

Acute pancreatitis presenting as back pain: a case report

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A man with acute back pain presented to a chiropractic clinic with clinical symptoms and signs suggesting abdominal disease rather than mechanical spine pain. He was referred to a local hospital emergency where a diagnosis of acute pancreatitis secondary to chronic cholecystitis was made. The diagnostic images are compared to normal studies. The characteristic clinical examination findings found with back pain due to acute pancreatitis are compared to those typically seen with mechanical spine pain.

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KEY WORDS: acute pancreatitis, back pain, chiropractic, manipulation.

Un homme souffrant de douleur aiguë au dos s'est présenté chez un chiropraticien avec des signes et symptômes suggérant plutôt un problème abdominal que vertébral. Référé à l'urgence d'un hôpital local, il fut diagnostiqué comme ayant une pancréatite aiguë secondaire à une cholécystite chronique. Les images diagnostiques sont comparées à des études normales. Les caractéristiques physiques d'un mal de dos d'origine pancréatique sont comparées aux résultats typiques d'un mal de dos dû à une mauvaise mécanique vertébrale.

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MOTS CLÉS : pancréatite aiguë, maux de dos, chiropratique, manipulation.

Introduction

Back pain is a symptom with many possible causes. The most common type is musculoskeletal back pain secondary to trauma, degenerative change, or postural stresses.^{1,2} This type of back pain is the most common patient complaint seen by chiropractors.³⁻⁶ However, patients with less common causes of back pain can present to chiropractic offices. This report follows the clinical course of a patient with back pain due to acute pancreatitis, from the time he presented to a chiropractic office to follow-up, six months after the date he was discharged from hospital.

The clinical findings seen in this case of back pain due to acute pancreatitis are compared to those typically seen with mechanical back pain.

Case report

A 44-year-old man, presented to our chiropractic clinic with increasing back, chest and left shoulder pain of 14 hours duration. There was no injury. The patient described a sharp, severe and diffuse pain in thoracolumbar region. The chest and shoulder pain was dull. He was restless, yet the pain was aggravated by movement. He was more comfortable sitting quietly with his knees pulled to his chest. He complained of nausea. There were no genito-urinary symptoms.

Similar episodes of back pain were reported to have occurred during the preceding 10 to 15 years. He claimed that these episodes of back pain frequently improved within one or two days of chiropractic treatment to the back.

On physical examination, the patient was in obvious discomfort. His skin color was slightly jaundiced. The skin felt cool and clammy. The pulse was rapid, but the blood pressure, heart and lung sounds and body temperature were within normal limits. Lumbar and cervical spine ranges of motion were full. Straight leg raising was 90 degrees. There was diffuse thoracolumbar paraspinal muscle tenderness. The neurological examination, including sensory and motor examination of the lower limbs, deep tendon reflexes, pathological reflexes and cranial nerve examination were unremarkable. The lumbar spine examination was normal and sacroiliac joint stress did not

