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MRI Evidence of Improvement in Glucocorticoid Induced Osteonecrosis After Intraneural Facilitation™ (INF®) Treatment

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List of Abbreviations:

T-LL: T-cell Lymphoblastic Lymphoma; ALL: Acute Lymphoblastic Leukemia; ON: Osteonecrosis; INF®: Intraneural Facilitation™; MRI: Magnetic Resonance Imaging; AROM: Active Range of Motion; WNL: Within Normal Limits; CT: Computed Tomography; IL1B: Interleukin 1 Beta; VIPN; Vincristine-Induced Peripheral Neuropathy; ATP: Adenoside Triphosphate

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1. Abstract

1.1. Objective

To evaluate the effectiveness of Intraneural Facilitation™ (INF®) treatment in improving glucocorticoid-induced osteonecrosis (ON) in a pediatric patient undergoing treatment for T-cell lymphoblastic lymphoma.

1.2. Methods

A thirteen-year-old female patient with ON, who received INF® treatment after conventional therapies failed. The patient underwent INF® therapy to enhance neural microvascular blood flow, with subsequent improvements in pain, strength, and functionality.

1.3. Results

After three months of INF® treatment, the patient showed significant improvements, including increased mobility, reduced pain, and improved quality of life. Follow-up MRIs demonstrated a substantial reduction in the size of ON lesions in both femoral and tibial regions.

1.4. Conclusion

This case suggests that INF® treatment may improve symptoms of glucocorticoid-induced ON by enhancing neural and vascular circulation, potentially reversing ON through sympathetic nerve regeneration. Further research with larger cohorts is recommended to confirm these findings.

2. Background

Glucocorticoid therapy is used in conjunction with chemotherapy to successfully treat 86.7% of pediatric T-cell lymphoblastic lymphoma (T-LL) cases [1]. While T-LL is a distinct diagnosis, it is treated using protocols similar to those for acute lymphoblastic leukemia (ALL) [2, 3]. Chemotherapy agents used in conjunction with the steroid therapy protocol AAL0434, include Vincristine and methotrexate. Glucocorticoid therapy is an important therapy for TLL, with osteonecrosis (ON) being considered a primary therapeutic side-effect [3, 4]. Incidence of steroid induced ON varies widely depending on when the study occurred and the diagnostic methods. For instance, Chen et al. found that 17.6% of patients treated with glucocorticoids for ALL had symptomatic ON, while asymptomatic ON affected up to 53.9% of these patients [5]. Ten percent of the 500,000 joint replacements are due to steroid-induced necrosis [6]. An estimated 30 million Americans require glucocorticoid treatment, and only trauma causes more instances of osteonecrosis than steroid-induced ON [6]. ON typically develops between two months and five years after the diagnosis of ALL, with most symptomatic cases occurring in the first two years [7]. Children aged 10 years or older are more prone to developing ON [7]. Steroid-induced ON pathogenesis is multifactorial, with resultant osseous blood flow reduction [8]. These factors include fat cell hypertrophy, intravascular coagulation, increased intraosseous pressure, and dysfunction, all contributing to impaired micro-

circulation and bone ischemia [9]. Intraneural facilitation (INF®) is a novel non-surgical treatment manual therapy with anecdotal success in improving sensory impairment associated with chemotherapy-induced neuropathy and diabetic neuropathy. In a pilot study, INF® treatment improved Neuro Com sensory organization test balance and modified total neuropathy scale scores in patients with diabetic neuropathy [10]. In a clinical trial, INF® treatment improved pain perception, composite static balance score on Neuro Com, and protective sensory in patients with diabetes type II [11]. A retrospective study concluded in 2024 observed neuropathic improvement in pain symptoms [12]. This case study aims to describe how INF® treatment may have substantially improved a patient's steroid-induced ON by increasing neural microvascular blood flow and subsequent intramedullary circulation.

