
CASE REPORTS

THORACOLUMBAR COMPRESSION FRACTURES PRESENTING WITH AN ACUTE ILEUS

Edward L. Treadwell, MD, Paul R. Cunningham, MD, Henry M. Kowalski, MD, and Michael D. Weaver, MD
Greenville, North Carolina

Corticosteroids are commonly used in the treatment of connective tissue diseases such as systemic lupus erythematosus. Although they are usually efficacious, osteoporosis leading to spine compression fractures is not uncommon. In this case report, we describe an elderly patient with systemic lupus erythematosus on long-term corticosteroid therapy who presented with symptoms of acute abdomen with minimal low back symptoms. No intraabdominal process was found by abdominal studies and exploratory laparotomy. Increased lower back symptoms led to further skeletal spine studies, which initially demonstrated a compression fracture at the twelfth thoracic (T12) vertebra. Later, a T8 and a fourth lumbar (L4) compression fracture were also found. Her abdominal and lower back symptoms resolved on conservative therapy. Although the rate of these occurrences are unknown, compression spine fractures should be considered in elderly patients presenting with acute abdomen after being on long-term corticosteroid therapy.

Key words • systemic lupus erythematosus • corticosteroid therapy • acute abdomen • thoracolumbar compression fractures

Thoracic or lumbar fractures are not uncommon in elderly patients, especially patients treated with long-term corticosteroid therapy. A diagnosis can usually be readily suspected by an acute onset of upper or lower back pain, especially upon performance of activities that place excessive strain on the thoracic or lumbosacral spine. However, the presentation can be atypical and present a diagnostic challenge to the physician.

In this report, we describe a patient who had been on long-term corticosteroid therapy for systemic lupus erythematosus and presented with predominant symptoms of an acute abdomen.

CASE REPORT

The patient, a 66-year-old retired, elderly, black female was admitted to the Pitt County Memorial Hospital with a 1-week history of increasing crampy abdominal pain, superimposed on an "abdominal ache." She noted that the pain would radiate down to her midspine, a symptom which she described as a spasm. The pain was associated with mild anorexia and constipation but no nausea, vomiting, fever, chills, dysuria, or other symptoms. She had been taking laxatives and other home remedies for relief of her abdominal symptoms and "constipation" without

From the East Carolina University School of Medicine and Pitt County Memorial Hospital, Greenville, North Carolina. Reprint requests should be addressed to Dr Edward L Treadwell, Section of Rheumatology/Immunology, Department of Medicine, East Carolina University, Greenville, NC 27858-4354.

success. On a repeat review of her history, she did admit to falling 3 days prior to the onset of her symptoms, after which she noticed some very mild discomfort in her lower back. Because of the increasing severity of her abdominal symptoms, she was brought to the emergency room for further evaluation.

She had a 7-year history of intermittent symmetrical pain and swelling in many joints, including her shoulders, elbows, wrists, hands (metacarpophalangeal joints), ankles, and feet (metatarsophalangeal joints). At the onset of her symptoms, she was diagnosed with a "collagen vascular disease" by her physician and had been maintained on various doses of ibuprofen and an average dose of 5 mg daily of prednisone. In March 1988, she had her first admission to Pitt County Memorial Hospital for acute, right-sided "pleuritis" and a mildly inflammatory pleural effusion consisting of a total white blood count of 5560 cells/mm³ (predominantly mononuclear), with a differential of 95% monocytes and 5% polymorphonuclear cells. Stains and cultures for bacteria, acid fast, and fungal organisms were all negative. The patient was treated with 40 mg of prednisone daily, with rapid improvement and abatement of her symptoms. Her prednisone was decreased to 10 mg/day along with 375 mg twice daily of naproxen, which she continued until her most recent admission. Other medications included 25 mg of daily hydrochlorothiazide for long-standing hypertension and 0.1 µg of levothyroxine following a partial thyroidectomy in 1957. In 1983, she had surgical removal of right renal calculus.

Other pertinent rheumatic symptoms included a more than 10-year history of dry eyes and dry mouth, possible Raynaud's phenomenon, consisting of mild blanching and numbness of her fingertips on exposure to cool environments, and numerous tender trigger points at various muscle insertions.