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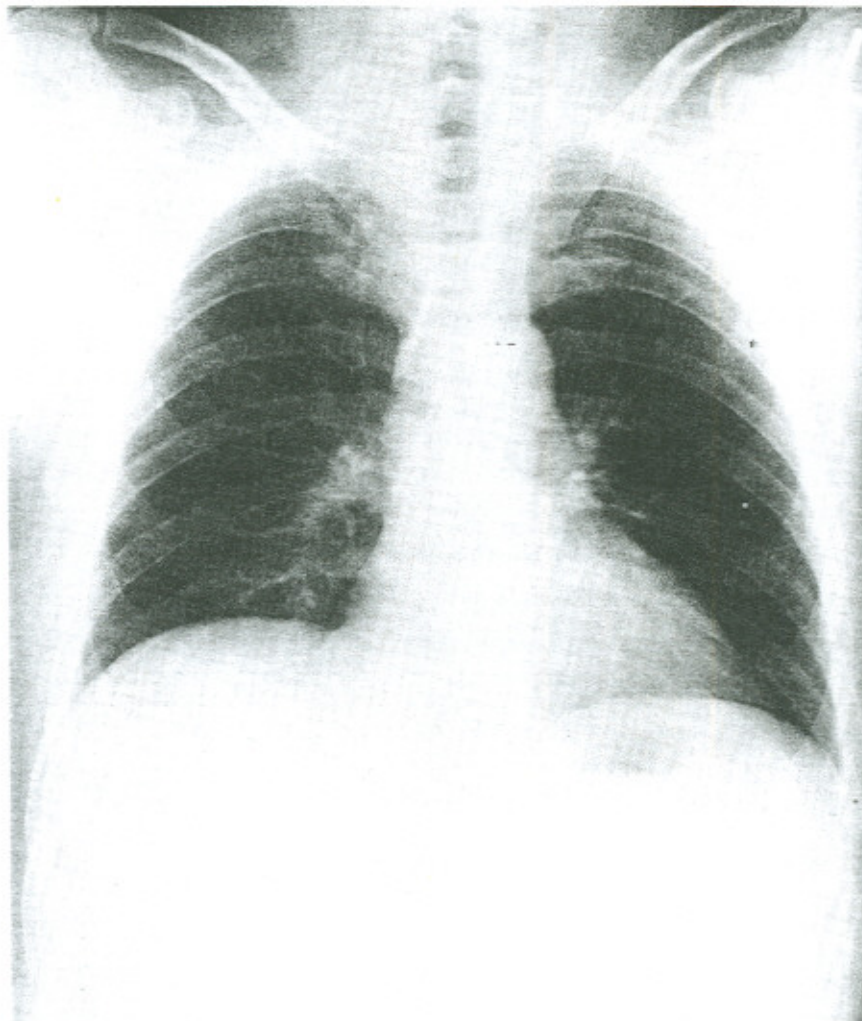


Figure 1 A P-A chest radiograph taken on arrival at emergency. Note the clear lung fields and clearly visualized, sharp costophrenic and cardiophrenic angles in this normal study.

reproduce his symptoms. There was marked abdominal rigidity with pain, tenderness and rebound tenderness in all quadrants.

The back symptoms and signs suggested abdominal disease rather than mechanical back pain. The patient was referred to the nearby hospital emergency department where he had lumbar spine and chest radiographs taken along with blood analysis (see Figure 1).

The lumbar spine and chest radiographs were normal. However, some of the blood chemistry values were abnormal (see Table 1).

An inflamed and markedly contracted gallbladder, packed with non-opacified stones was seen on the abdominal ultrasound (see Figure 2a and 2b).

He was admitted to hospital. Two days following admission he developed a spiking fever and increasing symptoms. Repeat chest radiographs (see Figure 3) and an upper abdominal CT scan (see Figure 4a and 4b) showed pleural effusion and lower

lobe atelectasis. The pancreas was diffusely enlarged. The surrounding tissue planes were indurated with edema and inflammation, spreading into the anterior perirenal spaces (see Figures 5a and 5b).

A diagnosis of acute pancreatitis secondary to chronic cholecystitis with cholelithiasis was made.

In hospital, he was intubated with a nasogastric tube to aspirate gastric contents. This was done to reduce pain and decrease the stimulation of pancreatic secretion. Nothing was given by mouth, rather he was given intravenous electrolytes to maintain electrolyte balance and to avoid dehydration. This treatment continued for several days while the symptoms abated and the serum amylase levels returned to near normal levels. He subsequently underwent a cholecystectomy. He was discharged from hospital a few days later without any complications. Six months later he was well without backache or abdominal symptoms.

