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# Current Insights into Post-Traumatic Lymphedema

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## Abstract

Post-traumatic lymphedema (PTL) is a chronic and often under-recognized sequela of soft tissue trauma, leading to persistent swelling, functional impairment, and increased risk of infection. While lymphedema is traditionally associated with oncologic interventions, growing evidence highlights the significant burden of PTL in trauma patients. This review provides a comprehensive analysis of the current understanding of PTL, including epidemiology, risk factors, pathophysiology, diagnostic modalities, and treatment strategies. PTL often occurs after high-impact musculoskeletal injuries (such as open fractures with significant soft tissue loss) or burns (especially if deep or circumferential). This risk is increased if injury occurs at critical areas of increased lymphatic density (such as anteromedial leg, medial knee, medial thigh, medial elbow, or medial arm). Advances in imaging techniques, including indocyanine green lymphography and magnetic resonance lymphangiography, have improved early detection and classification of PTL. Management approaches range from conservative therapies, such as complete decongestive therapy (CDT), to surgical interventions, including lymphaticovenous anastomosis (LVA), vascularized lymph node transfer (VLNT), and vascularized lymph vessel transfer (VLVT)/lymph-interpositional-flap transfer (LIFT). We report on our experience with two patients. At our center, we diagnose and stage PTL with ICG lymphography and trial CDT for 6 months. If there is no significant improvement, we recommend LVA. If there is insufficient improvement after 12 months, we recommend LIFT/repeat LVA/VLNT. We also treat open fractures with significant soft tissue defects with LIFT, as prophylaxis against PTL. PTL remains an underdiagnosed condition, necessitating increased awareness and intervention to prevent long-term disability.

**Keywords:** lymphedema; trauma; indocyanine green lymphography; lymphaticovenous anastomosis; vascularized lymph node transfer; vascularized lymph vessel transfer; lymph-interpositional-flap transfer

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## 1. Introduction

Lymphedema is a chronic and debilitating condition that affects 250 million people worldwide [1]. It is characterized by persistent swelling from lymphatic dysfunction and often results in functional impairments and recurrent infections, leading to significant morbidity and mortality [2,3]. Lymphedema is caused by the accumulation of proteinaceous fluid within the interstitial compartment from abnormalities of the lymphatic transport system [4]. In developed countries, the most common etiology for lymphedema is cancer and its treatment, most notably surgical lymph node dissection and radiotherapy [5,6].

However, a notable, yet under-recognized, cause of secondary lymphedema is trauma. Despite the rapid advancements made over recent years in treating lymphedema, PTL remains an underdiagnosed condition, with sparse publications in the literature, most of which are case reports. This review aims to bridge existing gaps in knowledge, propose standardized diagnostic criteria, and highlight emerging therapeutic strategies for optimizing patient outcomes in trauma-related lymphatic injury.

## 2. Results

### *2.1. Epidemiology and Etiology*

Post-traumatic lymphedema is defined as lymphedema that develops after trauma and can occur after open or closed injuries. The exact incidence of PTL is unknown, with no large sample size study describing its incidence. The traumatic causes of PTL are wide-ranging but most commonly include musculoskeletal injuries or burns to the limbs. Most reports of PTL describe fractures as inciting events and can occur with or without surgical intervention for the fractures. Most of these fractures occur in the lower limbs and are often high-energy fractures, such as open tibia and/or fibular fractures [7]. However, even minor musculoskeletal injuries, such as contusions, have been reported to cause PTL, albeit transient [8]. Lymphedema of the hand has also been reported after distal radius fractures [9,10], with or without surgical intervention for the fracture. Facial [11] and lower eyelid lymphedema [12] from facial fractures have also been reported. Post-traumatic abdominal lymphedema presenting as chylous ascites or chyloretroperitoneum has also been reported. However, these are rare and often occur with other abdominal injuries, as the impact required to injure the cisterna chyli or thoracic duct is high [13]. With regard to PTL from burns, Hettrick et al. described a prevalence of 1.0% and identified circumferential extremity burns and fascial excision as risk factors for developing PTL [14]. Notably, these publications described are either case series with small sample sizes or case reports; there are no studies with large sample sizes.

Importantly, the specific site of injury in the limb matters. Critical areas with increased lymphatic density have been identified, and injuries to these areas are more prone to developing lymphedema [15], even in small but focal areas [16]. These locations include the anteromedial leg [8,17], medial knee, medial thigh, medial elbow, and medial arm [18] and correspond to the lymphosomes that drain into the inguinal lymph nodes (for the lower limb) and axillary lymph nodes (for the upper limb), namely the inferior inguinal lymphosome and lateral axillary lymphosome, respectively [19].

