Osteopathic Approach to Sacroiliac Dysfunction in a Patient With Steroid Myopathy: Case Report and Literature Review

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Long-term steroid use has a well-documented risk of myopathy that imposes functional limitations for patients and challenges for health care providers. Proximal weakness from steroid myopathy affects support structures around the pelvic girdle and likely predisposes patients to somatic dysfunction. To the authors’ knowledge, there are no prior reports in the literature that describe an osteopathic manipulative medicine (OMM) approach for patients with steroid myopathy. In the present case report, a 59-year-old woman with acute myeloid leukemia received a blood stem cell transplantation and developed gastrointestinal graft-versus-host disease. High-dose steroids were prescribed, and she developed proximal weakness from steroid myopathy. The patient’s acute inpatient rehabilitation was impacted by new onset left sacroiliac dysfunction. A patient-focused OMM approach was used to assist the patient in maximizing her sacroiliac function. The proximal weakness seen with steroid myopathy necessitates special considerations for an OMM approach to address somatic dysfunction associated with this disease.

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Long-term steroid therapy is integral in the management of a variety of inflammatory, autoimmune, and neoplastic disorders. Unfortunately, there have been well-documented risks of long-term steroid use. In 1958, 10 years after the introduction of steroid therapy, Dubois reported the first case of iatrogenic steroid myopathy. Myopathies are diverse conditions that primarily affect skeletal muscle and result in proximal weakness, fatigue, decreased endurance, and deformities. The present report focuses on chronic acquired steroid-induced myopathy. This toxic myopathy is due to long-term administration of exogenous systemic glucocorticosteroid and commonly presents with proximal muscular weakness and eventual muscle atrophy. The purpose of the present report is to provide the first description, to our knowledge, of an osteopathic manipulative medicine (OMM) approach for management of somatic dysfunction associated with steroid myopathy. We describe an unexplored aspect of patient management within a well-documented disease state. In addition, we provide a review of the small body of literature for the pathogenesis, diagnosis, and management of steroid myopathy.
A 59-year-old woman with acute myeloid leukemia (AML) was admitted to a university hospital with complaints of diarrhea, nausea, and vomiting. The patient had a past medical history of goiter, fibrocystic breast disease, and migraines. Her previous home-scheduled medications included acyclovir, budesonide, calcium citrate supplement, cyclosporine, ergocalciferol, esomeprazole, fluoxetine, magnesium supplement, levothyroxine, omega-3 fatty acids, prednisone, and voriconazole. The patient denied a history of cigarette smoking but reported consumption of alcohol in social settings. She had a family history of cancer, including a father with prostate cancer, bladder cancer, and colon cancer; a brother with renal cell carcinoma; and a sister with AML.

At the time of admission to the hospital, the patient’s vital signs were as follows: temperature, 37.3°C; heart rate, 88 beats per minute; respiratory rate, 20 breaths per minute; and blood pressure, 162/86 mm Hg. She had an oxygen saturation of 97% while breathing room air. She was able to move all extremities against gravity. The patient reported that nearly 1 year ago, she received peripheral blood stem cell transplantation from an unrelated donor for AML. Two days after admission, an esophagogastroduodenoscopy-obtained biopsy specimen confirmed a flare of upper gastrointestinal inflammation consistent with graft-versus-host disease (GVHD). The patient had a prior history of GVHD, with the most recent flare having occurred 8 months earlier. Before admission, the patient was taking prednisone, 7.5 mg every other day, and budesonide, 3 mg twice daily, for immunosuppression. After the biopsy procedure, the patient started intravenous prednisolone therapy at 2 mg/kg twice daily, continued taking budesonide, and began taking tacrolimus in an effort to modulate the immune response of the GVHD. In addition, 29 days after admission, the patient began extracorporeal photopheresis immunomodulatory therapy twice weekly for a total of 6 treatments.

A synthetic glucocorticoid screen was performed at admission to the hospital; results were returned 13 days later and identified high levels of budesonide absorption. Three weeks after the patient’s high-dose steroid was increased, she reported difficulty rising from a chair to the standing position. The timing of the increase in high-dose steroid use, the physical examination findings, and the laboratory results were consistent with the diagnosis of steroid-induced myopathy. Budesonide was initially changed to methylprednisolone, and over 4 weeks the steroid dosage was tapered to her original maintenance dose of prednisone, 7.5 mg once daily. The patient was closely monitored for recurrence of gastrointestinal GVHD. During her hospital stay, the patient continued to receive chemotherapeutic agents that included rituximab and infliximab. In addition, various prophylactic agents were administered because of the patient’s immunocompromised state.