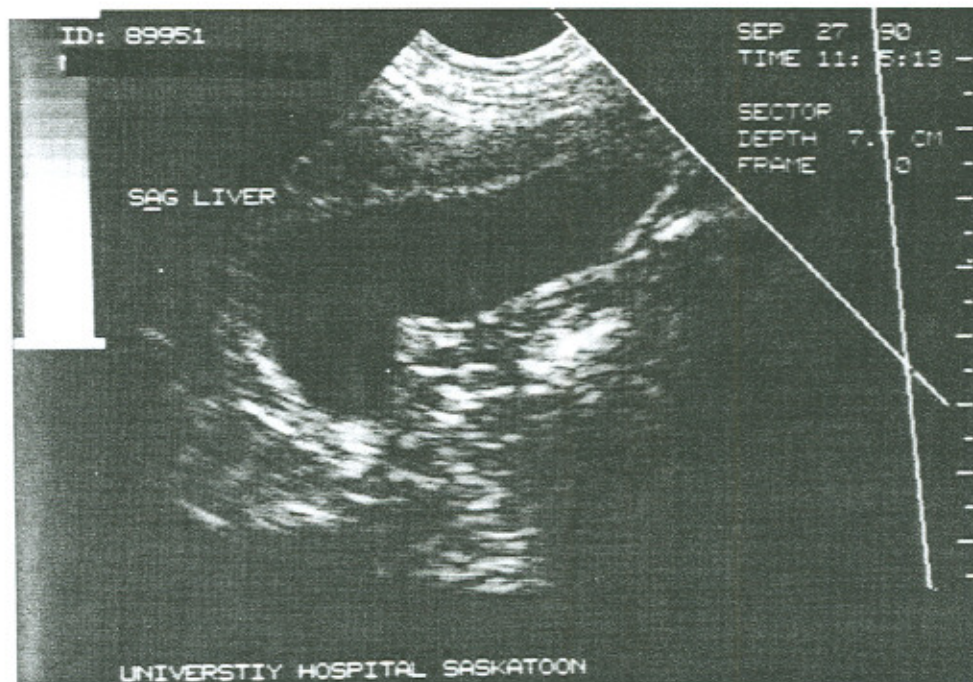


Figure 2a Normal abdominal ultrasound. Sagittal section of the gallbladder. Note the large lumen and thin gallbladder wall.

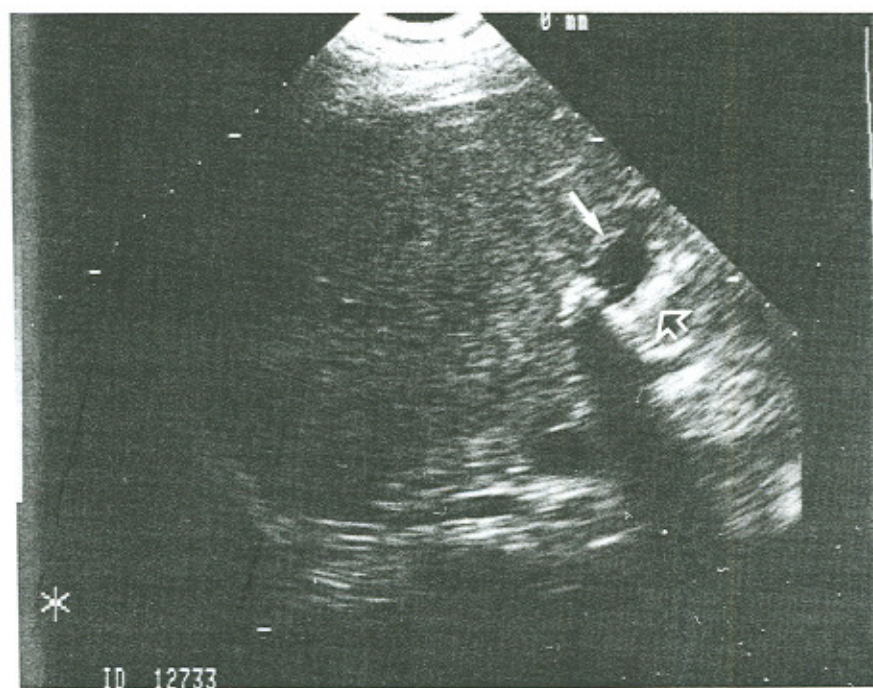


Figure 2b Abdominal ultrasound taken on arrival at emergency. The gallbladder is inflamed and contracted. The lumen is very small (solid arrow) and the gallbladder wall is thick and inflamed (open arrow). There is a gallstone within the gallbladder.