Post-traumatic lymphedema is a distinct subset of post-traumatic edema. There are multiple causes of post-traumatic edema, ranging from being part of the normal healing process due to an inflammatory response [14], to post-traumatic osteoarthritis when joints are involved, to venous congestion from deep vein thrombosis [20] or post-traumatic venous insufficiency, to PTL. However, PTL is distinct from other causes of post-traumatic edema as the etiology is disruption of lymphatic channels (as evidenced by dermal backflow seen on indocyanine green (ICG) lymphography, which will not be seen in other causes of post-traumatic edema [21]). In other forms of post-traumatic edema, there is compensatory increased lymphatic flow, but no damaged lymphatic channels [22].

### *2.2. Proposed Pathophysiology*

The exact mechanism of PTL is unknown, but some mechanisms have been proposed.

The lymphatic system can be divided into superficial (subdermal) and deep (subfascial), but they are not independent systems, as pathology in one system can affect the other. However, the superficial (subdermal) system is more efficient than the deep (subfascial) [23], and it exists in a complex network that is abundant in the reticular dermis

and subcutaneous fat [14]. This superficial location makes it susceptible to trauma, even if superficial, including burns (especially those of deep partial thickness and deeper). One proposed mechanism is that damaged lymphatics from the trauma fail to repair via lymphangiogenesis, and this risk is increased with complex trauma involving vascular and/or extensive soft tissue injury [24]. Although lymphatic capillaries have a strong ability to repair healthy tissue, this ability is greatly compromised in damaged tissue [25]. This may be related to the size of the lymphatic gap that results from the injury. A gap that is too wide or filled with too much scar tissue may impair the body's ability to achieve reconstitution of the lymphatic pathway. The lymphatics can be damaged directly, for example, in open penetrating injuries, or via shearing in blunt injuries, as seen in Morel-Lavallée lesions [26]. Another proposed mechanism is that the scar tissue that develops during wound healing obstructs lymphatic flow, especially that of the superficial system, and leads to lymphedema [27]. In these instances, PTL often persists beyond 3 months and can be deemed pathological.

### *2.3. Diagnosis and Classification*

Lymphedema can be diagnosed via lymphoscintigraphy, magnetic resonance lymphangiography, or indocyanine green (ICG) lymphography. Lymphoscintigraphy remains the gold standard for diagnosing lymphedema, but ICG lymphography has been gaining popularity and adoption in recent years, especially for diagnosing PTL. This is because it can diagnose and stage the disease, allow real-time lymphatic mapping without radiation exposure, and can be repeated without adverse effects [28]. In 2015, Ito et al. first described the use of ICG lymphography in diagnosing PTL [21]. ICG lymphography has since been the primary diagnostic modality in all PTL studies [16].

In terms of staging, lymphedema is classically staged using the International Society of Lymphology (ISL) classification: stage 0 refers to subclinical lymphoedema, stage I refers to lymphoedema that resolves with limb elevation, stage II refers to lymphoedema with tissue fibrosis that does not resolve with limb elevation, and stage III refers to elephantiasis with fibroadipose deposition and gross skin changes [29] such as hyperkeratotic, verrucous, and papillomatous skin lesions.

In ICG lymphography, lymphedema severity is staged based on the dermal backflow pattern seen on ICG. Dermal backflow is pathognomonic of lymphedema. ICG stage I refers to splash pattern seen on ICG and corresponds to subclinical lymphedema; stage II refers to mild stardust pattern and corresponds to early lymphedema; stage III refers to moderate stardust pattern and corresponds to progressed lymphedema; stage IV refers to severe stardust pattern and corresponds to progressed lymphedema; and stage V refers to diffuse dermal backflow and similarly corresponds to progressed lymphedema [28]. Linear pattern, without any dermal backflow pattern, denotes normal lymphatic flow [30].

### *2.4. Treatment Options*

Treatment options for PTL range from non-surgical to surgical. Non-surgical treatment, namely complete decongestive therapy (CDT) by a certified lymphedema therapist, involves a reductive phase and a maintenance phase. The reductive phase involves manual lymphatic decompression (MLD), exercises, compression bandages, and skin care. MLD is a lymphatic massage technique that enhances the filling of cutaneous lymphatics and improves dilation and contraction of lymphatic vessels [31]. The maintenance phase involves lifelong lymph drainage by the patient, exercises, compression garments, and skin care. Principally, because CDT does not treat the underlying pathophysiology of lymphedema, it requires lifelong adherence for effect. Once the patient is no longer adherent to CDT, symptoms recur [32]. CDT has been shown to have positive results in PTL after fractures [33].

