

Reoperation of Lower Extremity Microsurgical Reconstruction When Facing Postsplenectomy Thrombocytosis

Ping Song, MD
Nirav Patel, MD, JD
Lee L.Q. Pu, MD, PhD, FACS

Summary: Patients who sustain high-energy polytrauma are a unique population in that their mechanism of injury may induce multiorgan damage requiring immediate interventions such as splenectomy and bony fixation for mangled extremities. This results in the intersection of certain conditions, such as postsplenectomy thrombocytosis with the need for soft tissue reconstruction, often with free tissue transfer, for limb salvage after severe trauma to the lower extremity. However, there are no treatment guidelines in the management of postsplenectomy thrombocytosis in the setting of a lower extremity free flap reconstruction. We present a patient who had initial free tissue transfer to the lower extremity complicated by delayed microvascular thrombosis at postoperative day 4, during which time his platelet count exceeded more than 1,000,000/mm³. However, a successful second lower extremity free tissue transfer was achieved after platelet-reductive treatment with platelet apheresis during the perioperative period. Our patient went on to heal from his second free flap without further complications. Thus, thrombocytosis in the setting of free tissue transfer requires perioperative intervention to correct this hematologic condition. An innovative utilization of platelet apheresis may ensure the success of free tissue transfer by addressing the thrombocytosis in microsurgical patients after splenectomy. (*Plast Reconstr Surg Glob Open* 2019;7:e2492; doi: [10.1097/GOX.0000000000002492](https://doi.org/10.1097/GOX.0000000000002492); Published online 8 November 2019.)

INTRODUCTION

To optimize the outcome of microsurgical lower extremity reconstruction, the plastic surgeon must strive to improve the patient's inherent ability to successfully undergo free tissue transfer or decrease the risk factors in such settings. Extreme levels of thrombocytosis may possess higher risk in lower extremity reconstruction, especially if the patient demonstrates preoperative thrombotic events such as the development of deep vein thrombosis or pulmonary emboli.

In this report, we highlight our indications for consideration of apheresis and offer our novel algorithm for the management of thrombocytosis in the setting of failure from previous free tissue transfer to the lower extremity. In addition, we confirm the role of platelet apheresis as a

safe and efficacious means of treating thrombocytosis in the microsurgical patient.

PATIENT REPORT AND OUR MANAGEMENT STRATEGY

The patient was a 43-year-old White man who presented to our hospital with a mangled left lower extremity after a motorcycle collision. Significant medical history included myocardial infarct within past 6 months. The patient was hemodynamically unstable and underwent exploratory laparotomy with splenectomy for splenic laceration. Afterward, the patient's multiple orthopedic injuries were stabilized. During his initial postsplenectomy course, the patient also developed acute segmental pulmonary emboli of the right lung.

Our reconstructive team was consulted for soft tissue reconstruction of a near circumferential left Gustilo IIC middle and distal third leg wound with transection of the posterior tibial artery and vein in the distal leg. His peroneal artery was also not patent (Fig. 1). On hospital day 11, a medial gastrocnemius flap was performed to cover the middle third defect and both transected posterior tibial artery and vein were explored and found useable for future microvascular anastomoses. Two days after, a contralateral free latissimus dorsi muscle flap was performed

From the Division of Plastic Surgery, Department of Surgery, University of California Davis Medical Center, Sacramento, Calif. Received for publication April 10, 2019; accepted August 26, 2019.

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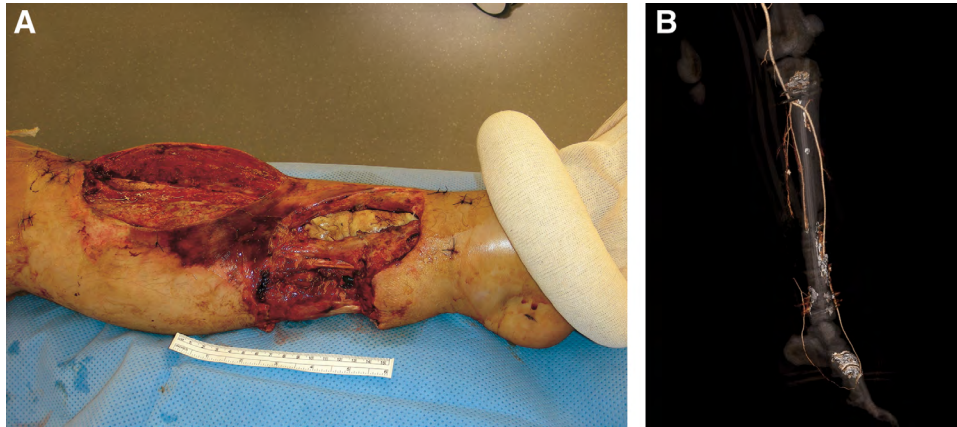


