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Abnormal Coagulation Profiles in Tonsillectomy and Adenoidectomy Patients

Jonathan L. Schmidt, MD,* Kathleen L. Yaremchuk, MD,* and Samuel A. Mickelson, MD*

Preoperative coagulation profile screening is routinely performed in otolaryngology before tonsillectomy and adenoidectomy surgery in the United States. Recently there has been controversy as to whether this routine testing is necessary. To evaluate the need for this testing, we reviewed a series of patients with particular attention to abnormal coagulation profiles. Of 91 consecutive patients undergoing tonsillectomy, adenoidectomy, or both, four had abnormal preoperative coagulation profiles. Of these patients, one had von Willebrand disease, one had hypofibrinoginemia, and two had a transient acquired lupus-like anticoagulant. The latter condition, which causes a temporary prolongation of the activated partial thromboplastin time, is discussed in detail along with a review of the pertinent literature. We conclude that coagulopathies occur frequently enough to justify preoperative screening even in the absence of a positive history. (Henry Ford Hosp Med J 1990;38:33-5)

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pproximately 750,000 adenotonsillectomies are performed in the United States annually (1). In this common surgical procedure the incidence of postoperative hemorrhage ranges from 0.6% to 7% (2,3). In an effort to reduce this complication, preoperative determination of the prothrombin time (PT) and the activated partial thromboplastin time (APTT) has become routine in otolaryngology. Although this preoperative evaluation of coagulation has been recommended by many authors (4-8), others disagree. They claim that preoperative coagulation testing has value only when there is a history of abnormal bleeding or bruising (9-11). However, most of this experience concerned the general surgical wound, which is primarily closed, and not the tonsillectomy, which leaves a raw surface to heal by secondary intention. In our clinic all patients undergoing tonsillectomy and/or adenoidectomy have PT and APTT determination as part of their preoperative evaluation. Abnormal values may indicate a serious coagulopathy or a reversible condition such as an acquired coagulation inhibitor.

Coagulation profiles of 91 consecutive patients undergoing tonsillectomy, adenoidectomy, or both (all performed by the same surgeon) were reviewed. Four patients were significantly abnormal; one had von Willebrand disease, one had hypofibrinoginemia, and two had a transient acquired lupus-like anticoagulant.

Methods and Materials

A total of 91 patients underwent tonsillectomy (31 patients), adenoidectomy (28 patients), or both (32 patients) by one surgeon (SAM) in 18 months. Routine preoperative hematologic evaluation included a complete blood count, PT, APTT, and fibrinogen determination. (Coagulation evaluation was performed on an OrthoCoagulab 16S, OrthoDiagnostics, Raritan, NJ.) The normal APTT value (97.5% of the population) is 23 to 36 seconds. Four patients were found to have persistent abnormalities.

Case Reports

Case 1

A 21-year-old female was scheduled for a tonsillectomy for recurrent tonsillitis. One week prior to surgery PT was 12.0 seconds (control 11.3 seconds) and APTT was 38.5 seconds. Repeated on the morning of surgery, APTT was still abnormal at 40.0 seconds. She had no personal or family history of bleeding problems and her only current medication was oral contraceptives (Ortho Novum® 1/35, Ortho Pharmaceutical Corp, Raritan, NJ).

Three weeks later hematologic investigation disclosed that the APTT was normal at 35 seconds, but factor VIII antigen and chromogen were low at 24% (normal 63% to 128%) and 35% (normal 63% to 128%), respectively. The factor VIII activity was 72% (normal 50% to 200%) and the ristocetin level was 25% (normal > 40%). The diagnosis made was von Willebrand disease. The simple bleeding time of 14.25 minutes (normal 2.3 to 9.5 minutes) significantly improved to 10.0 minutes after infusion of 17 μg of desmopressin (DDAVP). Accordingly, 17 μg of DDAVP was given one hour before the patient's uneventful tonsillectomy. Preoperative blood loss was 2 mL and there was no abnormal postoperative bleeding.
Case 2
A 16-year-old female was scheduled for tonsillectomy to rule out malignancy as the cause for marked tonsillar asymmetry. In the past she had experienced significant menorrhagia.

