 grams%, hematocrit 45.9, and a white blood count of 11,400. Differential was said to be normal. Platelets were reported to be slightly in excess. A bleeding time and platelet count were not performed. The patient underwent a rhytidectomy of the upper eyelids and a Byron Smith Modification of the Kuhnt-Szymanowski procedure on the lower eyelids under local anesthesia.1 There was no unusual bleeding during surgery; wounds were sutured securely, and she was sent home in good condition. She was seen 18 hours postoperatively with typical moderate hematoma of the eyelids.

During the next 12 hours, the patient took four Bufferin tablets for a headache. The following morning she began to develop marked swelling of the lower eyelids with bleeding at the wound sites (Figure 2). The bleeding time was found to be 12 minutes, the platelet count was one million, and the patient was hospitalized. On hospital admission the eye findings were as described. The only other physical finding of note was a spleen tip palpable at the left costal margin. The admitting laboratory values were as follows: Hemoglobin 16.3 gram%; hematocrit 51.5. The white count was 13,400 with a normal differential. Platelet count was 1,080,000. The Template bleeding time was 9 1/2 minutes (normal 3 to 7 minutes). Partial thromboplastin time was 42.9 with a control of 33.6. The prothrombin time was 10.8 with control of 10.6 which represents 90% activity. The patient’s excessive bleeding was felt to be due primarily to

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abnormal platelets from her myeloproliferative syndrome significantly aggravated by the ingestion of ASA.

During the next 24 hours the patient required 26 units of platelets and two units of fresh frozen plasma to lower her bleeding time to six minutes. At that point she had a wound dehiscence of both lower eyelids and a large, organized clot in the left lower lid (Figure 3). Once platelet function was normalized and bleeding controlled she was taken to the operating room where organized clots were removed, bleeders were meticulously cauterized, and wounds resutured (Figures 4A & 4B). Fortunately she had a satisfactory functional and cosmetic result (Figure 5). One year later the patient suffered a nondisplaced rib fracture in an automobile accident. She was given two Emerin with codeine tablets by an emergency room physician and sent home. Six hours later she had a total hemotorax on the affected side and required platelet transfusions and a thoracotomy as lifesaving measures.

**DISCUSSION**

This case illustrates the profound effect of ASA on hemostasis when there is an underlying platelet abnormality, in this case, a myeloproliferative syndrome. Platelet function in the control of local bleeding by forming a vascular plug. The mechanism of platelet action can be summarized by the sequence — adhesion, aggregation, release, aggregation and vasoconstriction. When the vascular wall is disrupted, platelets adhere to the injured surface and become sticky. Other platelets then aggregate at the site and undergo a granular synthetic and release action.

This results in the formation of cyclic endoperoxides (PGG2 and PGH2) and thromboxane A2 as well as ADP which are felt to mediate aggregation. Thromboxane A2, in particular, is felt to be the most potent aggregator of platelets and vasoconstriction and, therefore, has the most potent influence on the formation of the platelet plug.

A single 200-mg dose of aspirin acetylates about 90% of platelet cyclo-oxygenase. The enzyme is required in the platelet for the production of PGG2, PGH2, and thromboxane A2. It should be noted that congenital cyclo-oxygenase deficiency and the ingestion of ASA by normal individuals generally result in only a mild prolongation of the bleeding time and usually no clinical bleeding. This suggests that in normal individuals this pathway alone is not essential for adequate platelet function. However, if there is an underlying platelet or hemostatic problem, this effect is amplified, and prolongation of bleeding time and clinical bleeding are much more significant.

Physicians should be aware of the nature of these underlying disorders which may predispose a patient to excessive bleeding during surgery, particularly if the patient takes ASA.

A. Disorders of connective tissue; these would include amyloidosis, scurvy, and Ehlers-Danlos syndrome.

B. Disorders of platelet adhesion which would include uremia, von Willebrand's disease, and Bernard-Soulier syndrome.

C. Disorders of platelet interaction which include disseminated intravascular coagulation, presence of fibrin degradation products, liver disease, thrombocythemia and afibrinogenemia, macroglobulinemia, and the presence of certain synthetic penicillins, particularly Carbencillin and Ticarcillin.

D. Disorders of platelet release, particularly storage pool
deficiency. These include congenital cyclo-oxygenase deficiency, collagen vascular disease, myeloproliferative syndromes, leukemias, ethanol, and a variety of drugs — most notably aspirin, but also including nonsteroidal anti-inflammatory drugs and nitrofurantoins.

Patients in the above categories obviously need to be screened carefully for defects in their hemostatic mechanism, with particular attention to platelet function. This is best assessed by the use of a Template bleeding time, available in most clinical laboratories. A careful personal family and drug history, physical examination, and Template bleeding time constitute the most useful screening procedures to rule out potential platelet bleeding disorders.

REFERENCES