In the treatment of burns, myofascial manual lymphatic drainage (a hybrid technique using elements of musculoskeletal medicine and manual lymphatic drainage) increased wound microcirculation, reduced wound healing time, and reduced rates of hypertrophic scarring [34]. The authors recommend implementing the method 2–24 h after surgery, and then performing it once a day till healing is achieved.

Surgery for PTL can be classified into debulking and physiologic surgeries. Debulking surgeries, such as liposuction or excision surgeries, aim to debulk the lymphedematous fibroadipose tissue, but do not treat the underlying pathophysiology of lymphedema. Physiologic procedures, on the other hand, aim to treat the underlying obstruction and restore lymphatic flow, and include lymphaticovenous anastomosis (LVA), vascularized lymph node transfer (VLNT), vascularized lymph vessel transfer (VLVT), and lymph-interpositional-flap transfer (LIFT). Specific to PTL, debulking surgery has only been described in one study, in which it was used as a first-stage procedure to optimize the local environment one month in advance of a vascularized lymph node transfer (VLNT) [35].

#### 2.4.1. Lymphaticovenous Anastomosis (LVA)

LVA involves the supermicrosurgical anastomosis of upstream lymphatic venules to vein venules, bypassing lymphatic obstruction and shunting lymphatic fluid directly into the venous system. Specific to PTL, LVA has been described in a case report of a post-traumatic lymph fistula and intractable ulcer in a severely obese patient [36]. The patient underwent LVA under local anesthesia at three locations at the ankle, and the wound healed 2 weeks after surgery (without recurrence after 3 months). Additionally, the lymphedema improved without the need for compression therapy. LVA was also performed in a case report of post-traumatic penile lymphedema, and resulted in decreased swelling and pain after surgery, without recurrence at 6 months, and without the need for compression garments [37]. There are no case series of LVA for the treatment of PTL.

#### 2.4.2. Vascularized Lymph Node Transfer (VLNT)

Vascularized lymph node transfer (VLNT) refers to the microsurgical transfer of a group of lymph nodes and their vascular pedicle from a donor site to an affected recipient site. At the recipient site, the vascular pedicle of the lymph nodes is anastomosed to recipient blood vessels, without any direct lymphatic anastomoses [38]. The proposed mechanisms for VLNT include lymphangiogenesis from growth factors produced by the transplanted lymph nodes, such as VEGF-C [38], as well as having the transplanted lymph nodes act as a wick via afferent lymphatic channels, which partially drain into the efferent vein of each lymph node [39]. There are multiple options for VLNT donor sites, including submental, supraclavicular, lateral thoracic, thoracodorsal, omentum, and groin. A potential complication of VLNT is iatrogenic donor-site lymphedema, and this should be taken into consideration when choosing a donor site to minimize morbidity. There are no case series of PTL treated with VLNT, but there are case reports. Becker et al. described two cases of VLNT from the groin to the elbow to treat massive, localized hand lymphedema, and described that they were both definitely cured, with return to normal life in 6 months [35]. The VLNT surgeries were preceded by local dermolipectomy and local advancement flap one month prior. One case report describes using a chimeric thoracodorsal lymph node flap with a thoracodorsal artery perforator flap for PTL of the lower limb, with limb circumference reduced by 11%, and the patient reported improved quality of life after 6 months [40]. Another case report describes using a combined latissimus dorsi and split serratus anterior flap (due to bifocal defect) with thoracodorsal lymph nodes to treat lower limb PTL, and lymphedema was clinically reduced at 8 weeks post-operatively [41].

#### 2.4.3. Vascularized Lymph Vessel Transfer (VLVT)/Lymph-Interpositional-Flap Transfer (LIFT)

VLVT involves the use of a free flap with a high density of lymphatic vessels, without any lymph nodes [42]. First described by Koshima et al. in 2016 based on the first dorsal metatarsal vessels [43], further descriptions have used donor sites with a higher density of lymphatic vessels, such as the groin with the superficial circumflex iliac artery perforator (SCIP) flap [44]. VLVT is theorized to mediate its therapeutic efficacy through the peristaltic, pumping actions of healthy lymphatic vessels [39]. The benefits of VLVT are that supermicrosurgical anastomosis of lymphatic vessels is not required [42], and there may be reduced risk of donor-site lymphedema, as no lymph nodes are harvested. In 2018, Yamamoto et al. described the lymph-interpositional-flap transfer (LIFT), which is a modification of the VLVT in which the flap is inset such that the axial ends of donor and recipient lymphatic vessels are aligned and in close proximity, so that lymphangiogenesis can occur [42].