Although PT was 12.0 seconds (control 11.3 seconds) and APTT was 33 seconds, the preoperative fibrinogen level was only 140 mg/dL (normal 200 to 400 mg/dL). Repeat PT and APTT were again within normal limits, investigation for von Willebrand disease was negative (factor VIII activity 184%, antigen 67%, chromogran 92%, and ristocetin cofactor level 80%), platelet count was 330,000/µL, and thrombin clotting time was 8.3 minutes (control 7.3 minutes).

The hematologist suggested the diagnosis of congenital hypofibrinogenemia which had been exacerbated by menorrhagia. Cryoprecipitate was made available for the procedure but was not required for the uneventful tonsillectomy. Estimated blood loss was 50 mL.

Case 3
A 5-year-old girl presented with tonsillar and adenoideal hypertrophy and recurrent streptococcal tonsillitis. She had no personal or family history to suggest a bleeding diathesis.

Tonsillectomy and adenoidec-tomy were postponed when testing revealed her APTT to be 39.0 seconds. PT was normal at 12.0 seconds (control 11.5 seconds) and the von Willebrand tests were normal (factor VIII activity 95%, antigen 89%, and ristocetin cofactor level 132%), as was the fibrinogen level of 270 mg/dL. The APTT reverted to normal at 33.0 seconds one week later. Mixing studies suggested the presence of a coagulation inhibitor and further evaluation revealed a lupus-like anticoagulant.

Review of the patient's medical records disclosed that antibiotic treatment with penicillin, amoxicillin, and cefadroxil had all been administered within the preceding two months for recurrent tonsillitis. This therapy was associated with the hematologic diagnosis of transient lupus-like anticoagulant.

The tonsillectomy and adenoidectomy was performed without complication. The estimated blood loss was 20 mL.

Case 4
An eight-year-old girl had tonsillar hypertrophy which caused snoring, mouth breathing, and apnea. Preoperative tests disclosed a normal PT of 12.5 seconds (control 11.7 seconds) but elevated APTT of 39 seconds. This value was confirmed by a repeat test five days later and APTT remained elevated for the following two months. There was no personal or family history of any coagulopathy.

The von Willebrand screen was negative, and levels of factors II, IX, XI, and XII were within normal limits. The fibrinogen level was mildly depressed at 170 mg/dL, and the presence of a mild nonspecific inhibitor of coagulation was demonstrated by mixing studies. Further testing identified the inhibitor as a lupus-like anticoagulant. The administration of fresh frozen plasma was unnecessary when the tonsillectomy was performed without incident; estimated blood loss was 5 mL. The patient had experienced an episode of untreated viral pharyngitis a month before the first abnormal APTT test.

Discussion
The coagulation process can be arbitrarily divided into four steps: 1) formation of factor IXa, 2) formation of factor X, 3) formation of thrombin, and 4) formation of fibrin. Factor XI is activated by factor XIIa (Hageman factor) and the contact system complex, a reaction which is not calcium dependent. Factor IX is activated by factor Xla in the presence of calcium. Factor X is activated by factor XIa in the presence of calcium. Factor VII, in the presence of tissue thromboplastin released from damaged vascular tissue, converts factor X to factor Xa in a calcium-dependent reaction; and 2) factor IXa in the presence of factor VIII and calcium may also activate factor X. This complex, factor IXa-Vlll-calcium-X is bound to platelet lipid mycelles.

The conversion of prothrombin to thrombin is achieved by factor Xa in a complex with factor V and calcium. Binding of factor V to the platelet membrane is critical to the reaction. Thrombin, the enzyme derived from prothrombin, cleaves fibrinopeptides AB from the Aα and Bβ chains of the fibrinogen molecule. The remaining major portion of the molecule, fibrin monomer, polymerizes to form fibrin. This polymer is stabilized by factor XIII through conversion of weak hydrogen bonds to stable covalent bonds. The actions of thrombin and platelet factor XIII are calcium dependent.

