Deep Venous Thrombosis Following Mohs Micrographic Surgery: Case Report

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Deep venous thrombosis (DVT) of the lower extremity is a common adverse outcome following large surgical procedures on the lower extremities, such as orthopedic surgery.[1] It is a much rarer occurrence following Mohs micrographic surgery especially when patients have not immobilized the extremity. Reports of postoperative thrombotic or thromboembolic disease in the dermatologic literature are sparse and involve mainly cases where anticoagulation was discontinued prior to the procedure in individuals with underlying hypercoagulable states.[2,3]

Here we report a case of DVT of the left calf developing within 6 days of Mohs micrographic surgery on the left lower extremity. The case highlights an uncommon, but not impossible, adverse effect of dermatologic surgical procedures of the extremities and underlines the importance of physician vigilance in the postoperative period to recognize and institute treatment for such an adverse outcome.

Case Report

The patient was a 65-year-old Caucasian female with a past medical history of mitral valve prolapse, osteoporosis, and occasional subjective palpitations and a past surgical history of spinal surgery for osteoporotic bone disease. She was referred to us for Mohs micrographic extirpation of a basal cell carcinoma on the left lower anterior pretibial area. Her only medication was risedronate. She denied the use of any aspirin, nonsteroidal drugs, antiplatelet agents, or anticoagulants. The patient underwent the Mohs procedure following antibiotic prophylaxis. For each stage of Mohs surgery, she was placed in a supine recumbent position with the lower extremities resting on the leg support of a standard surgical examination room table. Following each Mohs stage, hemostasis was obtained using electrodesiccation, and a temporary pressure dressing was applied. Between stages the patient waited approximately 30 minutes in a sitting position in our Mohs waiting room. No restriction was placed on ambulation during this period. After three stages of Mohs surgery (approximately 3 hours after starting), the surgical site was free of tumor and was repaired with a standard side-to-side two-layered closure with minimal tension on the wound edges (Figure 1). A pressure bandage including a circumferential leg wrap dressing was applied to the surgical site postoperatively. Wound care instructions required the pressure dressing to be in place for 24 hours, after which time twice daily dressing changes using a nonstick bandage following cleansing of the wound with hydrogen peroxide and application of a bland emollient.

Postoperative instructions also included the avoidance of strenuous cardiovascular exercise, but maintenance of usual other activities, including rapid walking. Leg elevation was also recommended when not walking. Bed rest was not recommended.

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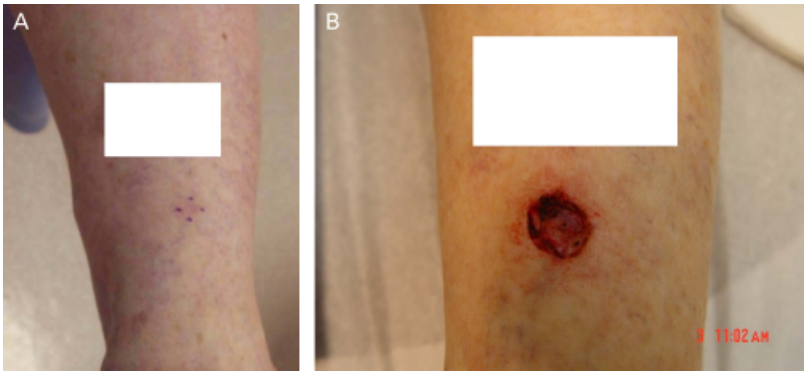

Figure 1. Presurgical site on left calf (A) and Mohs defect (B).

Evaluation of her usual routine revealed that she was a retired female who did not work while standing, nor did much standing while cooking, and she had a preoperative exercise routine involving light cardiovascular activity.

Postoperative course was unremarkable until 5 to 6 days following the surgery when she developed a severely tender linear nodule on the medial lower left leg just proximal to the ankle as well as increased prominence of her already varicosed veins in the left foot. Evaluation by a vascular surgery consultation was obtained immediately and a subsequent lower extremity ultrasonography study revealed DVT of the popliteal vein and its branches (Figure 2), as well as superficial venous thrombosis (SVT) of a segment of the greater saphenous vein.

The patient was offered outpatient injectable heparin followed by oral anticoagulation, but opted for admission to the hospital for intravenous anticoagulation. Her hospital course was uneventful. All symptoms of tenderness in the left calf resolved within 1 week following anticoagulation.

Discussion

Factors that increase the risk of DVT of the lower extremities do so either by increasing the baseline propensity for thrombosis or by precipitating an acute thrombotic event.[4] Thrombosis is promoted by one of three mechanisms. Hypercoagulability can result from genetic factors, such as increased coagulants in prothrombin mutation G20210A or decreased anticoagulants in antithrombin deficiency, protein C or S deficiency, and Factor V Leiden; or acquired factors such as malignancies, hyperhomocyteinemia, hormone replacement or oral contraceptive use, pregnancy, nephrotic syndrome, antiphospholipid syndrome, or increased levels of clotting factors. Direct vessel injury resulting from trauma, surgery, intravascular catheters, endothelial injury for chemotherapeutic agents, vasculitis, antiphospholipid syndrome and hyperhomocysteinemia can also be responsible for thrombosis. Blood pooling and stasis secondary to obesity, pregnancy, sedentarianism, and advanced age that may be

