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Case Report

Microsurgery in the Sickle Cell Trait Population: Is It Actually Safe?

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ABSTRACT

Although sickle cell disease has long been viewed as a contraindication to free flap transfer, little data exist evaluating complications of microsurgical procedures in the sickle cell trait patient. Reported is the case of a 55-year-old woman with sickle cell trait who underwent a deep inferior epigastric perforator (DIEP) microvascular free flap following mastectomy. The flap developed signs of venous congestion on post-operative day two but was found to have patent arterial and venous anastomoses upon exploration in the operating room. On near-infrared indocyanine green angiography, poor vascular flow was noted despite patent anastomoses and strong cutaneous arterial Doppler signals. Intrinsic microvascular compromise or sickling remains a risk in the sickle cell trait population as it does for the sickle cell disease population. Just like in sickle cell disease patients, special care should be taken to optimize anticoagulation and minimize ischemia-induced sickling for patients with sickle cell trait undergoing microsurgery.

Introduction

Sickle cell disease has long been viewed as a relative contraindication to free tissue transfer due to the higher reported rates of anastomotic thrombosis and partial or total flap failure. The mechanism is believed to be secondary to sickling in the flap microvasculature during "flap off" time where there is flap hypoxia, ischemia, and vascular stasis [1]. Under hypoxic conditions, the abnormal sickle hemoglobin HbS polymerizes, elongating the erythrocyte into a "sickle cell". The misshapen erythrocytes increase the viscosity of blood and become lodged at narrow capillary bridging points resulting in ischemic events [2].

Relative to sickle cell disease, sickle cell trait is often viewed favorably in the context of microsurgery despite a paucity of data [1]. It is theorized that because patients with sickle cell trait are relatively asymptomatic and have reduced red blood cells with HbS compared to sickle cell disease patient, they are unlikely to experience flap failure. We present a case of a patient with sickle cell trait who underwent a free flap reconstruction and post-operatively developed clinical flap venous congestion and poor uptake of indocyanine green despite patent anastomoses and a strong cutaneous arterial Doppler signal.

Case Report

A 55-year-old woman with known sickle cell trait (Hgb A-S-G Philadelphia complex heterozygote with 47% of hemoglobin containing either of the two mutations at initial presentation) was diagnosed with a left-sided breast cancer and underwent a left-sided mastectomy, sentinel lymph node biopsy, and deep inferior epigastric perforator (DIEP) flap reconstruction. The patient was evaluated by hematology pre-operatively and transfused two units of packed red blood cells to lower her abnormal hemoglobin percentage. Her intra-operative course was uneventful, and there was a robust arterial and venous doppler signal after successful anastomosis. Total ischemia time was 47 minutes. Because of her high percentage of abnormal hemoglobin, extra precautions were taken. A bolus of 3000 units of heparin was given prior to severing the vessels on the flap, and a heparin drip at 500 units/hour was initiated intra-operatively and maintained on this dose post-operatively. On post-operative day two, the DIEP flap had clinical signs of venous congestion including global flap swelling, brisk capillary refill on the skin paddle, loss of a cutaneous venous Doppler signal, and brisk, dark blood on pinprick. The patient was taken to the operating room for exploration. The arterial and venous anastomoses were notably patent. There was no appreciable kink in the perforators or main pedicle and no evidence of a missed superficial dominant venous circulation.

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Indocyanine green was injected intravascularly to evaluate flap circulation. Despite the presence of patent arterial and venous anastomoses as well as a cutaneous arterial Doppler signal, there was poor uptake of indocyanine green within the flap (Figure 1), suggesting an intrinsic physiological issue within the flap microvasculature.

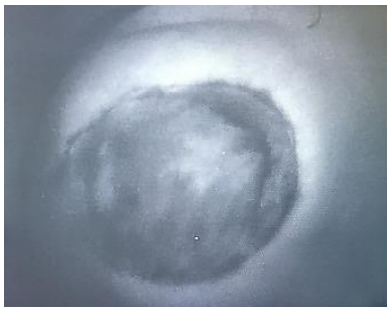

Figure 1: Intra-operative near-infrared angiography demonstrating poor uptake of indocyanine green despite patent arterial and venous anastomoses.

The patient then received a heparin bolus of 3000 units followed by an increase of the heparin drip to 1000 units/hour. On repeat pinprick/scratch test, brighter red bleeding was observed suggesting an improving flap physiology. Given the patent anastomoses and encouraging clinical signs, the decision was made to medically optimize the patient. The patient received supplemental oxygen via nasal cannula, continued aspirin, heparin drip, verapamil, phosphodiesterase inhibitors, and nitroglycerin paste. The patient was maintained on subcutaneous therapeutic enoxaparin injections and aspirin for 1 month after discharge. The flap remained viable throughout the post-operative period.

Discussion

Sickle cell disease is an autosomal recessive hemoglobinopathy caused by the presence of the abnormal beta globin gene hemoglobin S (HbS). In sickle cell disease, there are two abnormal beta globin genes for HbS and 100% of the hemoglobin are HbS. However, in sickle cell trait, there is one HbS gene, and up to 40% of the hemoglobin are HbS [2]. The incidence of sickle cell trait is estimated to be greater than 1 in 100 and translates to almost 60,000 new cases every year in the United States [3]. Patients with sickle cell disease have reported to experience an increased risk of free flap loss than the general population, comparable to patients with established risk factors such as acute burn or diabetes [1]. However, there is a paucity of data regarding the outcomes of flap surgery in sickle cell trait patients. Spear *et al.* (2003) reported a case of skin flap necrosis in a patient with sickle cell trait receiving bilateral reduction mammaplasty utilizing superomedial pedicles [4]. In the absence of intraoperative complications, the authors suggested sickle cell trait as a possible risk factor for the flap failure [4]. McAnneny *et al.* (2012) reported a sickle cell trait patient who developed clinical signs of flap venous congestion after breast reconstruction despite having patent anastomoses [5]. Pathological analysis of the flap post failure identified sickled cells within the microvasculature [5].

Significant heterogeneity exists in the proportion of abnormal hemoglobin in the sickle cell trait population. Patients with higher proportions of abnormal hemoglobin may have microsurgical risk profiles that approach the sickle cell disease population. In sickle cell trait patients, prolonged ischemia times, hypoxia, and hypothermia of the free flap can precipitate red blood cell sickling, which can lead to microvascular thrombosis and subsequent flap failure [1, 5]. Microsurgery in sickle cell trait patients with hemoglobin S levels greater than 30% should be approached with similar caution as patients with sickle cell disease [1, 6]. Pre-operative blood transfusions should be considered in these patients in order to reduce microvascular complications [1]. Minimizing ischemia times and optimizing anticoagulation should also be considered in sickle cell trait patients undergoing microsurgery [7, 8]. In this case, increasing the heparin dose and providing supplemental oxygen to increase the partial pressure of oxygen may have improved flap physiology to salvage the reconstruction.

Conclusion

We report a case of a 55-year-old woman with sickle cell trait who developed signs of venous congestion following a DIEP flap, most likely secondary to sickling. Sickle cell trait patients with high proportions of hemoglobin S may experience increased risk of flap failure similar to patients with sickle cell disease. Special care should be taken to optimize anticoagulation and minimize ischemia-induced sickling for patients with sickle cell trait undergoing microsurgery.

Conflicts of Interest

None.

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