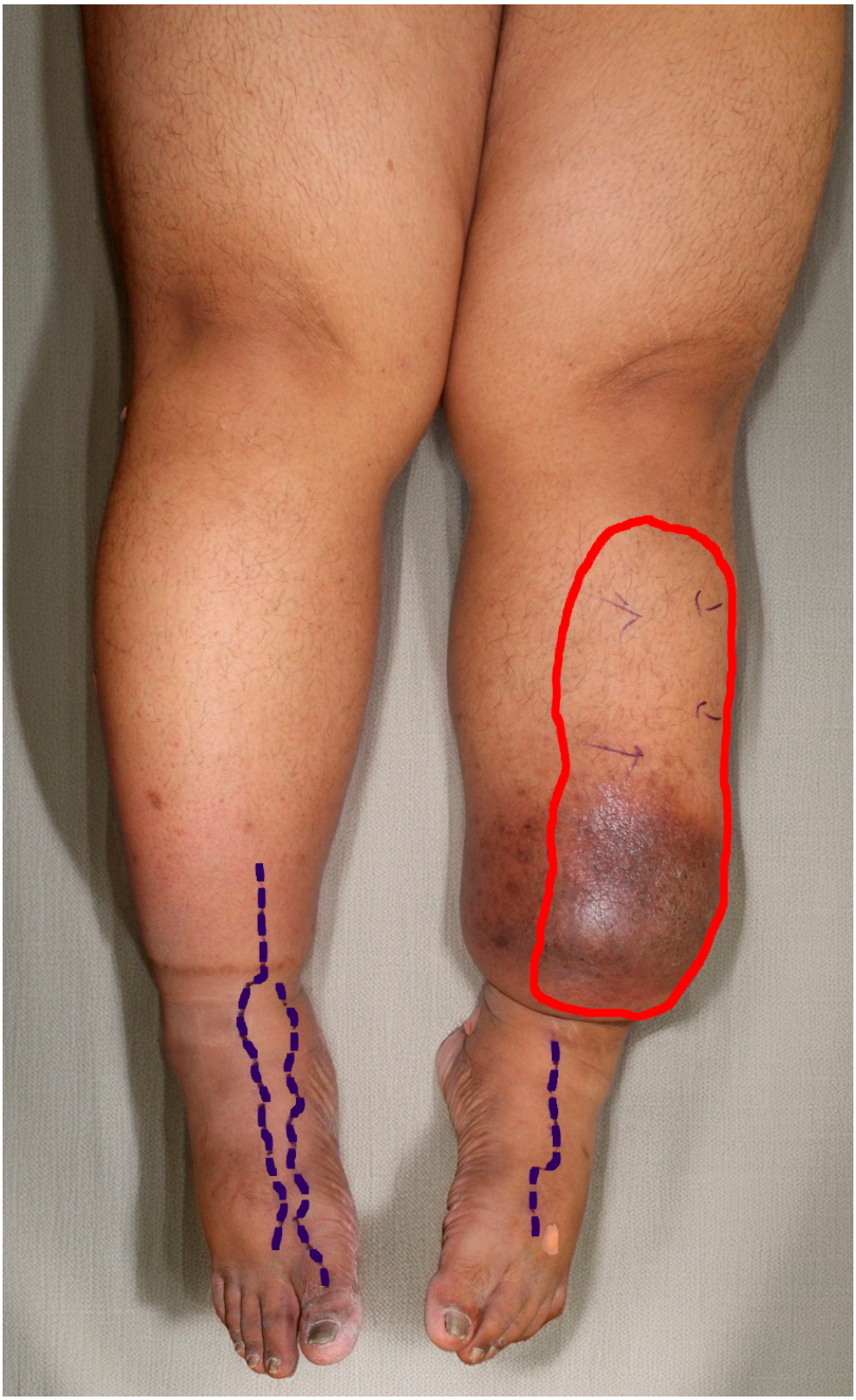
The first report of VLVT used in PTL was a case report in 2019 and describes the use of a superficial circumflex iliac artery perforator lymphatic free flap (SCIP-LV), with a subsequent 56% reduction in excess volume and ICG lymphography demonstration of lymph flow restitution through the flap at 4 months [45]. A follow-up study was performed with 11 patients and followed up for a minimum of 12 months. Five patients had PTL and had SCIP-LV for treatment, while six did not have PTL and were treated with SCIP-LV for prevention. For patients within the treatment group, the mean reduction in excess limb volume was 63%, while quality of life improved by 51%. No patients in the prevention group developed PTL [15].

