

Ankylosed spines are prone to fracture

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SUMMARY

Fracture of an ankylosed spine is often overlooked. Because the force that damages an ankylosed spine is frequently slight, patients do not realize they are injured. Doctors can miss the fracture for the same reason and because patients already have a history of back pain. Plain radiographs sometimes fail to demonstrate the fracture site.

RÉSUMÉ

Chez le patient atteint d'ankylose de la colonne, la fracture d'une vertèbre passe souvent inaperçue. Puisqu'un traumatisme mineur peut fréquemment causer des dommages à une colonne ankylosée, les patients ne réalisent pas l'importance de leur blessure. Pour la même raison, les médecins peuvent manquer la fracture d'autant plus que ces patients ont déjà des antécédents de douleurs à la colonne. Les radiographies simples sont parfois incapables de mettre en évidence le site de la fracture.

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THE ANKYLOSED SPINE IS MORE prone to fracture than normal spines. This phenomenon has been reported in both ankylosing spondylitis and diffuse idiopathic skeletal hyperostosis.¹⁻⁷ Fractures are more common in patients with ankylosing spondylitis because of associated spinal osteoporosis.¹

The force causing fracture of the ankylosed spine is often minor, and patients might be unaware that it seriously damaged the spine. If patients do visit physicians, the complication of spinal fracture could be overlooked because of the minor nature of the trauma and the patients' history of spinal pain. If the diagnosis is considered, plain radiographs of the spine might fail to demonstrate the fracture site.^{1,2} The following case report illustrates these problems.

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Case report

A 43-year-old man with a 20-year history of ankylosing spondylitis had radiographic evidence of ankylosis of the entire spine. He slipped and fell in his kitchen; the next morning, he had no pain at rest, but on movement he had midthoracic pain. He was brought to the emergency department by ambulance and handled as if he had a spinal fracture. Examination showed no tenderness of the spine, and results of the neurological examination were normal.

Plain radiographs of the thoracic spine showed no definite abnormality (Figure 1). However, a radionuclide bone scan showed increased radionuclide uptake at the T8-9 level (Figure 2). Tomography at the T8-9 level demonstrated a fracture (Figure 3). Radiographs of the cervical spine, when compared with radiographs done 6 months previously, showed a fracture through the ossified anterior longitudinal ligament at the C5-6 level (Figure 4). No corresponding abnormality was seen on the radionuclide bone scan. The patient was treated conservatively for spinal fractures of the cervical and thoracic region, and at a 6-month follow-up examination, the fractures had healed with no neurological complication.

Figure 1. Plain radiographs of the thoracic spine: A) Anteroposterior view, B) lateral view.

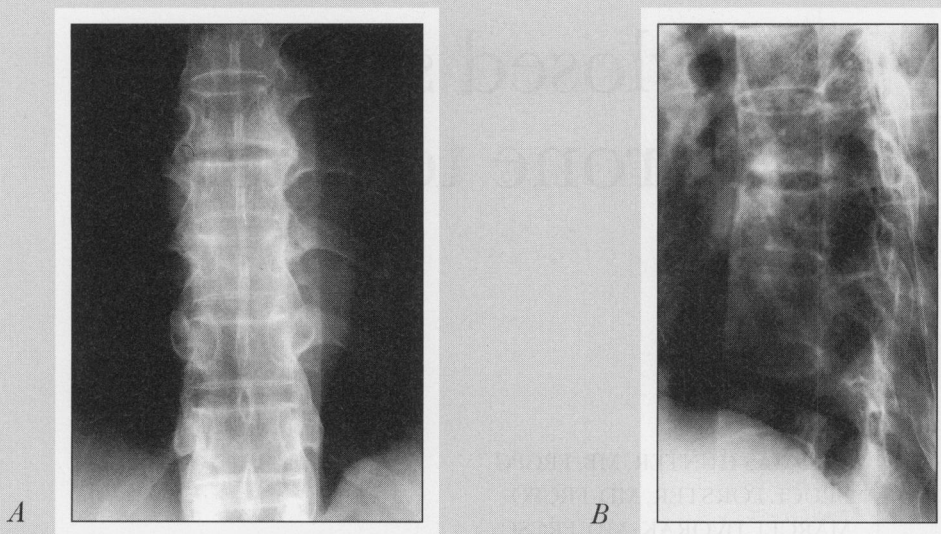


Figure 2. Radionuclide bone scan showing increased uptake of radionuclide at the T8-9 level