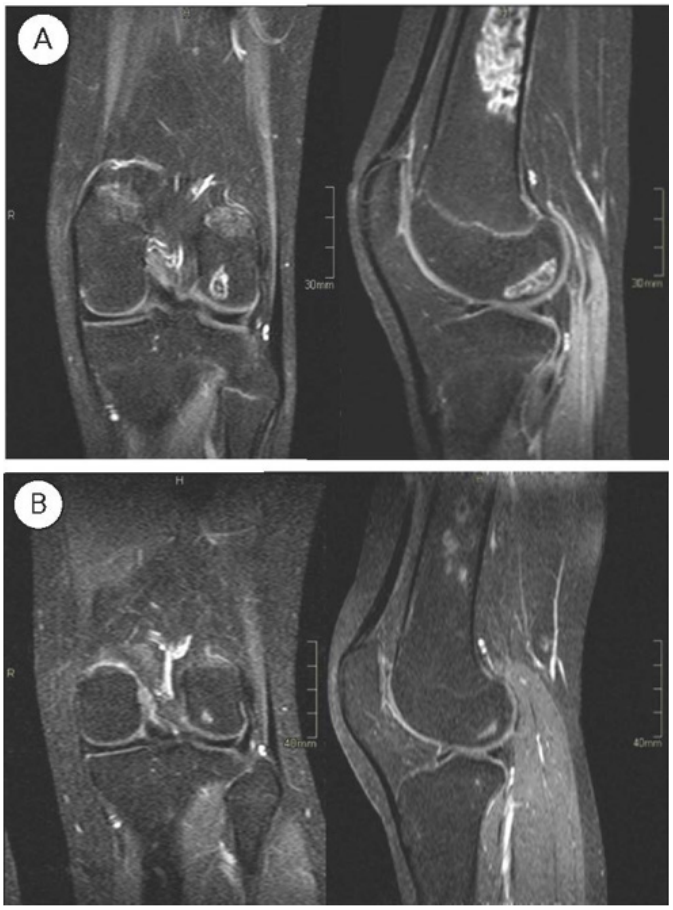
3. Case Presentation

The 13-year-old patient presented to the emergency room reporting migraines, rib soreness, increased fatigue, and one month of the same day onset of facial swelling and increased shortness of breath. Chest x-ray and CT showed a right chest mediastinal mass, and the patient was admitted to the hospital in the care of pediatric oncology. A bone marrow biopsy was performed, which was negative for malignant infiltration, with pathology showing positive for precursor T-cell lymphoblastic lymphoma. The patient enrolled in the chemotherapy protocol AALL0434. Per the AALLO434 protocol, she received steroid therapy from days 1-28 (prednisone). Other medications received that are part of protocol AALL0434 and pertinent to this case include Vincristine, methotrexate, and Nelarabine. Once the ALL0434 protocol was initiated, the patient began experiencing a decline in function, marked by increased lower extremity pain and decreased mobility. These symptoms were attributed to vincristine-induced sensory and motor neuropathy, resulting in pain, reduced sensation, decreased active dorsiflexion, and impaired functional mobility. Medication management provided limited relief. Eight months post-diagnosis, MRI imaging confirmed the presence of osteonecrosis (ON), contributing further to her pain. Two months later, the patient began physical therapy with goals focused on pain reduction and regaining independence in functional activities. During the subjective portion of her initial physical therapy examination, the patient complained of joint stiffness, muscular weakness, impaired balance, and hypersensitivity in bilateral lower extremities. Her pain at rest was a 4/10, which increased significantly during activity. The patient exhibited poor posture, core weakness, and difficulty standing. Visual observation revealed atrophy in the gluteal, hamstring, and quad muscle groups. Manual muscle test scores were 4-/5 in bilateral upper extremities, with bilateral quadriceps, hamstrings, gastrocnemius, and anterior tibialis muscle groups all exhibiting a 3+/5 muscle grade. The active range of motion (AROM) was within normal limits (WNL) except for the right knee at -5 degrees of knee extension, and the bilateral ankles lacked dorsiflexion beyond 0. Sensory testing revealed sharp/dull was intact grossly, monofilament light touch was 3.22, and vibration using the biothesiometer was WNL in both great toes. With palpation assessment, the patient had skin hyperalgesia with light touch in both lower extremities. A visual assessment revealed dark skin discoloration on the distal phalanges, especially when sitting on the edge of a mat. Due to pain and muscle weakness, the patient could not ambulate and required standby assistance with pivot transfer. In her second therapy session, the patient refused assisted gait, transfer, or strengthening activities due to pain in her lower extremities, and she was referred for INF® therapy. INF® therapy is a specialized manual treatment developed in South Carolina and introduced at Loma Linda University in 2011 [12]. It is designed to improve blood flow to nerves suffering from ischemia, using a systematic series of three gentle manual holds. These are performed by trained physical therapists who have an in-depth understanding of neurovascular anatomy and physiology [10-13]. The process begins with the first hold, called the facilitation hold, which places the joint on the contralateral side of the body into its most relaxed or "loose-packed" position [11, 12]. This positioning is believed to allow the nerve to stretch more than the accompanying artery, because arteries have more elastic tissue, thus creating a gentle pull on the tiny coiled vessels (called nutrient vessels) that connect nerves and arteries [10-13]. This stretch may enlarge the junction where these vessels meet the nerve, improving blood flow into the nerve's outer layer, the epineurium [10-13]. This process sets up a pressure gradient, increasing circulation into the outer chamber of the nerve and preparing it for deeper microcirculatory flow [10-13]. Next is the second hold, which applies a targeted stretch on the affected side to guide the increased blood flow from the epineurium into the deeper trans perineurial vessels [10-13]. These vessels bridge the outer layer of the nerve (epiperineum) with its inner capillary network (endoneurial capillaries) [10-13]. This step targets the areas most impacted by poor circulation, helping blood reach the internal nerve structures where it's needed most. The third hold, often called the sub hold, uses bolsters and distant pressure points to encourage ongoing circulation [11, 12]. It applies principles similar to Bernoulli's principle: by creating a low-pressure zone away from the treatment site, it helps draw blood through the high-resistance, ischemic endoneurial capillaries [10-13]. This final step promotes thorough perfusion of both the outer (epineurial) and inner (endoneurial) vascular systems of the nerve [10-13].