At admission to the hospital, the patient performed activities of daily living with no assistance and lived alone in a 2-story home with 4 steps to enter. On physical therapy evaluation approximately 6 weeks after hospital admission, the patient reported the maximum assistance of 2 caregivers for sit-to-stand transfers. Once standing, she was able to ambulate 140 ft using a wheeled walker with contact guard assistance. Thus, the steps to the patient’s home were a barrier to discharging the patient directly home. Therefore, the patient was admitted to an inpatient acute rehabilitation service approximately 7 weeks after hospital admission. She gradually progressed to near antigravity pelvic girdle strength as the steroids were tapered. The patient improved from requiring the maximum assistance of 2 caregivers for sit-to-stand transfers. Once standing, she was able to ambulate 140 ft using a wheeled walker with contact guard assistance. Thus, the steps to the patient’s home were a barrier to discharging the patient directly home. Therefore, the patient was admitted to an inpatient acute rehabilitation service approximately 7 weeks after hospital admission. She gradually progressed to near antigravity pelvic girdle strength as the steroids were tapered. The patient improved from requiring the maximum assistance of 2 caregivers to requiring standby assistance with transfers, though the patient exerted considerable effort during transfers.

On admission to the acute rehabilitation service, routine laboratory test results revealed elevated liver function levels thought to be related to voriconazole, an antifungal agent heavily metabolized by the liver. These results lim-
similar weakness noted in hip abduction, adduction, and extension. All other knee, foot, and ankle motions demonstrated full 5/5 strength bilaterally. Passive range of motion was full and bilaterally symmetric in the upper and lower extremities. Muscle tone was normal.

Osteopathic structural examination revealed flat back posture with mild forward head and elevated left iliac crest. Palpation revealed symmetric muscular atrophy at the pelvic and shoulder girdles. No signs of effusion or ecchymosis were present. Focal tenderness and tissue texture changes were noted over the left PSIS that reproduced the patient’s chief musculoskeletal complaint. Standing and supine landmarks revealed relative left-sided asymmetries with an elevated iliac crest, superior and anterior PSIS, superior ischial tuberosity, and anterior and inferior anterior superior iliac spine. The standing flexion test revealed anterior rotation of the left PSIS greater than the right. With the ischial tuberosities stabilized, the seated flexion test revealed symmetric motion at the sacral sulcus and PSIS. Findings of the FABER (Flexion Abduction External Rotation) test were positive for left sacroiliac (SI) joint pain. Supine pelvic roll indicated a restriction of motion to the right and freedom of motion to the left. On the basis of the patient’s report of symptoms and the reproduction of pain and asymmetries noted on structural examination, the patient received a diagnosis of left anterior innominate sacroiliac dysfunction.

Osteopathic Manipulative Medicine: Evaluation and Treatment

On the day of new-onset pain symptoms, the patient presented to the osteopathic physician with an acute pain that worsened during transfers and standing and that substantially limited her ability to tolerate walking. Her symptoms improved with sitting and applying ice. She denied radicular symptoms, new weakness, prior similar symptoms, recent illness, fevers, chills, gastrointestinal changes, sensation changes, or pain at rest.

Physical examination revealed normal vital signs with normal cardiopulmonary and abdominal examination results. Skin and extremity examination results were normal other than baseline bilateral pretibial edema. There were no signs of upper motor neuron disease. Sensation testing showed diminished light touch sensation in a stocking-glove distribution consistent with a previously noted peripheral neuropathy. Neurologically, the patient was alert and oriented with no evidence of cognitive decline. Deep tendon reflexes were hypoactive and symmetric at 1+ in the upper and lower extremities. There were no signs of upper motor neuron disease. Manual muscle testing revealed that upper extremity shoulder abduction was bilaterally symmetric and diminished at 4/5. All other elbow, wrist, and hand motions demonstrated full 5/5 strength bilaterally. Lower extremity testing revealed that hip flexion strength was bilaterally symmetric and markedly diminished at 2/5 with similar weakness noted in hip abduction, adduction, and extension.