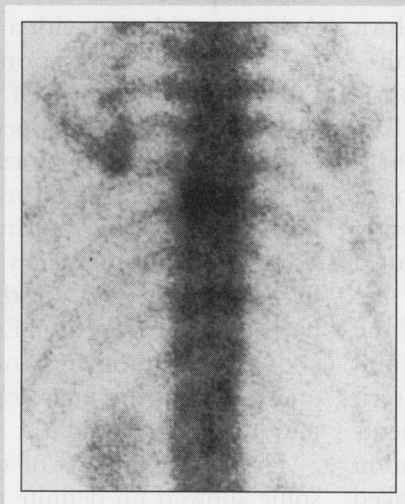


Figure 3. Tomography of the thoracic spine showing a fracture at the T8-9 level

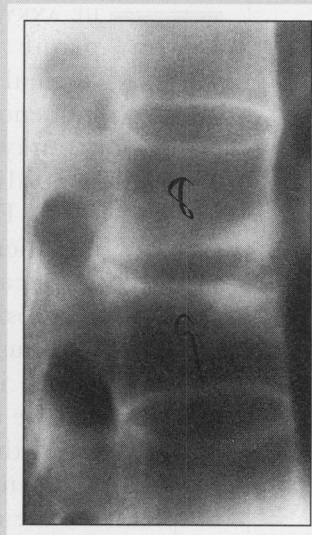
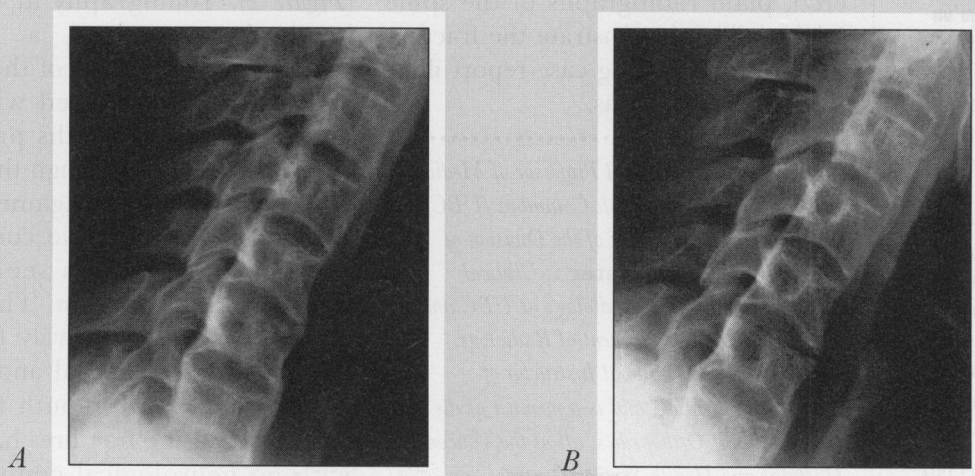


Figure 4. Plain lateral radiographs of the cervical spine: A) Radiographs were taken 6 months before the fall and B) the day following the fall, showing a fracture through the ossified anterior longitudinal ligament at the C5-6 level.



Discussion

This case illustrates many of the clinical features associated with spinal fractures in patients with ankylosing spondylitis. First, most patients have a history of chronic spinal pain followed by a pain-free period once the bony ankylosis has occurred. The redevelopment of mechanical spinal pain, precipitated or aggravated by activity and relieved by rest, should suggest a spinal fracture. In addition, patients might notice a change in posture or a change in their field of view. The fracture might occur at more than one level. If the fracture occurs in the thoracic region, patients must be watched closely, as laceration of the aorta could occur.^{8,9}

The fracture might be difficult to see on plain radiographs, especially at the cervicothoracic junction, which could be the most common site of fracture in patients with ankylosing spondylitis.^{1,2} Therefore, if the diagnosis is considered, patients should be managed as if they have a spinal fracture until other studies can be completed.

Radionuclide bone scanning is now available in many smaller hospitals and could help to locate the spinal fracture, which can then be confirmed by radiographic tomography (*Table 1*).²

Computed tomography (CT) is becoming increasingly available. As the fracture is often in the transverse plane and undisplaced, CT performed axially might miss the fracture depending on the slice thickness. Reformating the data into the sagittal plane might make such a fracture apparent.^{10,11} However, plain film tomography, if available, might be just as useful.