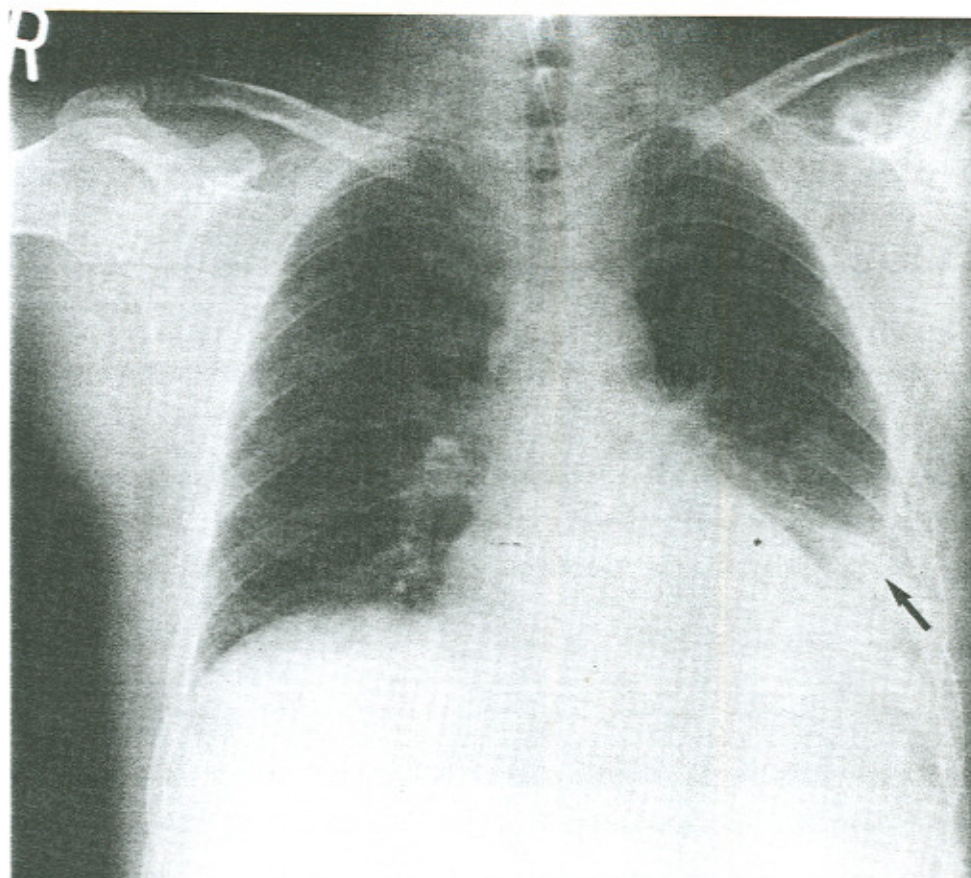


Figure 3 Patient's P-A chest radiograph taken 2 days after the admission to hospital. There is pleural effusion in the left lower lobe (arrow), blunting of the left costophrenic and cardiophrenic angles partially obscuring the cardiac silhouette.

Table 1
The patient's blood chemistry profile compared to normal values

SUBSTANCE	VALUE		UNITS
	Patient	Normal	
Na+	141	135-145	mmol/L
K+	4.3	3.5-5.0	mmol/L
Cl-	101	101-111	mmol/L
CO ₂	26	24-32	mmol/L
Urea	3.9	2.5-7.5	mmol/L
Creatinine	70	60-136	umol/L
*Glucose	8.4	3.6-6.1	umol/L
*Total bilirubin	41.0	10-29	umol/L
*Amylase	1503	30-115	Units/L
*AST	484	10-30	Units/L
*ALP	172	43-122	Units/L
*ALT	587	6-56	Units/L
*GGT	1296	8-78	Units/L

*Indicates abnormal values.