Physical examination on admission for abdominal pain revealed an elderly female in moderate to severe distress with a temperature of 98.2°, blood pressure of 120/60, respirations of 32, and a pulse rate of 96 and regular. She was alert and oriented. She had diffuse abdominal distention with no audible bowel sounds by auscultation. There was positive rebound tenderness, especially at the right upper quadrant. She had mild point tenderness bilaterally at her midback. The white blood count was moderately elevated at 12 200/mm³ with a differential of 64 polymorphonuclear cells, 14 band cells, and 11 lymphocytes. Hemoglobin was 12.0 g/dL, and a Westergren sedimentation rate was elevated at 94 mm/hr. Electrolytes, chemistries,

urinalysis, and thyroid studies were all normal. Antinuclear antibody studies showed an indirect immunofluorescent titer of 1:10 000; speckled pattern on Hep-2 cells.¹ Antibodies to Sm, RNP, SS-A, and SS-B by double immunodiffusion were positive.¹ A rheumatoid factor by latex agglutination was less than 1:10, and an anti-nDNA by the *Crithidia luciliae* indirect immunofluorescent method was negative. Acute abdominal roentgenographic films showed distended loops of her distal colon. An ultrasound of her abdomen was normal with no evidence of cholelithiasis. Her hepatobiliary ductal system was not dilated, and her pancreas was believed to be normal.

The day following admission, the patient's abdominal pain continued to increase along with peritoneal irritation. An exploratory laparotomy was performed to exclude a perforated viscus. No perforation, infection, or other processes were found. An omentum biopsy revealed no evidence of vasculitis. Postoperatively, her corticosteroid therapy, which had been increased to 80 mg/day of intravenous prednisolone on admission, was slowly decreased, and her symptoms slowly abated with resolution of her abdominal pain. Her bowel sounds returned, and she remained afebrile following her surgery. Her white blood count decreased to within the normal range. Her predominant symptom was increasing midback discomfort. Radiographs of her thoracolumbar spine revealed a possible compression fracture of her T12 vertebra (Figure 1A). Early osteophytes were noted at the T5 to T9 vertebrae. A dual phase technetium 99m methylene diphosphonate bone scan showed increased uptake specifically localized at the T12 area, consistent with an acute or subacute compression fracture, possibly another inflammatory process (Figure 1B). A magnetic resonance imaging scan was performed and was consistent with a T12 compression fracture (Figure 1C).

Her prednisone was tapered to 10 mg daily, and she continued other preadmission medications with an occasional analgesic. She was discharged 10 days postoperatively. Two months later, her abdominal symptoms had resolved, and she had only residual mild back discomfort. Repeat radiographs of her thoracolumbar spine demonstrated further compression of the T12 vertebra and new compression fractures at T8 (Figure 2) and L4 (not shown). Four months later, she had no abdominal or back pain.

DISCUSSION

Gastrointestinal involvement in patients with

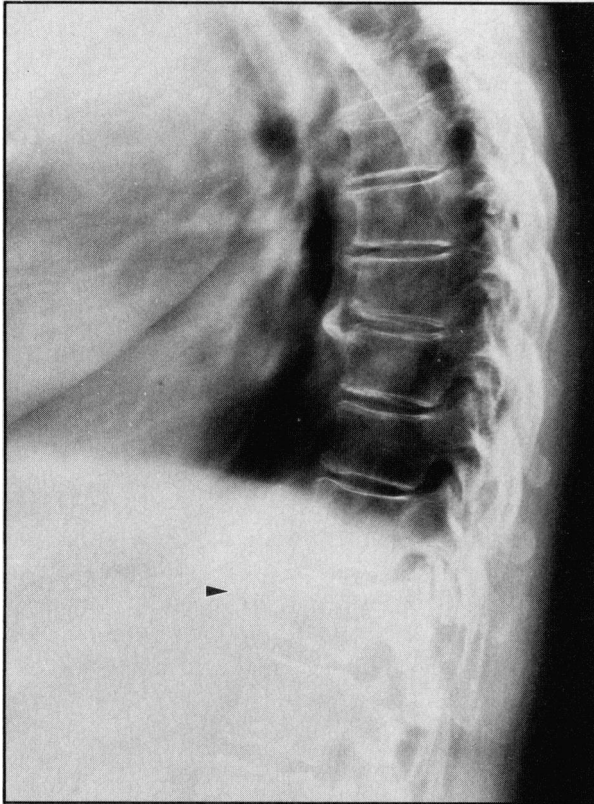


Figure 1A. Radiographic views of the thoracolumbar spine demonstrates an early compression fracture at the T12 vertebral level (arrow). Early osteophytes are also seen at T5-T9 vertebrae.

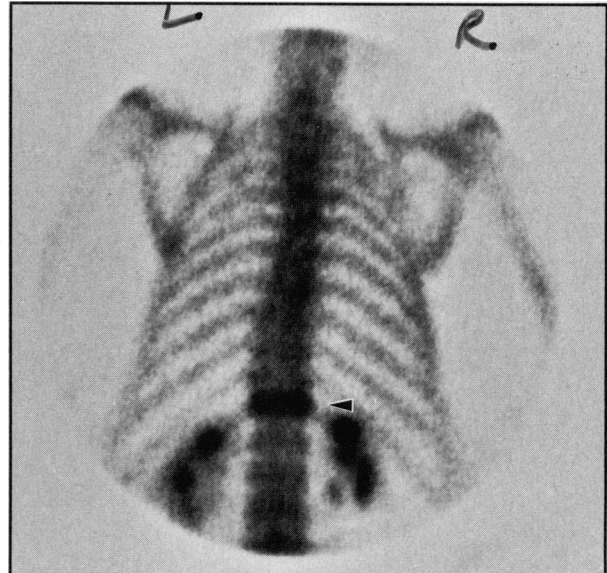


Figure 1B. A dual phase technetium 99m methylene diphosphonate bone scan. Increased uptake is noted at the T12 vertebra (arrow).