## 3. Our Clinical Experience

We describe two patients with PTL.

### *3.1. Patient 1*

The patient is a 32-year-old Malay gentleman who sustained a left anterior leg hematoma after tripping over a curb. The swelling from the hematoma reduced significantly in 2 months, but the patient started experiencing a worsening of swelling 3 years later. PTL was diagnosed via ICG lymphography, with linear lymphatic channels disrupted by the left leg hematoma, resulting in dermal backflow. Despite 6 months of intensive complete decongestive therapy (CDT), the swelling did not improve (Figure 1a). As such, he underwent LVA at four incision sites located over the left anterior foot dorsum, left medial calf, left anterior shin, and left medial distal thigh. Dilated lymphatic vessels were found in all four incision sites, and anastomoses were completed with venules with varying degrees of venous reflux. All anastomoses demonstrated expansion and antegrade flow of lymph fluid into the venule after manual compression by hand distal to the LVA site. Compression therapy was initiated immediately after completion of LVA while on the operating table to minimize venous reflux into the lymphatic vessel. Significant improvement in limb volume from 13,180 mL (Figure 1a) to 9490 mL (Figure 1b) was noted at the 18th month post-operative mark. This equates to a 28% reduction in limb volume. The patient also experienced improvement of LeQOLis score from 71 (assessed after 6 months of CDT and pre-operatively) to 19 one and a half years post-operatively.



(**a**)

**Figure 1.** *Cont.*

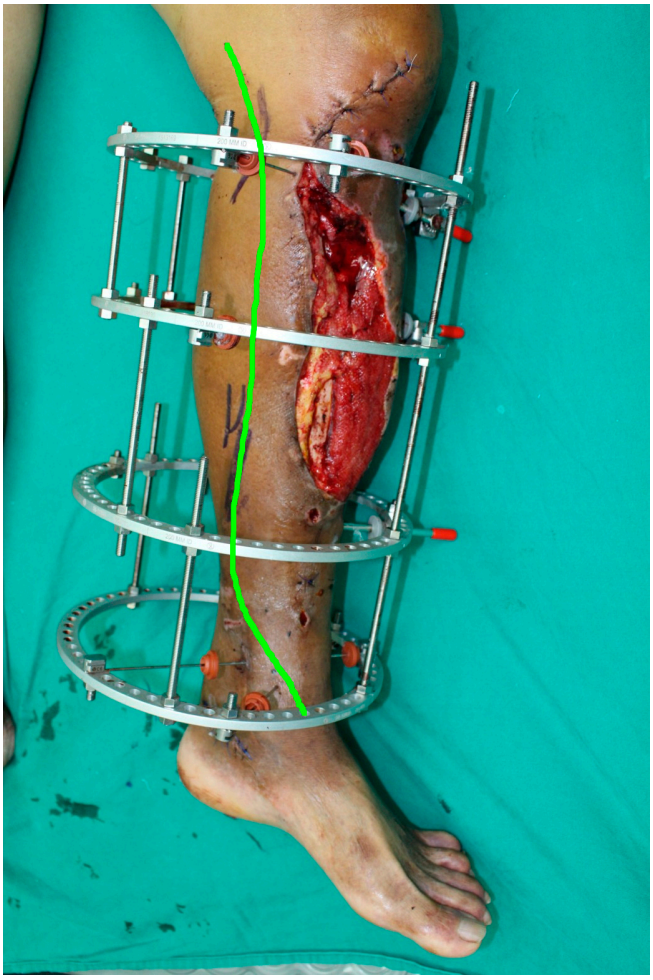


(**b**)

**Figure 1.** (**a**) Patient 1 sustained a left anterior leg hematoma after tripping over a curb. Despite 6 months of intensive complete decongestive therapy (CDT), the swelling did not improve, with linear lymphatic channels (dotted blue lines) disrupted by the left leg hematoma, resulting in dermal backflow (encircled in red) seen on ICG lymphography. As such, he underwent LVA at 4 incision sites; (**b**) Patient 1, 18 months after LVA with a 28% reduction in limb volume and improvement of LeQOLis score from 71 to 19.

