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RESEARCH ARTICLE

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Heparin-induced thrombocytopenia and thrombosis in primary lymphedema patients who underwent vascularized lymph node transplantations

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Abstract

Background: Heparin-induced thrombocytopenia and thrombosis (HITT) may result in microsurgical flap failure. This study investigated the outcomes of HITT in primary lymphedema patients who underwent vascularized lymph node transplantations (VLNT).

Methods: Between 2012 and 2019, primary lymphedema patients who underwent VLNTs were retrospectively included. The 4Ts score was used to categorize patients into HITT (scores of 5–7) and non-HITT (score < 5) groups. Outcome evaluations included the re-exploration rate, success rate, circumferential differences, cellulitis episodes, and Lymphedema Specific Quality of Life Questionnaire (LYMQoL) scores.

Results: Twenty-six and 15 patients with 31 and 16 VLNTs were included in the HITT and non-HITT groups, respectively. The HITT group had significantly greater first, second and third re-exploration rates of 38.7% (12/31), 25.7% (8/31), and 6.5% (2/31) than the non-HITT group (6.3%, 0%, and 0%, all $p < 0.01$), respectively. The platelet counts significantly decreased by 21.0% in the HITT group compared with the non-HITT group (14%) on postoperative Day one ($p < 0.01$) with a cutoff value of 17% and AUC = 0.88.

Conclusions: HITT may cause a high re-exploration rate of VLNTs in primary lymphedema patients. The 17% reduction in platelets on postoperative day one was an early sign for detecting HITT.

KEYWORDS

4Ts score, heparin-induced thrombocytopenia and thrombosis, platelet count, primary lymphedema

1 | INTRODUCTION

The etiology of primary lymphedema involves defects or anomalies of regional lymph nodes and lymphatic vessels.[1] Primary lymphedema is relatively uncommon compared with secondary lymphedema, involving approximately 1–3 in 100 000 individuals. Females are affected approximately 3.5-fold more often than males.[2,3] Primary lymphedema is related to gene mutations, especially genes correlated with the regulation of vascular endothelial growth factor C.[4,5] At least 20 genes have been recognized to be associated with primary lymphedema, and approximately 30% of patients have these genes.[4] Primary lymphedema individuals, therefore, often have comorbidities with vascular diseases.[4,5] Furthermore, immune imbalance, such as hyperactivation of CD4 T cells, has been proven to induce lymphedema in primary lymphedema patients.[6,7]

Primary lymphedema is classified into three types: congenital lymphedema, lymphedema praecox, and lymphedema tarda.[2] While usually symptomatic in teens or adulthood, the long-standing history