Figure 1C. A magnetic resonance imaging scan of the thoracolumbar spine. Partial compression of the T12 vertebra is shown as demonstrated in Figures 1A and 1B.

systemic lupus erythematosus is common, although it often goes unrecognized.^{2,3} Manifestations include peritonitis, ascites, pancreatitis, gastritis, hepatomegaly, hepatitis, and vasculitis involving any of the gastrointestinal segments. Vasculitis of the small or large bowel has the potential for the highest morbidity and mortality. Findings may range from segmental edema to discrete ulceration, gangrene, and finally perforation. These patients may present insidiously or with abdominal distention with an acute abdomen, especially when a perforated viscus is present. Common symptoms include abdominal pain, nausea, vomiting, diarrhea, bleeding, esophagitis, and dysphagia.

Common therapeutic agents used in the treatment of systemic lupus erythematosus may simulate problems caused by the disease, making evaluation of these patients difficult. Aspirin or nonsteroidal, anti-inflammatory agents may cause esophagitis, gastritis,

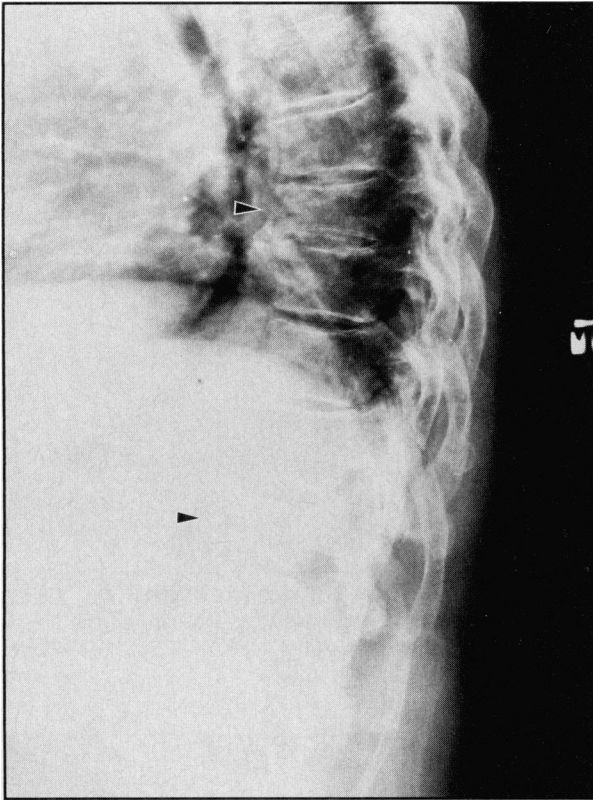


Figure 2. Repeat radiographic views of the thoracolumbar spine demonstrates further compression of the T12 vertebra (lower arrow) and a new compression at T8 (upper arrow).

ulceration, and bleeding. Corticosteroids may cause pancreatitis, peptic ulcer disease, diverticular perforation, and a fatty liver. Pancreatitis or liver enzyme abnormalities may also be caused by azathioprine or thiazides. Our patient presented with an acute abdomen in the form of an adynamic ileus. Because the initial abdominal studies were negative for a specific etiology, a laparotomy was performed to exclude an intestinal perforation or obstruction, especially from vasculitis. As no specific active pathology was found, the patient's compression fractures were implicated as a cause of clinical presentation.

Acute pelvic or other fractures of the lumbar spine have been recognized as the cause of an adynamic ileus.⁴⁻⁷ From our literature review, specific reference to compression fractures of the skeletal spine in

association with an acute ileus was not noted. We postulate that our patient had a neurogenic ileus resulting from her vertebral compression fractures which presented as a "retroperitoneal syndrome." To define this association, more in-depth study of a larger number of patients with compression fractures will be needed. The magnetic resonance imaging scan was useful in confirming the diagnosis of a compression fracture as well as excluding other possible causes of lower back pain. The usefulness of this procedure in evaluating various disorders of the thoracic, lumbar, and sacral spine has been previously reported, and magnetic resonance imaging is increasingly being employed for diagnostic evaluation of lower back syndromes.⁸

Compression fractures, especially in the elderly and patients on long-term corticosteroid therapy, should be considered in the differential diagnosis of patients presenting with an acute abdomen and any magnitude of lower back pain. Rapid diagnosis of a compression fracture may prevent the need for surgical intervention.

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