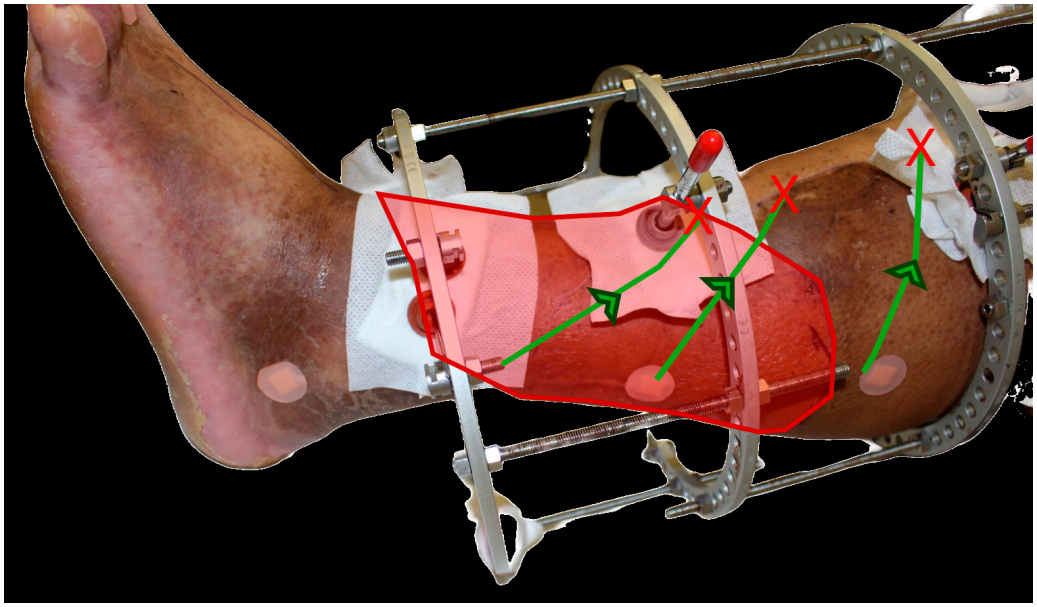
### *3.2. Patient 2*

Patient is a 58-year-old Chinese gentleman who sustained a left open tibia fracture (Gustilo 3B) after a road traffic accident, with a soft tissue defect of 21 × 8 cm in size. The fracture was stabilized with external fixation (Figure 2a), and the soft tissue defect was reconstructed with lymph-interpositional-flap transfer (LIFT) free anterolateral thigh (ALT) flap, with the primary objective of soft tissue coverage and secondary objective of reconstituting lymphatic flow. Post-operatively, the patient experienced swelling and tightness of the affected leg, especially at the lateral aspect (Figure 2b). ICG lymphography demonstrated dermal backflow of the entire lower limb from the foot to the knee. As the ring fixators were in situ for 4 months, decongestive therapy (which is the standard post-operative treatment 1 month after LIFT to promote lymphatic flow through the reconstituted lymphatics) could not be initiated for the patient. Internal fixation was accomplished 4 months after the injury. One and a half years after the LIFT ALT flap, anteromedial dermal backflow was resolved, while the lateral dermal backflow was reduced, and fluorescent dye could be seen flowing into the flap during ICG lymphography (Figure 2d), demonstrating reconstitution of lymphatic channels. LeQOLis scores improved from a maximum score of 57 at three months post-injury, to 22 one and a half years post-injury. He experienced a maximum reduction of 13.7% in lower limb volume (Figure 2c) compared to his maximum limb volume at the 3-month post-injury mark.



(**a**)

**Figure 2.** *Cont.*

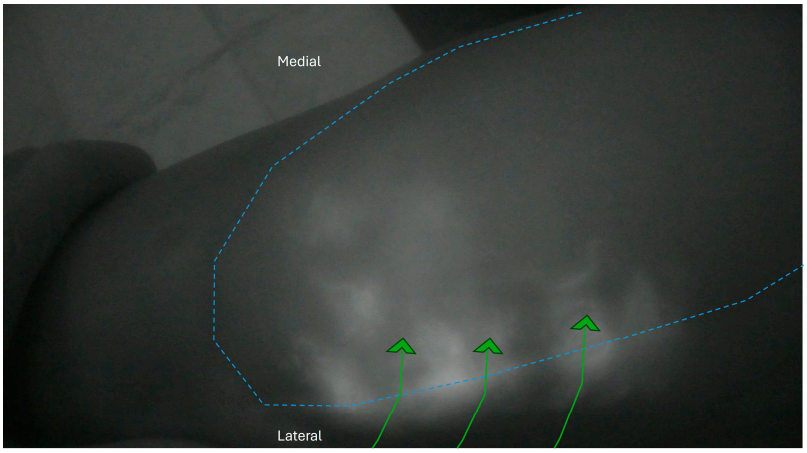


(**b**)