For practical purposes the coagulation cascade has been divided into two pathways: the intrinsic system representing the pathway used in the absence of tissue extract, and the extrinsic system representing the pathway used in the presence of tissue juices. The concept of two pathways, although not strictly factual, has proved valuable for the study of blood coagulation and the laboratory investigation of clotting disorders. A defect in the intrinsic pathway usually causes a prolongation of APTT and a defect in the extrinsic pathway causes a prolongation of the PT. Prolonged APTT and/or PT is usually caused either by deficiency of one or more coagulation factors or by the presence of a circulating inhibitor of the clotting system. Circulating anticoagulants or inhibitors of clotting factors are substances produced endogenously which affect various in vitro tests of coagulation.

In the present series, four (5%) of 91 consecutive patients undergoing tonsillectomy and/or adenoidectomy by one surgeon were found to have abnormalities of coagulation. One patient had von Willebrand disease, one had congenital hypofibrinogenemia, and two had acquired, transient lupus-like inhibitors. The von Willebrand disease and hypofibrinogenemia have been well discussed in the literature (12) but acquired inhibitors of coagulation may not be familiar to surgeons.

There are two types of acquired inhibitors: blocking inhibitors, and specific factor depressants. Blocking inhibitors are usually proteins which interfere with an entire coagulation reaction but are seldom responsible for clinical bleeding (13). Blocking inhibitors occur in systemic lupus erythematosus, collagen vascular diseases, malignancies, viral infections, or following exposure to certain drugs such as penicillin and/or its derivatives (14-16). Specific factor depressants inactivate a certain clotting factor. Inhibitors against factors V, VIII, IX, XI, and XIII have been described, with factor VIII inhibitors being the most common (13,16-18). Specific factor depressants are usually antibodies and can be responsible for severe bleeding. Specific factor depressants occur in postpartum women, autoimmune disorders such as rheumatoid arthritis, and in dermatologic problems such as psoriasis and pemphigus. Coagulation tests (PT or APTT) may be prolonged either by inhibitors or by factor deficiencies. To differentiate the presence of inhibitors from specific factor deficiencies, mixing studies are performed.
When mixing one-part patient plasma with one-part normal plasma fails to correct the prolonged PT or APTT, the diagnosis of circulating inhibitor is considered to be established. The most common explanation of the development of blocking inhibitors in otherwise healthy children is the exposure to either penicillin and its derivatives or the presence of viral infection (13,19). Our series provides an example of each.

Orris et al (13) investigated 13 children with abnormal APTTs and discovered that all had recently received penicillin or a derivative. The one child who had clinical bleeding developed petechiae and was found to have thrombocytopenia. All 13 patients had transient blocking inhibitors. Ten of these patients subsequently underwent surgical procedures (one for excision of an infected thyroglossal duct cyst and the remainder for tonsillectomy) after the blocking inhibitors disappeared and coagulation studies had returned to normal. No bleeding complications occurred.

Other authors correlated recent viral infections with the development of prolonged APTT. Viruses thought to be involved include Epstein-Barr, adenovirus, cytomegalovirus, and infectious mononucleosis. Development of circulating coagulation inhibitors by other mechanisms may be analogous to the occurrence of idiopathic thrombocytopenic purpura after a viral infection which is thought to cause the development of antibodies to the platelet membrane. In the latter disorder the antibodies usually disappear spontaneously within two months.

The occurrence of acquired inhibitors of coagulation may be more common than is indicated by the medical literature (19). Because of the transient nature of the inhibitors and their usual lack of clinical consequences, their presence is usually discovered only by routine preoperative screening. Patients who have viral infections or who have received penicillin recently are frequently encountered in otolaryngology. Whenever an abnormal coagulation test result is identified, even a minor one, the test must be repeated, and if the abnormality persists hemato logic consultation is necessary. Screening tests do not differentiate a mild from a potentially serious coagulopathy (12). A result just out of the normal range may in fact be a clue to a hematologic disorder that could lead to disastrous complications of a surgical procedure.

In this series of just 94 patients, two potentially serious disorders were discovered. Tonsillectomy and adenoidectomy differs from other surgical procedures in that it leaves an open wound to heal by secondary intention over two weeks. Even normal patients may have significant postoperative bleeding and those with bleeding disorders must be identified. Transient lupus-like anticoagulants, which cause an elevation of the APTT, can be differentiated from serious coagulopathies only by further hematologic testing. If only a transient inhibitor is identified, the surgical procedure can be safely performed when the APTT has returned to normal.

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References