In clinical practice, the second and third holds are often repeated on the affected side for the remainder of the treatment session, enhancing and sustaining the circulatory effects [10-13]. Together, these three steps aim to reestablish healthy blood flow within damaged or compressed nerves, supporting nerve recovery and function [10-13]. With INF therapy, the patient reported immediate relief, a "lightness in her feet" and significant improvement with INF® treatment. After one month of INF® therapy, the patient reported decreased pain in her legs and feet and reduced swelling in her legs. Improvement in her lower extremity discol-

oration was also noted. After two months of therapy, the patient still used a wheelchair but had no pain with bed mobility and pivot transfers. After two and a half months, the patient reported continued improved activities of daily living, distal extremity color, and decreased paresthesia. The patient stopped using her wheelchair and could walk again with a front-wheeled walker. After three months of INF® treatment, the patient ambulated independently. Furthermore, the patient did not have swelling in her lower extremities and had standard color in her toes. The patient's INF® therapy regimen continued for 18 months as she received maintenance chemotherapy, including monthly doses of Vincristine (total of 38 doses, maximized at 2mg), daily oral mercaptopurine, and weekly oral methotrexate and intrathecal methotrexate every three months. The patient complained of increased lower extremity pain after her chemotherapy administration but reported immediate improvement after an INF® treatment course. During these 18 months, her activity level dramatically increased. The patient reported that she was able to walk and run for functional distances. The patient started participating in social activities, including school dances and outings to local shopping malls. The patient's lower extremity strength increased to 4+/5 in knee extension and all ankle motions. Two years after her initial MRI, a follow-up MRI (FIGURES 1 & 2) was completed to assess her ON status. The follow-up MRI showed a significant reduction in osteonecrosis in both knees. Lesions in the distal femoral and tibial diaphysis showed substantial size reduction. Measurements of the distal lateral femoral epiphyseal lesions revealed a marked decrease in lesion size compared to the baseline MRI (Table1).

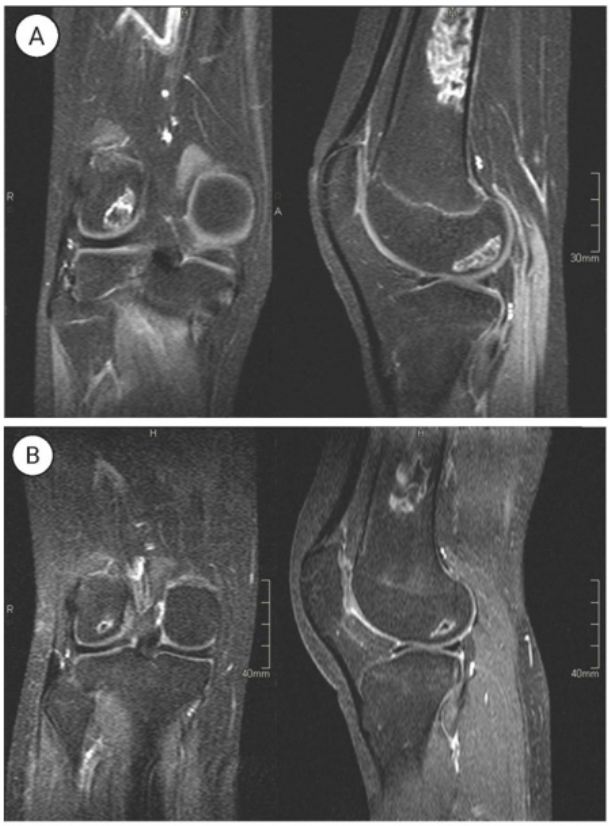
Figure 1: Initial and Follow-up MRI for Left Distal Femoral Lateral Epiphysis.