Considering the patient’s pelvic girdle weakness, passive techniques were selected to address her left anterior innominate sacroiliac dysfunction. Initially, the physician (D.J.K.) used an articulatory technique to induce a posterior rotatory torque on the left innominate by applying a downward force on the left anterior superior iliac spine by the cephalic hand and an upward force on the left ischial tuberosity by the caudal hand. This technique did not fully correct the SI asymmetry or resolve her symptoms. Next, a combined articulatory technique that addressed restrictions in pelvic rotation was used. The physician stood to the patient’s left, which was the
Pathogenesis and Epidemiology

The mechanism of steroid myopathy is unclear but is possibly related to the steroid’s influence on cellular receptors and intracellular signaling molecules. This influence may reduce protein synthesis, increase protein catabolism, and ultimately lead to muscle fiber atrophy. This influence may reduce protein synthesis, increase protein catabolism, alter carbohydrate metabolism, alter mitochondrial function, disturb electrolyte balance, or decrease sarcolemma excitability. Patient immobility alone has been shown to amplify the catabolic effects of steroids on skeletal muscle.

Prednisone dosages greater than 30 mg to 60 mg per day are associated with an increased risk of myopathy. Fluorinated steroids, such as dexamethasone or triamcinolone, hold a higher risk of toxic myopathy. Systemic steroids pose a greater risk of myopathy, whereas inhaled steroids and epidural steroid injections are rarely associated with myopathy. Proximal weakness can occur anytime within weeks to years after the initiation of higher dose steroid therapy. The incidence of steroid myopathy is unknown. Women, the elderly population, malnourished individuals, and patients with cancer may be at increased risk of steroid myopathy.

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Discussion and Review of the Literature

Few prior reports in the literature describe an OMM approach for patients with steroid myopathy. A keyword search in both medical and layperson Internet search engines revealed few articles in the literature related to this topic. Although there are a number of different OMM approaches to address somatic dysfunction associated with steroid myopathy, the hallmark features of this disease pose unique challenges for treating physicians.
ternate-day regimen has also been advocated.\textsuperscript{13,21} Switching from a fluorinated to a nonfluorinated steroid, such as prednisone, prednisolone, or hydrocortisone, should also be considered.\textsuperscript{15}

Rehabilitation has an important role in the treatment of patients with steroid myopathy to maximize their functional capacity. For slowly progressing myopathies, rehabilitation specialists recommend maintaining range of motion and a submaximal strengthening program.\textsuperscript{4}

Animal studies on myopathies have demonstrated that high-intensity strengthening may be detrimental.\textsuperscript{22} Resistant exercises should be limited to muscles with greater than antigravity strength.\textsuperscript{4} Skilled therapy should focus on functional activities such as self-care, pressure relief, dressing, transfers, balance, and gait. Consultation from a neurologist should be considered to explore the potential differential diagnosis of proximal weakness. A physiatrist can assist with the diagnosis and management of steroid myopathy through a rehabilitation program.

**Manipulative Medicine Considerations**

Steroid myopathy imposes many functional limitations upon the patient. The insidious onset of proximal muscle weakness limits self-care, bed mobility, balance, transfers, and gait. Proximal muscle weakness affects the structural integrity of the lumbo-pelvic complex and thus can have important implications for osteopathic physicians and physical therapists. Collectively, the gluteal muscles, quadratus femoris muscle, and iliopsoas muscle have been referred to as the “rotator cuff of the hip.”\textsuperscript{6(p603)} The contributions from the proximal muscles and related fascia to the stability of the pelvis and sacrum cannot be understated.

Osteopathic physicians should individualize physical examinations to meet the needs and capabilities of patients. For patients with steroid myopathy, the physical examination must be adapted to account for pelvic girdle weakness. Although passive static landmark tests should not be altered, the dynamic tests may need to be adapted or excluded. In the present case, the standing and seated
pain medication options. The patient’s left SI pain was the limiting factor in her ability to progress through her rehabilitation program. Fortunately, careful selection of OMM techniques provided a nonpharmacologic approach that allowed her to manage her symptoms, tolerate therapies, and continue to regain her function.

**Prognosis**

With proper management, a complete resolution from steroid myopathy is likely; however, residual weakness and atrophy is possible. Patients typically begin to regain strength 1 to 4 months after reducing or discontinuing steroid use.

**Conclusion**

Steroid myopathy imposes functional limitations on patients and unique challenges for health care providers. The proximal weakness seen with steroid myopathy necessitates special considerations for an OMM approach to address somatic dysfunctions associated with this condition. The present case further demonstrates how creative adaptation of osteopathic examination and treatment techniques can benefit patients with complex medical conditions. Further research is needed on case reports to explore the efficacy of OMM.

**References**