Magnetic resonance imaging can also detect a radiographically occult spinal fracture, because of the associated bone marrow edema.¹¹ The fracture itself, however, is probably more visible with CT or tomography. Magnetic resonance imaging might also be helpful in diagnosing an epidural hematoma, which is more common in spinal fractures in patients with ankylosing spondylitis.¹² ■

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References

1. Hunter T, Dubo H. Spinal fractures complicating ankylosing spondylitis. *Ann Intern Med* 1978;88:546-9.
2. Hunter T, Dubo HIC. Spinal fractures complicating ankylosing spondylitis. A longterm follow-up study. *Arthritis Rheum* 1983;26:751-9.
3. Hunter T. The spinal complications of ankylosing spondylitis. *Semin Arthritis Rheum* 1989; 19:172-82.
4. Fardon DF. Odontoid fracture complicating ankylosing hyperostosis of the spine. *Spine* 1978;3:108-12.
5. Houk RW, Hendrix RW, Lee C, Lal S, Schmid FR. Cervical fracture and paraplegia complicating diffuse idiopathic skeletal hyperostosis. *Arthritis Rheum* 1984;27:472-5.
6. Corke CF. Spinal fracture and paraplegia after minimal trauma in a patient with ankylosing vertebral hyperostosis. *BMJ* 1981; 282:2035.

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Table 1. How to diagnose a fracture in an ankylosed spine

Step 1. Suspect the diagnosis if there are:

- a history of direct or indirect trauma to the spine of even a mild degree (usually a simple fall),
- complaints of mechanical spinal pain precipitated or aggravated by activity and completely or partially relieved by rest, or
- a change in posture, including improved posture.

Step 2. Once the diagnosis is suspected, manage the patient as having an unstable spinal fracture until the diagnosis has been excluded. (Suspect that there might be more than one fracture site.)

Step 3. Perform radiographic examination of the spine.

Step 4. If the radiographs suggest a spinal fracture, confirm with computed tomography.

Step 5. If findings from radiographs are negative and if it is unclear which areas should be submitted to tomography, then a bone scan might help identify the fracture site.

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INDICATIONS AND CLINICAL USE

As an adjunct to diet for the reduction of elevated total and LDL-C levels in patients with primary hypercholesterolemia; also in combined hypercholesterolemia and hypertriglyceridemia, when hypercholesterolemia is the abnormality of most concern.

To determine which patients to treat, initially establish that the elevation in plasma lipids is not due to underlying conditions such as poorly-controlled diabetes mellitus, hypothyroidism, the nephrotic syndrome, liver disease, or dysproteinemias. Then ascertain whether elevated LDL-C level is the cause for elevated total serum cholesterol, particularly in patients with total triglycerides over 4.52 mmol/L (400 mg/dL) or with markedly elevated HDL-C values, where non-LDL lipoprotein fractions may contribute significantly to total cholesterol levels, without apparent increase in cardiovascular risk.

CONTRAINDICATIONS

Hypersensitivity to any component. Active liver disease or unexplained persistent elevations of serum transaminases. Pregnancy and lactation (see PRECAUTIONS).

WARNINGS

The effects of simvastatin-induced changes in lipoprotein levels, including reduction of serum cholesterol, on cardiovascular morbidity or mortality have not been established.

1. Hepatic effects: In clinical trials, marked persistent increases in serum transaminases occurred in 1% of adult patients who received simvastatin (see ADVERSE REACTIONS). Increases were not associated with jaundice or other clinical signs or symptoms. There was no evidence of hypersensitivity. Serum transaminases fell slowly to pre-treatment levels when drug was interrupted or discontinued.

All patients should have liver function tests at baseline and periodically thereafter. Patients who develop elevated serum transaminase levels require special attention, prompt retesting and more frequent testing.

Discontinue drug if transaminase levels show evidence of progression, particularly a rise to 3 times the upper limit of normal that persists.

Use with caution in patients who consume substantial quantities of alcohol and/or have a history of liver disease. Discontinue drug if active liver disease or unexplained persistent transaminase elevations develop during therapy (see CONTRAINDICATIONS).

Moderate elevations of serum transaminases, reported with simvastatin, have also been observed with other, comparative lipid-lowering agents. These changes generally appeared within the first 3 months after initiation of therapy, were often transient, not accompanied by any symptom, and did not need interruption of treatment.