Fig. 1. A, Presentation of the left lower extremity trauma at the time of initial consultation. B, Preoperative CT angiogram shows the transected posterior tibial artery. In addition, the peroneal artery is also transected. CT, computed tomography.

to cover the distal third wound, using both posterior tibial artery and vein stumps as recipient vessels. The free latissimus flap surgery was uneventful, and the patient was monitored in the surgical intensive care unit on oral aspirin and intravenous dextran. On postoperative day 4, the patient developed an infected hematoma of the left knee with systemic signs of fever and hypotension. Clinical exam was suspicious for anastomotic thrombosis of the latissimus flap, and the patient was taken back to the operating room. Intraoperatively, it was noted that both arterial and venous anastomoses thrombosed. Thromboembolectomy was performed with a 2-mm Fogarty catheter and after

redo arterial and venous microvascular anastomoses, both pedicle artery and vein were patent. However, in less than 1 hour while he was still in the recovery room, both micro-anastomoses were thrombosed again. At this point, the flap became unsalvageable. Temporary vacuum-assisted closure device was placed in the operating room after debridement of the necrotic flap.

In light of the patient's failure of his first free tissue transfer, we noted his platelet count to have a coincidental peak that exceeded more than 1,000,000 platelets/ μL at the time of his initial free tissue transfer. After appropriate consultation, the apheresis service was involved to

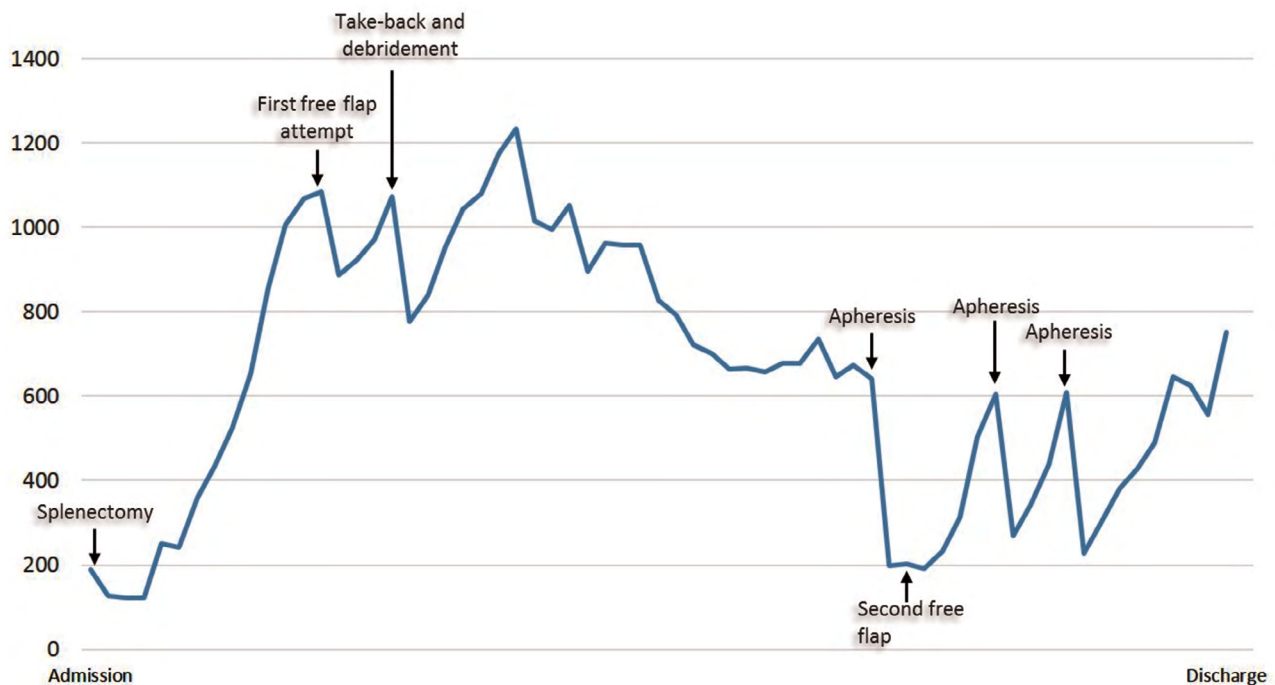


Fig. 2. Platelet count throughout the hospital admission. Significant events are labeled. Note the reactive thrombocytosis and the peak in platelet count during initial reconstruction.



