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Case Report

Monitoring free flap venous congestion using continuous tissue glucose monitoring: A case report

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ABSTRACT

Blood glucose levels (BGLs) are a good indicator of postoperative venous congestion caused by a thrombus at the anastomotic site of a free flap. Tissue glucose levels (TGLs) are believed to be superior to BGLs for two reasons: TGLs are thought to represent a tissue's congestive status more directly than BGLs and are able to be measured by a continuous tissue glucose monitoring device (CTGMD), whereas BGLs must be measured manually by sampling the flap, hindering the patient's sleep and increasing the nurse's workload. A case is described in which a postoperative thrombus developed in a free flap vein three times. TGL in the flap was monitored by a CTGMD (Free Style Libre®, Abbott, U.S.A.), and BGL was monitored in parallel by conventional sampling of the flap. When venous congestion developed at the anastomotic site, TGLs decreased faster than BGLs; after the congestion was ameliorated by exsanguination. BGLs increased faster than TGLs, indicating that TGLs are a better indicator of venous thrombosis at the anastomotic site than BGLs.

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Introduction

Blood glucose monitoring after microsurgical anastomosis of a flap is a simple and reliable method to detect thrombosis at the anastomotic site^{1–3}. We revised this method using a continuous tissue glucose monitoring device (CTGMD; Libre®, Abbott, U.S.A.), which only requires tissue fluids for measurements of tissue glucose levels (TGLs). During such measurements, the flap would not need to be sampled for conventional measurements of blood glucose level (BGL). To evaluate this new monitoring technique, the device was used to measure TGLs of free flaps postoperatively. Both continuous tissue glucose monitoring and conventional blood glucose monitoring were performed, and the data were compared.

Case Report

A 29-year-old female with a chronic ulcer on her right lateral malleolus, which was caused by postoperative infection after orthopaedic surgery, was referred to our hospital. Debridement of the ulcer and coverage with a superficial inferior epigastric artery perforator flap was performed with an end-to-end arterial anastomosis to the dorsalis pedis artery and two end-to-end anastomoses to the subcutaneous and dorsalis pedis veins. A CTGMD was applied to the flap after surgery, and TGLs were continuously monitored for six days (Figure 1). The device was designed to automatically obtain TGL every 15 min and work for two weeks without replacement. Conventional blood glucose monitoring of the flap was also performed by measuring BGL of the flap via pin pricks every three hours. When the flap became pale or BGL dropped to <40 mg/dL, the cut-off value for flap congestion, a doctor would be called for an evaluation.

Venous thrombosis occurred at the anastomotic site three times postoperatively. By 14.5 h after surgery, TGL began to decrease; two hours later, the CTGMD indicated a "low" level, meaning below 40 mg/dL, whereas BGL was 75 mg/dL. Because the flap had apparently developed congestion, the patient was taken to the examination room; the comitant vein of the flap was found to be thrombosed and was subsequently exsanguinated. After approximately 45 minutes, BGL increased to 124 mg/dL, whereas TGL was 41 mg/dL (Figure 2). A second surgery was performed, and the comitant vein was re-anastomosed to the greater saphenous vein. Subsequently, BGLs stayed above 80 mg/dL, and TGLs improved but stayed relatively low. More than 9 h later, TGL dropped, and BGL decreased to 57 mg/dL. The flap again developed congestion; exsanguination was again performed 3 min later. Within 20 min, TGL increased to 52 mg/dL; BGL increased to 106 mg/dL in less than 2 h (Figure 2). After the second surgery, TGL stayed above 50 mg/dL for an hour but eventually decreased to 49 mg/dL 2 h later, whereas BGL stayed high. In >2 h, BGL dropped to 59 mg/dL, and the flap again developed congestion. The anastomotic site was found to be thrombosed and was exsanguinated within the hour. Within minutes, TGL was 43 mg/dL and BGL increased to 134 mg/dL (Figure 2). After the third surgery, both BGL and TGL increased to normal levels. The postoperative course was uneventful, and both TGL and BGL stabilized above 80 mg/dL; however, TGL began to decrease on postoperative day 5, while BGL stayed above 100 mg/dL (Figure 3). The flap colour was normal and there were no signs of congestion. The flap was found to be compressed by a cushion during sleep. The cushion was removed on postoperative day 6, and TGL increased to 71 mg/dL within approximately 20 min (Figure 3).

Discussion

Free flaps have a risk of thrombosis at the anastomotic site postoperatively. Therefore, postoperative monitoring of the flap is vital. Hara et al.² reported that BGL was an indicator of flap venous congestion. We propose that TGL is superior to BGL as an indicator of flap hypoxia caused by thrombosis at the anastomotic site. As each event of venous congestion occurred in our patient, TGL decreased earlier and faster than BGL. However, after exsanguination, BGL increased earlier and faster than TGL. This phenomenon indicates that TGL represents tissue status more directly than BGL. Our hypothesis is as follows: When venous congestion occurs, the tissue becomes hypoxic, and anaerobic glycolysis is accelerated, resulting in tissue glucose depletion. As the venous network functions as a glucose reservoir, blood glucose depletion is delayed, resulting in a slower decrease in BGL than in TGL. However, Download English Version:

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