2. Muscle Effects - CPK: Transient elevation of creatine phosphokinase (CPK) levels commonly seen, usually have no clinical significance. - **Myalgia** and muscle cramps have also been observed. - **Myopathy** reported rarely (0.05%); consider possibility in any patient with diffuse myalgias, muscle tenderness and/or marked elevation of creatine phosphokinase (≥ 10 times the upper limit of normal). Ask patients to promptly report unexplained muscle pain, tenderness and weakness. With *lovastatin*, a closely related HMG-CoA reductase inhibitor, the risk of myopathy is known to be substantially increased by concomitant immunosuppressive drugs including cyclosporin, or gemfibrozil or lipid-lowering doses of niacin. Severe rhabdomyolysis that precipitated acute renal failure was reported. Also, rhabdomyolysis with or without renal impairment was reported in seriously ill patients receiving concomitant erythromycin and lovastatin.

Therefore, carefully consider benefits and risks of concomitant use of simvastatin with immunosuppressive drugs, fibrates, erythromycin or lipid-lowering doses of niacin. Consider interrupting simvastatin in any patient with an acute, serious condition, suggestive of a myopathy or a risk factor predisposing to development of renal failure or rhabdomyolysis, such as: severe acute infection, hypotension, major surgery, trauma, severe metabolic, endocrine or electrolyte disorders and uncontrolled seizures.

PRECAUTIONS

General: Before starting therapy, attempt to control hypercholesterolemia with appropriate diet, exercise, weight reduction in overweight and obese patients, and to treat underlying medical problems (see INDICATIONS). The patient should inform subsequent physicians of prior use of simvastatin.

Ophthalmic evaluations: Current data do not indicate adverse effects on the human lens, but long-term effects have not been established. Periodic ophthalmological exams are recommended, keeping in mind that even without drugs, an increased prevalence in lens opacities could be expected with aging. **Use in homozygous familial hypercholesterolemia:** simvastatin is unlikely to be of clinical benefit. **Effect on Lipoprotein(a) [Lp(a)]:** In some patients, the beneficial lowering of total and LDL cholesterol may be partly blunted by increased Lp(a) levels. Pending further experience, Lp(a) plasma levels should be measured when feasible in patients given simvastatin. **Hypersensitivity:** A few instances of eosinophilia and skin eruptions appear to be associated with simvastatin. If hypersensitivity suspected, discontinue drug. **Carcinogenesis:** In animal studies, increased incidences of hepatocellular adenomas and carcinomas, pulmonary adenomas and harderian gland adenomas were noticed in mice receiving 500 times the maximum recommended human dose. Female rats receiving 31 times the maximum recommended human dose exhibited an increased incidence of thyroid follicular adenomas. (See TOXICOLOGY Section of Product Monograph.)

Use in obstetrics: Simvastatin is contraindicated during pregnancy and there are no data on such use. Because the HMG-CoA reductase inhibitors are able to decrease the synthesis of cholesterol and possibly other products of the cholesterol biosynthesis pathway that are essential components for fetal development, simvastatin may cause fetal harm. Administer to women of childbearing age only when they are highly unlikely to conceive. If a patient becomes pregnant, apprise her of potential hazard to the fetus, and discontinue drug. **Nursing mothers:** Whether simvastatin is excreted in human milk is unknown. However, because of the potential for serious adverse reactions, women taking simvastatin should not nurse (see CONTRAINDICATIONS). **Pediatric use:** Safety and effectiveness have not been established; therefore simvastatin therapy in children is not yet recommended. **Use in patients with impaired renal function:** Exercise caution if renal function impairment is significant.

Drug Interactions

Concomitant therapy with other lipid-lowering agents: Cholesterol-lowering effects of simvastatin and cholestyramine appear additive. Exercise caution when coadministering with other lipid-lowering agents, particularly gemfibrozil and niacin (see WARNINGS). **Erythromycin:** See WARNINGS: Muscle effects. **ACE Inhibitors:** Hyperkalemia associated with myositis was reported in a single patient with insulin-dependent diabetes mellitus and mild renal insufficiency who received another HMG-CoA reductase inhibitor: lovastatin with an ACE inhibitor, lisinopril. **Coumarin anticoagulants:** Determine prothrombin time in patients on concomitant coumarin anticoagulants before starting simvastatin therapy and monitor periodically, because anticoagulant effect of warfarin appeared to be slightly enhanced by simvastatin use. **Digoxin:** Digoxin plasma concentrations were slightly elevated by coadministration of simvastatin. **Propranolol:** No clinically significant pharmacokinetic or pharmacodynamic interaction noted with concomitant simvastatin. **Antipyrene:** Simvastatin had little or no effect on the pharmacokinetics of antipyrene. **Other concomitant therapy:** Exercise caution with coadministration of immunosuppressants (see WARNINGS). In clinical studies, simvastatin was used with beta-blockers, calcium-channel blockers, diuretics and NSAIDs, without evidence of clinically significant adverse interactions.