Fig. 3. Results at 14 months after his second free flap reconstruction.

aid our perioperative management of thrombocytosis. Thus, the decision was made to treat the thrombocytosis with platelet apheresis before undergoing a second free flap. After the first apheresis session, platelet counts reached a nadir of $197\text{K}/\text{mm}^3$, and the patient underwent a free rectus abdominis muscle flap and skin grafting to his left lower extremity, again using the posterior tibial artery stump and vein stump as recipient vessels. However, platelet counts began to rise again postoperatively and 2 additional apheresis sessions were performed on postoperative days 6 and 9 after the second flap. **Figure 2** shows the platelet count over the course of the hospital admission with major events highlighted. The second free flap encountered no additional complications except an additional skin grafting procedure. He was discharged on postoperative day 22 from his second free tissue transfer and completely healed his left lower extremity wound during a 14-month follow-up (**Fig. 3**). He also subsequently healed his fracture site after bone grafting during additional follow-up by our orthopedic trauma team (**Fig. 4**).

DISCUSSION

The presence of thrombocytosis may occur in several settings including reactive thrombocytosis (ie, postsplenectomy) after high-energy trauma. Therapeutic platelet apheresis has been shown in the literature to be a safe and reliable tool used in both the acute setting for cerebral vascular emergencies associated with myeloproliferative disease states and maintenance therapy for patients with reactive or primary inherited thrombocytosis.¹¹⁻¹³ Our patient had over 1 million platelets/ μL during his first flap failure. Even though the literature has not directly identified thrombocytosis as direct causation to free flap failure, the American Society for Apheresis considers values over 1 million platelets/ μL as high risk for serious thrombotic or hemorrhagic events.

Our report aims to demonstrate a treatment strategy to counter thrombocytosis-induced flap failure in lower extremity microsurgical reconstruction. This patient was

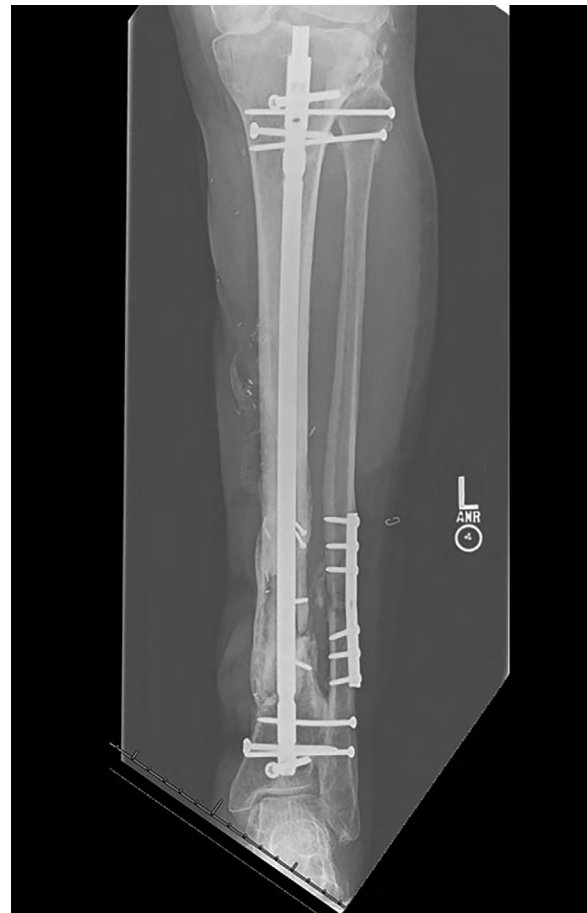


Fig. 4. Follow-up x-ray shows appropriate healing of the fracture site 30 months after bone grafting.

the first at our institution who developed thrombocytosis-associated flap failure in the setting of routine free flap protocol. In addition, the patient developed preoperative pulmonary emboli which suggested he was at higher risk from thrombocytosis. After consultation with our platelet apheresis team, we proceeded with 1 preoperative platelet apheresis session, and 2 additional postoperative sessions to maintain platelet counts within normal physiologic range until postoperative day.¹⁰

We posit that patients with thrombocytosis, who demonstrate 1 or more signs of spontaneous thrombotic events before microsurgical reconstruction, should be considered at high risk and should undergo the appropriate cyoreductive therapy for thrombocytosis. Platelet apheresis can be an effective option and should be considered when a microsurgical patient presents with thrombocytosis especially after splenectomy.¹⁴ We believe extreme thrombocytosis indeed carries a higher risk of flap-related complications. Spontaneous preoperative thrombotic events such as deep vein thrombosis and pulmonary emboli, in the setting of thrombocytosis, serve to forewarn the microsurgeon to the importance of cyoreductive therapy before undertaking a free tissue transfer.

CONCLUSIONS

We highlight platelet apheresis as an innovative and effective treatment for significant traumatic postsplenectomy thrombocytosis in reoperation of microsurgical lower extremity reconstruction. Our success for reoperative microsurgical reconstruction in this patient again demonstrates the value of platelet apheresis and should be included in a microsurgeon's armamentarium when managing perioperative thrombocytosis.

Lee L.Q. Pu, MD, PhD, FACS
 Division of Plastic Surgery
 University of California, Davis
 2221 Stockton Boulevard, Suite 2123
 Sacramento, CA 95817
 E-mail: llpu@ucdavis.edu

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