(**c**)

**Figure 2.** *Cont*.




(**d**)

**Figure 2.** (**a**) Patient 2 sustained a left open tibia fracture (Gustilo 3B) after a road traffic accident, with a soft tissue defect of 21 × 8 cm in size. The fracture was stabilized initially with external fixation. There is a linear flow of lymphatics (green line) at the anteromedial leg (i.e., anteromedial lymphatic tracts not disrupted); (**b**) 3 months after LIFT, Patient 2 still experienced swelling and tightness of the affected leg, especially at the lateral aspect, with ICG lymphography demonstrating dermal backflow (red shaded area). As the ring fixators were in situ for 4 months, CDT (which is the standard post-operative treatment 1 month after LIFT to promote lymphatic flow through the reconstituted lymphatics) could not be started for the patient post-operatively. Lymphatic channels (green lines, with arrows denoting direction of flow) at the lateral lymphosome were disrupted (red crosses) by the injury site, resulting in dermal backflow (red shaded area). To note, the patient also had dry and flaky skin in addition to limb swelling; (**c**) 18 months after LIFT, with resolution of PTL. Lower limb volume reduced by 13.7% and LeQOLis scores improved from 57 to 22. Improvement in skin quality is also visible; (**d**) 18 months after LIFT, with resolution of PTL. Fluorescent dye was injected laterally (where channels were previously disrupted) and can be seen flowing medially (green arrows) into the LIFT (dotted blue lines) during ICG lymphography—the previously disrupted lymphatic channels at the injury site have reconstituted.

## 4. Discussion

The two cases we described involved injuries to the lower limbs, specifically affecting the anteromedial leg, a region known for its high lymphatic vessel density. In our experience, most cases of post-traumatic lymphedema (PTL) resolve within six months, particularly when the initial injury is mild, such as a contusion. However, certain factors appear to predispose patients to chronic PTL. These include open fractures with extensive soft tissue loss, particularly Gustilo type 3B injuries or higher, occurring in areas of high lymphatic density. The risk is further amplified when the wound is deep, with loss of skin and subcutaneous fat, where many lymphatic collectors reside. Additionally, wounds managed with skin grafts rather than flap reconstruction may carry a higher risk of developing PTL.

We hypothesize that both the nature of the initial trauma and the subsequent healing process contribute to the development of chronic PTL. High-energy open fractures (Gustilo 3B or higher) typically result in substantial soft tissue disruption, including damage to lymphatic vessels. When such injuries occur in regions with dense lymphatic architecture, the disruption to primary lymphatic pathways is compounded, impairing drainage to the inguinal lymph nodes. If the soft tissue defect is allowed to heal by secondary intention or is reconstructed with a skin graft, the resulting fibrosis may further obstruct lymphatic flow. In contrast, flap reconstruction may preserve or introduce viable lymphatic vessels or nodes, potentially promoting lymphangiogenesis via growth factors such as VEGF-C [38]

or re-establishing lymphatic axiality within the affected lymphosome, thereby mitigating the risk of PTL.

We acknowledge that there are multiple etiologies for post-traumatic edema, and post-traumatic lymphedema is only one of them. As such, we recommend investigating other causes of post-traumatic edema as well, as per clinical suspicion, and treating them accordingly. Once other causes have been ruled out, we recommend the use of indocyanine green (ICG) lymphography to investigate for PTL. With regard to PTL, ICG lymphography facilitates confirmation of diagnosis, disease staging, and preoperative planning through real-time visualization of lymphatic architecture. In our clinical protocol, ICG is diluted in 5% dextrose and administered via subdermal injections at the second webspace on the dorsum of the foot, as well as the medial and lateral malleoli. Real-time lymphographic imaging is then performed using the Stryker SPY Portable Near-Infrared Imaging System, with dermal backflow patterns graded according to the ICG staging system described by Yamamoto et al. [28]. Given the time required for ICG to disseminate throughout the entire limb, we conduct the imaging in two stages: the first immediately after injection to assess initial lymphatic flow, and the second approximately four hours later to enable full visualization of lymphatic drainage across the limb.

In treating PTL, we recommend a multidisciplinary management approach, beginning with early identification and risk stratification by orthopedic and reconstructive surgeons. This should be followed by coordinated treatment involving certified lymphedema therapists and lymphedema surgeons. Management should be tailored to each patient, taking into account the full spectrum of available therapies—from conservative non-surgical options to advanced surgical interventions—based on the severity of disease, response to prior treatment, and individual clinical factors.