Drug/laboratory test interactions: Simvastatin may elevate serum transaminase and creatine phosphokinase levels (see ADVERSE REACTIONS). In differential diagnosis of chest pain in patients on simvastatin, determine cardiac and non-cardiac fractions of these enzymes.

ADVERSE REACTIONS

Simvastatin was found generally well tolerated, and adverse reactions usually mild and transient, based on experience in over 2300 patients, of whom over 1200 were treated for 1 year and over 230 for 2 years or more. In controlled clinical trials, 1% were withdrawn due to adverse experiences attributable to simvastatin. Adverse experiences occurring at an incidence of $\geq 0.5\%$ of 2361 patients treated with simvastatin in controlled clinical studies and reported to be possibly, probably or definitely drug related are shown in the table below:

	ZOCOR® (n = 2361) %
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Headache	1.0
Skin	
Rash	0.7
Miscellaneous	
Abdominal Pain	2.2
Asthenia	0.8

Ophthalmological Observations: see PRECAUTIONS.

Laboratory tests: Marked persistent increases of serum transaminases noted (see WARNINGS). About 5% of patients had elevations of CPK levels of at least three times normal value, attributable to the non-cardiac fraction of CPK, on one or more occasions. Myopathy reported rarely (see WARNINGS and PRECAUTIONS).

Others: Though not observed in clinical trials with simvastatin, the following have been reported with other HMG-CoA reductase inhibitors: hepatitis, cholestatic jaundice, vomiting, anorexia, paresthesia, psychic disturbances including anxiety, and hypospermia. Also reported rarely with lovastatin was a hypersensitivity syndrome which included one or more of the following: anaphylaxis, angioedema, lupus-like syndrome, polymyalgia rheumatica, thrombocytopenia, leukopenia, hemolytic anemia, positive ANA, ESR increase, arthritis, arthralgia, urticaria, asthenia, photosensitivity, fever and malaise.

SYMPTOMS AND TREATMENT OF OVERDOSAGE

No experience of deliberate or accidental overdosage. Treatment should be symptomatic and supportive. Liver function should be monitored, and appropriate therapy instituted. Dialyzability of simvastatin not known.

DOSE AND ADMINISTRATION

Before initiating simvastatin, place patient on standard cholesterol-lowering diet, and continue on this diet during treatment. If appropriate, implement a program of weight control and exercise. **Usual starting dose:** 10 mg/day, as a single dose in the evening. Make dosage adjustments, if necessary, at intervals of not less than 4 weeks, to maximum of 40 mg daily, given as a single evening dose. **Monitor cholesterol levels periodically and consider reducing dosage if cholesterol levels fall below targeted range, as recommended by the Canadian Consensus Conference on Cholesterol.**

Concomitant therapy: Cholesterol-lowering effects of simvastatin and cholestyramine appear additive. For use with other lipid-lowering agents, see WARNINGS and PRECAUTIONS.

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References:

1. The Scandinavian Simvastatin Survival Study Group. Randomized trial of cholesterol lowering in 4444 patients with coronary heart disease: the Scandinavian Simvastatin Survival Study (4S). *Lancet* 1994;344:1363-69.

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7. Mody GM, Charles RW, Ranchod HA, Rubin DL. Cervical spine fracture in diffuse idiopathic skeletal hyperostosis. *J Rheumatol* 1988;15:129-31.
8. Fazl M, Bilbao JM, Hudson AR. Laceration of the aorta complicating spinal fracture in ankylosing spondylitis. *Neurosurgery* 1981;8:732-4.
9. Schaberg FJ. Aortic injury occurring after minor trauma in ankylosing spondylitis. *J Vasc Surg* 1986;4:410-1.
10. Fishman EK, Magid D. Cervical fracture in ankylosing spondylitis: value of multidimensional imaging. *Clin Imaging* 1992;16:31-3.
11. Goldberg AL, Daffner RH, Schapiro RL. Imaging of acute spinal trauma: an evolving multi-modality approach. *Clin Imaging* 1990;14:11-6.
12. Farhat SM, Schneider RC, Gray JM. Traumatic spinal extradural hematoma associated with cervical fracture in rheumatoid spondylitis. *J Trauma* 1973; 13:591-8.

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