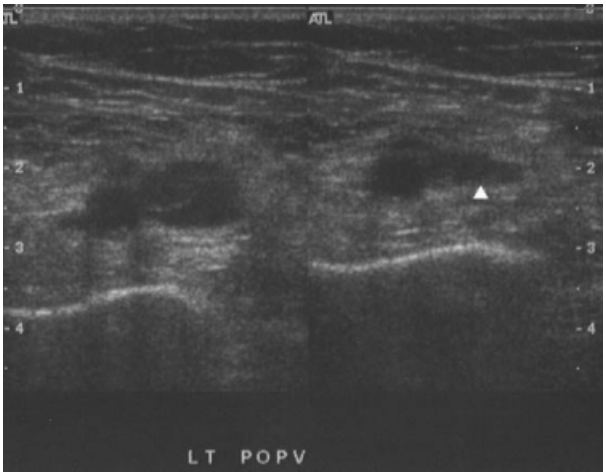

Figure 2. Left politeal vein with (right) and without (left) compression showing lack of compression (arrowhead) on gray-scale Doppler ultrasound consistent with DVT in the popliteal vein.

combined with hospitalization and a bed-ridden state, limb paralysis, right heart failure, and venous compression may all contribute to thrombosis. Varicosed veins and smoking may also play roles in the development of venous clots that can result in DVTs.

Surprisingly, only a handful of cases have been reported in the literature where DVT or thromboembolic disease was related to Mohs micrographic surgery. It is possible that some combination of underreporting and/or underdiagnosis may be responsible for these low numbers of reported cases. Schanbacher and Bennett[2] reported two cases of thromboembolic strokes closely following Mohs surgery, but both cases occurred in patients who had discontinued anticoagulant therapy for underlying high-risk states: a mechanical mitral valve in one case and coronary artery disease–related atrial fibrillation in the second. The first patient had also discontinued aspirin therapy. Both patients were restarted on warfarin 1 day following Mohs surgery after being off the anticoagulant for 1 week prior. In these two cases the authors suggest that the underlying predisposing conditions, superimposed on a rebound hypercoagulable state upon cessation and then restarting warfarin, was responsible for the adverse outcomes.

Similarly Alam and Goldberg[3] described two patients with thrombotic complications following the discontinuation of anticoagulation prior to Mohs surgery. The first patient had a history of a minor stroke and coronary artery disease and myocardial infarction in the past and was discontinued off aspirin and ticlopidine 1 week prior to Mohs surgery. Thirty-six hours following Mohs surgery for perivulvar Bowen's disease, the patient experienced symptoms of shortness of breath and had radiographic evidence of pulmonary embolism. The second patient had a history of coronary artery disease and coronary bypass surgery and atrial fibrillation and a remote breast cancer history. Significantly, this patient was also on hormone replacement therapy and discontinued off clopidogrel and ardeparin 1 week prior to Mohs surgery. Three days later the patient developed chest discomfort and was found to have a clotted prosthetic aortic valve.

Interestingly, in contrast to our case, none of the reported cases involved Mohs surgery on the lower extremity or even of any extremity for that matter. Also, none of the reported cases had clinical or radiographic evidence of concomitant SVT. Remarkably, all the reported cases also had antithrombotic therapy withheld and/or restarted prior to developing thrombotic complications. In contrast, our patient was on no prior antithrombotic therapy, nor was she begun on any medications that may have predisposed her to a prothrombotic state.

Our patient denied a history of smoking, was on no hormonal therapies, and had no known underlying hypercoagulable state (although no work-up was done). One possible risk factor our patient may have had, though she denied this retrospectively, was inactivity following left lower extremity surgery. Although we routinely advise leg elevation following lower extremity surgery, we stress that patients not become inactive, but limit their activity to less strenuous ones. She also had extensive varicosed veins and was of advanced age that may place her at increased risk for development of DVT.

Both on clinical and on radiographic grounds, our patient presented with a SVT and DVT in the linear area of swelling near the surgical site as described above. The relationship between SVTs and DVTs have recently been reviewed.[5] The two disorders share similar predisposing factors. In the literature, the coincidence of DVT in patients with SVT varies widely from 2.6% to 65%, with 50% to 75% of the DVTs being contiguous with the SVT,[5] as in our patient. The most common location for SVT is the lower extremity with 60% to 80% occurring in the greater saphenous vein, again as in our case.[6,7] Varicose veins are the most important risk factor for SVT.[5] SVT is a recognized and documented risk factor for both DVT and venous thromboembolism and for recurrent venous thromboembolism after a

first episode of DVT or pulmonary embolism.[8–10] In the case of our patient, it is impossible to unequivocally state that the relationship between her Mohs procedure and the subsequent development of DVT was definitely not coincidental, but the overwhelming likelihood is that some relationship to the surgery exists. Our patient had at least two risk factors that increased her risk of SVT development: extensive varicose veins and a lower extremity surgical procedure. Having formed a SVT, her indirect risk of DVT development, by extension, was also likely increased.

Insufficient evidence-based data exist in the literature to assess whether perioperative anticoagulation in Mohs patients with extensive varicose venous disease is warranted. In the absence of such data, our clinical experience would dictate that the risks of anticoagulation do not likely outweigh the potential benefits, given the rarity of DVT cases both in our hands and in the literature following Mohs surgery on the extremities.

The current case highlights the fact that a high index of suspicion is paramount in the postsurgical follow-up period after lower extremity cutaneous surgery. Though rare, unexplained new complaints of increasing lower extremity pain following lower extremity surgery in the limb in question require urgent diagnostic evaluation intended to rule out DVT and avoid an impending pulmonary embolism.

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