A. Initial MRI on 9/10/2013, coronal and sagittal post-contrast T1 (T1-weighted) image showed an enhancing lesion at the left distal femoral lateral epiphysis, consistent with osteonecrosis. This lesion measured 1.5-centimeters (cm) Craniocaudal (CC), 1.7-cm Transverse (TR) and 1.9-cm Anteroposterior (AP).

B. Follow-up MRI on 8/18/2015, coronal and sagittal post-contrast T1 image showed the enhancing lesion in the left distal femoral lateral epiphysis had significantly decreased in size, 0.9-cm CC, 0.4-cm TR and 0.9-cm AP (93% reduction in volume).

Figure 2: Initial and Follow-up MRI for Right Distal Femoral Lateral Epiphysis.



A. Initial MRI on 9/10/2013, coronal and sagittal post-contrast T1 (T1-weighted) image showed an enhancing lesion at the right distal femoral lateral epiphysis, consistent with osteonecrosis. This lesion measured 1.2-centimeters (cm) Craniocaudal (CC), 0.7-cm Transverse (TR) and 2-cm Anteroposterior (AP).

B. Follow-up MRI on 8/18/2015, coronal and sagittal post-contrast T1 image showed the enhancing lesion in the left distal femoral lateral epiphysis had significantly decreased in size, 0.9-cm CC, 0.8-cm TR and 1.3-cm AP (44% reduction in volume).

Table 1: Showing MRI Comparing 3-Axis Measurements of ON lesions before and after INF®

	Left Lateral Epiphysis	Right Lateral Epiphysis
9/10/13	1.5-cm CC, 1.7-cm TR and 1.9-cm AP	1.2-cm CC, 0.7-cm TR and 2.0-cm AP
8/18/15	0.9-cm CC, 0.4-cm TR and 0.6-cm AP	0.9-cm CC, 0.8-cm TR and 1.3-cm AP

4. Discussion and Conclusions

The reason for the development of glucocorticoid-induced ON is generally considered to be the impact steroid therapy has on the complex relationship between osteoclastic and osteoblastic activity [14]. Osteoclastic lifespan is enhanced while osteoblastic apoptosis is increased [14]. Osteocyte (a type of osteoblast) and bone marrow death occur, followed by necrotic tissue uptake and weaker bone reformation [15]. On closer inspection, glucocorticoid therapy induced ON appears multifactorial. Adipose cell hypertrophy and proliferation occur with steroid therapy, increasing osseous pressure and decreasing intramedullary blood flow [7, 9]. Vascular endothelial cells are also impacted by steroid therapy, leading to an increased risk of thrombotic occlusion and an increased risk of reduced intermedullary circulation [9]. Of interest for this case is the link between ON and denervation, with sympathetic nerve loss impacting local blood flow and bone resorption [16]. Sympathetic regulation of osteoclast and osteoblast activity has been demonstrated, with sympathetic nerves modulating bone reabsorption [17] and disrupting osseous homeostasis [18]. Lower concentrations of norepinephrine produced by fewer sympathetic nerves may increase norepinephrine affinity for alpha-adrenergic receptors, producing vasoconstriction [18]. A higher density of sympathetic nerves producing a higher concentration of norepinephrine may allow for vasodilation since norepinephrine affinity for alpha¬ adrenergic receptors is not nearly as strong at higher density levels [19]. When comparing the intertrochanteric bone cylinder of patients with ON versus patients with osteoarthritis, the patients with ON had a 3.5:1 ratio of sensory nerves to sympathetic nerves. However, the opposite was true of patients with osteoarthritis [16]. Reduced microcirculation, resulting in

