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

David J. Kohns, DO
David S. Fitch, DO

From the Department of Physical Medicine and Rehabilitation at the University of Michigan in Ann Arbor.

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Address correspondence to David J. Kohns, DO, Department of Physical Medicine and Rehabilitation, University of Michigan, 325 E Eisenhower Pkwy SPC 5744, Ann Arbor, MI 48108-3364.

E-mail: dkohns@med.umich.edu

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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.[1,2] In 1958, 10 years after the introduction of steroid therapy, Dubois[3] 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.[1] 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.[4]

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.

Report of Case

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 occured 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 required 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-

ited the selection of pain medications, such as acetaminophen, that have been linked to liver failure.[5] The antifungal agent was changed to posaconazole, and the patient's liver function test results eventually normalized.

Two weeks into the patient's acute rehabilitation stay (approximately 9 weeks after admission to the hospital), the patient reported to her physical therapist that she had new-onset pain localized over the left posterior superior iliac spine (PSIS), which occurred when she was in therapy working on transfers. She was referred to an osteopathic physician (D.J.K.) for evaluation.

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. 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.[6(pp762-783),7,8]

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.[7] 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

side of pelvic rotational freedom. The patient was in a supine position with the hips and knees held in adduction and flexion at 90°. The physician then slightly rotated the patient's pelvis by bringing both her knees and hips to her left. This combination put the pelvis in an indirect position of freedom. The physician then applied a gentle axial force through the knees toward the patient's waist. In 1 motion, while attempting to maintain this axial force, the physician passively brought the knees across the patient's body to induce a direct right pelvic rotation. Next, both lower extremities were returned to the mat and the spine to a neutral position. After this technique, the patient had full correction of her SI dysfunction with marked resolution of her left PSIS symptoms. The patient was then educated regarding the anatomy of her SI dysfunction and activities that could potentially aggravate her condition. An SI belt was used to help prevent future somatic dysfunction.

With her SI symptoms well controlled, the patient was able to continue to progress with her rehabilitation program. After approximately 4.5 weeks of receiving acute inpatient rehabilitation services, she was able to transfer independently, ambulate greater than 200 ft with a wheeled walker, ascend 4-in steps with a rail, and perform all activities without limitations from SI pain. The patient was discharged home. She regained antigravity strength in her pelvic girdle 11 weeks after her symptoms of steroid myopathy began.

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.

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.[9,10] This influence may reduce protein synthesis, increase protein catabolism, and ultimately lead to muscle fiber atrophy.[11] This influence may reduce protein synthesis, increase protein catabolism, alter carbohydrate metabolism, alter mitochondrial function, disturb electrolyte balance, or decrease sarcolemma excitability.[11] Patient immobility alone has been shown to amplify the catabolic effects of steroids on skeletal muscle.[12]

Prednisone dosages greater than 30 mg to 60 mg per day are associated with an increased risk of myopathy.[13,14] Fluorinated steroids, such as dexamethasone or triamcinolone, hold a higher risk of toxic myopathy.[15] Systemic steroids pose a greater risk of myopathy, whereas inhaled steroids and epidural steroid injections are rarely associated with myopathy.[16] Proximal weakness can occur anytime within weeks to years after the initiation of higher dose steroid therapy.[4,11] The incidence of steroid myopathy is unknown, but it has been reported in up to 60% of patients with cancer receiving steroid treatement.[11] Elderly patients, physically inactive patients, patients with weak respiratory muscles, patients with cancer, and patients with a negative nitrogen balance before starting glucocorticoid treatment may be at increased risk of steroid myopathy.[11,17]

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Diagnosis

Steroid myopathy is a diagnosis of exclusion. A comprehensive medical history is essential to guide the differential diagnosis that may include a myopathy. Critical information to aid in the diagnosis of steroid myopathy

includes distribution of weakness (symmetric and proximal), timing of symptoms (typically chronic and progressive), functional capabilities (difficulty with stairs and rising from a chair), normal sensation, patient age (often elderly), past medical history, and medications (steroid use). The differential diagnosis may include other forms of myopathy (toxic, inflammatory, endocrine, systemic), electrolyte disturbances, neuromuscular junction diseases, and infection.[4(pp1097-1124)]

Physical examinations for steroid myopathy would likely reveal proximal muscle weakness (pelvic girdle muscles involved early), normal muscle bulk (muscle atrophy can occur later, such as in the patient in the present case), and normal deep tendon reflexes and sensation. Respiratory muscle function is rare but may occur in steroid myopathy.[18]

No definitive diagnostic tests exist for steroid myopathy. Diagnostic laboratory studies of patients with steroid myopathy generally show normal or reduced creatine kinase levels.[4(p1123)] Muscle biopsy specimens typically reveal atrophy of type 2 greater than type 1 fibers thought to be related to reduced protein synthesis rather than an increased catabolic effect.[19] Electromyography (EMG) and nerve conduction studies (NCS) in patients with chronic steroid myopathy typically demonstrate normal motor and sensory NCS results and normal needle examination findings. Needle EMG evaluates type 1 muscle fibers, whereas steroid myopathy mainly affects type 2 muscle fibers. Unless the myopathy is severe, the needle EMG findings typically reveal normal insertional activity but can be helpful to differentiate steroid myopathy from polymyositis, which usually shows increased insertional activity because it is an inflammatory myopathy that can cause denervation.[20]

Treatment

The main treatment recommendation for patients with steroid myopathy is to decrease the dose of steroid below a threshold level or gradually taper it off completely. Reducing the dosage below 30 mg daily or using an alternate-day regimen has also been advocated.[13,21] Switching from a fluorinated to a nonfluorinated steroid, such as prednisone, prednisolone, or hydrocortisone, should also be considered.[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.[4] Animal studies on myopathies have demonstrated that high-intensity strengthening may be detrimental.[22] Resistance exercises should be limited to muscles with greater than antigravity strength.[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."[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

flexion tests were modified with the patient's upper extremities partially supported by a walker. The standing stork test, which is commonly used to assess SI dysfunction, could not be performed because the patient had less than antigravity hip flexor strength.

Similar to the physical examination, treatment techniques must also be adapted for patients with pelvic girdle weakness from steroid myopathy. Osteopathic physicians typically seek the least invasive technique to achieve the desired effect. The key feature to consider in the present case is that the patient was a 59-year-old woman who had been given chronic steroids and had developed proximal pelvic girdle weakness from steroid myopathy. First, because of the patient's sex, age, and chronic steroid use, she was at risk for osteoporosis. Therefore, addressing the SI dysfunction with more aggressive techniques such as high-velocity, low-amplitude manipulations and leg tugs were contraindicated.[7] Second, the patient had less than antigravity strength in her hip girdle muscles. The effectiveness of muscle energy techniques, which would have required the patient to form a full active contraction of the pelvic girdle muscles would have been limited.[7] Passive articulatory techniques were successful and well tolerated by this patient. Caution should be used against being overly aggressive with these articulatory techniques, as patients with proximal weakness do not have the ability to protectively splint their muscles. In addition, counterstrain or other soft tissue techniques should be considered.[6(pp1002-1016),7,8] An SI belt can also be used as an adjunct to help stabilize the pelvis.[23] Finally, the role of education is crucial for patients with steroid myopathy, who will need to modify their activities for weakness and instability about the pelvic girdle.

The present case further demonstrates the value of an osteopathic approach to the treatment of patients with complex medical conditions. The onset of the patient's SI symptoms coincided with an elevation of her liver enzyme associated with the use of prophylactic antifungal medication. These laboratory findings imposed limitations on 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.[13]

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.

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