At our center, we advocate for an initial trial of complete decongestive therapy (CDT) for a minimum duration of six months. This reason is two-fold. Firstly, in our experience, most cases of PTL are resolved within 6 months. Secondly, we acknowledge that even if there is evidence of PTL as confirmed by ICG lymphography, there may be other causes contributing to the post-traumatic edema, such as inflammation, which will reduce over time.

If there is no significant clinical improvement after 6 months of conservative treatment, and other causes of post-traumatic edema have been investigated and addressed, we discuss surgical options for treating PTL with the patient. Lymphaticovenous anastomosis (LVA) is typically our first-line surgical intervention due to its minimally invasive nature, rapid recovery time, and immediate decongestive effect. In cases where improvement remains suboptimal after 12 months, further interventions such as lymph-interpositional-flap transfer (LIFT), repeat LVA, or vascularized lymph node transfer (VLNT) may be considered. Among these, we prefer LIFT over VLNT, as LIFT avoids lymph node harvest and thereby theoretically reduces the risk of iatrogenic donor-site lymphedema.

In cases involving soft tissue defects, such as those seen in open fractures, we recommend primary reconstruction using the LIFT technique. Instead of just reconstructing the soft tissue defect with a free flap and insetting it in a random orientation, the LIFT technique orients the free flap such that donor and recipient lymphatic vessels are aligned in close proximity. This reconstructs the soft tissue defect, as well as facilitates lymphangiogenesis and restoring lymphatic continuity. In our experience, this approach yields optimal outcomes in managing PTL without much technical difficulty added to the surgery. As such, we advocate for the use of LIFT in all traumatic soft tissue reconstructions as a prophylactic strategy against PTL development [46]. Two flap options we commonly utilize are the anterolateral thigh (ALT) flap, which features lymphatic channels running in a superior-oblique direction (lateral to medial), and the superficial circumflex iliac artery

perforator (SCIP) flap, with channels oriented in an inferior-oblique direction along the long axis of the flap. While flap selection is ultimately guided by the nature and location of the defect, the SCIP flap is frequently favored in our practice due to its high lymphatic vessel density and favorable donor-site cosmesis.

With regard to post-operative management, we recommend initiating compression bandaging immediately following lymphaticovenous anastomosis (LVA) to promote lymphatic flow and minimize venous reflux. Once limb volume and contour is stabilized, patients are transitioned to compression garments, which should be maintained for a minimum of 12 months to sustain therapeutic benefit. In contrast, for patients undergoing lymph-interpositional-flap transfer (LIFT), we defer the initiation of compression therapy for approximately one month post-operatively to protect the integrity of the free flap and to ensure adequate tissue integration.

## 5. Conclusions

Although trauma is increasingly recognized as a significant cause of secondary lymphedema, post-traumatic lymphedema (PTL) remains an underdiagnosed and underreported condition, with publications being either case reports or case series with small sample sizes. It should be regarded as a distinct clinical entity that warrants further investigation through studies involving larger patient cohorts and extended follow-up durations. We advocate for the use of indocyanine green (ICG) lymphography for the diagnosis and staging of PTL, owing to its safety, accuracy, and widespread adoption by high-volume centers.

In terms of management, a multidisciplinary approach involving both surgeons and certified lymphedema therapists is essential. Treatment should be tailored to the individual, incorporating the full spectrum of conservative and surgical interventions. We recommend an initial six-month trial of complete decongestive therapy (CDT). If the patient does not experience meaningful improvement, lymphaticovenous anastomosis (LVA) should be offered as a first-line surgical option due to its minimally invasive nature, rapid recovery, and immediate therapeutic benefit. For patients who show inadequate response after 12 months, further interventions such as lymph-interpositional-flap transfer (LIFT), repeat LVA, or vascularized lymph node transfer (VLNT) may be considered. We prefer LIFT over VLNT, as it avoids lymph node harvest and thus reduces the theoretical risk of donor-site lymphedema. Additionally, we recommend performing LIFT prophylactically in cases of open fractures with significant soft tissue defects to prevent the onset of PTL.

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**Informed Consent Statement:** Written informed consent has been obtained from the patients to publish this paper.

**Data Availability Statement:** The raw data supporting the conclusions of this article may be made available by the authors on request, at the discretion of the authors.

**Conflicts of Interest:** The authors declare no conflicts of interest.

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