endothelial compromise, plays a prominent role in axonal denervation in patients with diabetic neuropathy [6, 20]. Multiple agents associated with protocol AALL0434 are associated with endothelial compromise and are implicated in the sympathetic axonal demise previously described. Increased cellular glucose is a byproduct of glucocorticoid therapy, which damages insulin receptors and increases cellular glucose [21]. Increased glucose is associated with AGE/RAGE reactions [22], resulting in vascular inflammation, including endoneurial capillary endothelial dysfunction [23]. A prominent side effect of vincristine is neurotoxicity. Vincristine is known to cause both sensory and motor peripheral neuropathy, often resulting in distal extremity weakness [24]. This typically presents as decreased ankle dorsiflexion strength in the feet and reduced grip strength due to involvement of the intrinsic muscles of the hands [25]. Mechanistically, vincristine promotes neural inflammation through disrupting axonal microtubule formation [26], inducing IL1B signaling in local macrophages, and damaging the blood-nerve barrier by promoting endothelial dysfunction [27]. INF® is posited to mechanically induce blood flow into constricted capillaries of osseous sympathetic nerves through relational vascular optimization. An INF® session involves three separate holds maintained semi-concurrently, during a treatment sequence, lasting one hour [10, 11]. The artery/epineurial arteriole relationship is optimized through the first hold, with vascular induction occurring through the second hold (stretching the perineurium), directing consistent pressurized circulation into dysfunctional endoneurial capillaries [10, 11]. The endothelial cells comprising these capillaries are conceptually constricted due to the combined ALL-treating agents, creating increased peripheral resistance [11]. However, with the flow induction previously described, endoneurial capillary dysfunction is reversed with the potential of resuming a normal blood nerve barrier. The patient's initial presentation at therapy, marked by weakness and pain, is likely multifactorial in origin. Periosteal pain from osteonecrosis and peripheral neuropathy-resulting from multi-therapeutic side effects impacting both the nerve axon and the nervi nervorum-may explain the patient's hyperalgesia and functional challenges [28]. The immediate reduction in symptoms, combined with significant functional improvement over the first 2½ months, may be attributed to both the intended effects of the therapeutic modality and the discontinuation of vincristine [29]. While vincristine-induced peripheral neuropathy (VIPN) is cumulative and tends to worsen during treatment, symptoms often improve once the drug is discontinued, although full resolution may not occur [30, 31]. The relationship between symptom improvement and MRI findings requires further investigation. Fascicular pressure, which creates a microcompartment syndrome, may result from steroid-induced endothelial dysfunction and hypercoagulability. Without fascicular lymphatic drainage, it is uncertain whether fascicular capillary circulation will be restored even after the steroid therapy has ended. The resultant osseous sympathetic neural dysfunction or denervation may insufficiently regulate intramedullary blood flow, thus indefinitely promoting ON. Induced pressurized flow into constricted capillaries creates a sheer force on red blood cells, promoting ATP release [32, 33]. ATP release is part of a cascade resulting in endothelial dilation. ATP first interacts with the endothelial cell, resulting in nitric oxide release [34], with nitric oxide stimulating the overlaying pericyte cell to promote capillary dilation [35]. Improved microvascular circulation promotes nerve regrowth and is a precursor to nerve regeneration [36], potentially leading to improved osteoclastic and osteoblastic osseous regulation. The pathophysiology associated with ON often results in articular surface collapse, osteoarthritis, subchondral bone fracturing from repetitive weight bearing [37], and femoral head collapse [38]. As ON progresses, it can severely impair quality of life, leading to both short-and long-term disability [4]. Therapeutic strategies include physical therapy and surgical interventions for late-stage ON [39]. Orthopedic surgery is considered if more conservative measures fail. "Orthopedic management of symptomatic ON may include core decompression, arthrodesis, and joint replacement" [39]. Skeletal immaturity in the pediatric population may increase the risk of a failed orthopedic surgery or a poor outcome [40]. Future research involving INF® treatment of glucocorticosteroid-induced ON is indicated to assess whether INF® can provide additional support to this patient population. Glucocorticoid-induced osteonecrosis is a significant complication in pediatric oncology, particularly in patients undergoing treatment for T-cell lymphoblastic lymphoma. INF® therapy may provide a non-surgical option for improving microvascular circulation in patients with ON. INF therapy may counteract steroid-induced endothelial dysfunction and provide an optimal environment for osteoclastic/osteoblastic regulating sympathetic nerve regrowth. In this case, MRI imaging showed the effectiveness of INF® therapy in reversing this patient's ON. Larger, controlled studies are needed to confirm the therapeutic efficacy of INF® and its role in treating glucocorticoid-induced osteonecrosis.

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