Table 2
Some common causes of acute pancreatitis

Gallstones
 Chronic alcohol abuse
 Drugs, e.g.: thiazides, furosemide, oral contraceptive
 Metabolic, e.g.: parathyroidism, hyperlipidemia
 Local inflammation due to gastric or duodenal ulcer
 Post-operative

*Adapted from Wall PD, Melzack R. Textbook of Pain.
 New York: Churchill-Livingstone, 1984: 352.

Discussion

Pancreatitis

Eighty percent of acute pancreatitis is due to biliary tract diseases, secondary to cholelithiasis or chronic alcohol abuse (see Table 2).⁷ Acute pancreatitis, secondary to cholelithiasis, is an acute inflammatory response. The pancreas likely returns to normal between attacks.⁷ However, recurrent episodes of acute pancreatitis, secondary to alcohol abuse, occur in a pancreas that has been permanently damaged.⁷ This patient denied frequent or excessive use of alcohol.

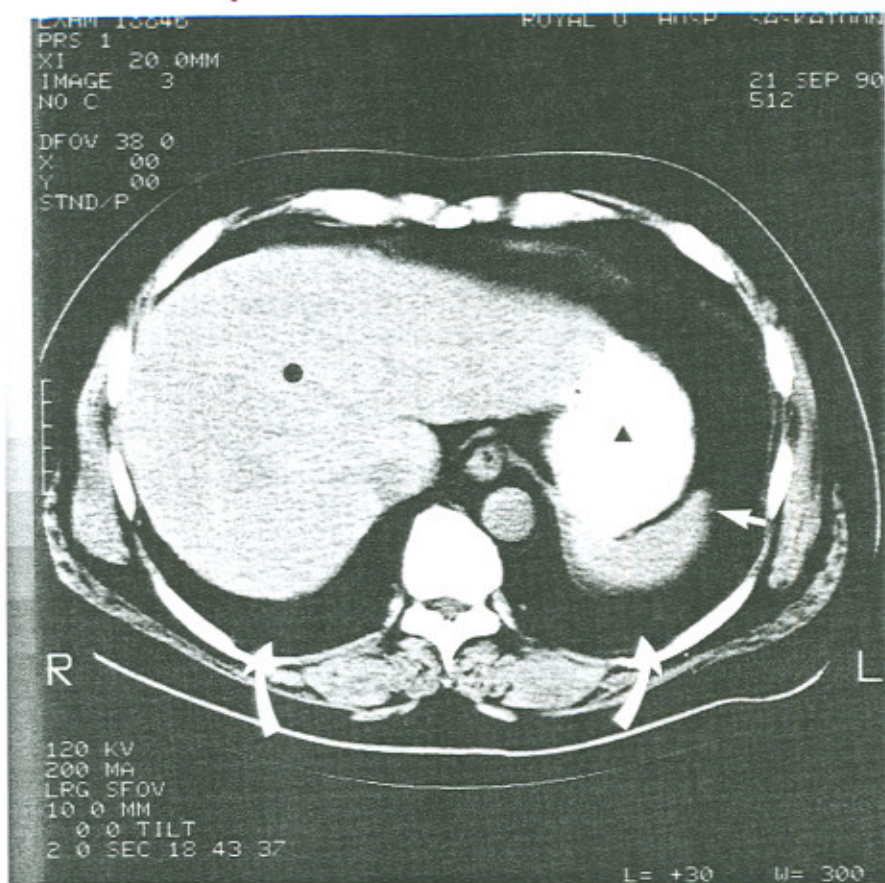


Figure 4a Normal CT scan of the upper abdomen. The liver is clearly defined (circle). The gastric antrum is filled with contrast substance (triangle). The spleen (straight arrow) and radiolucent posterior lower lobes of the lungs (curved arrows) are clearly seen.

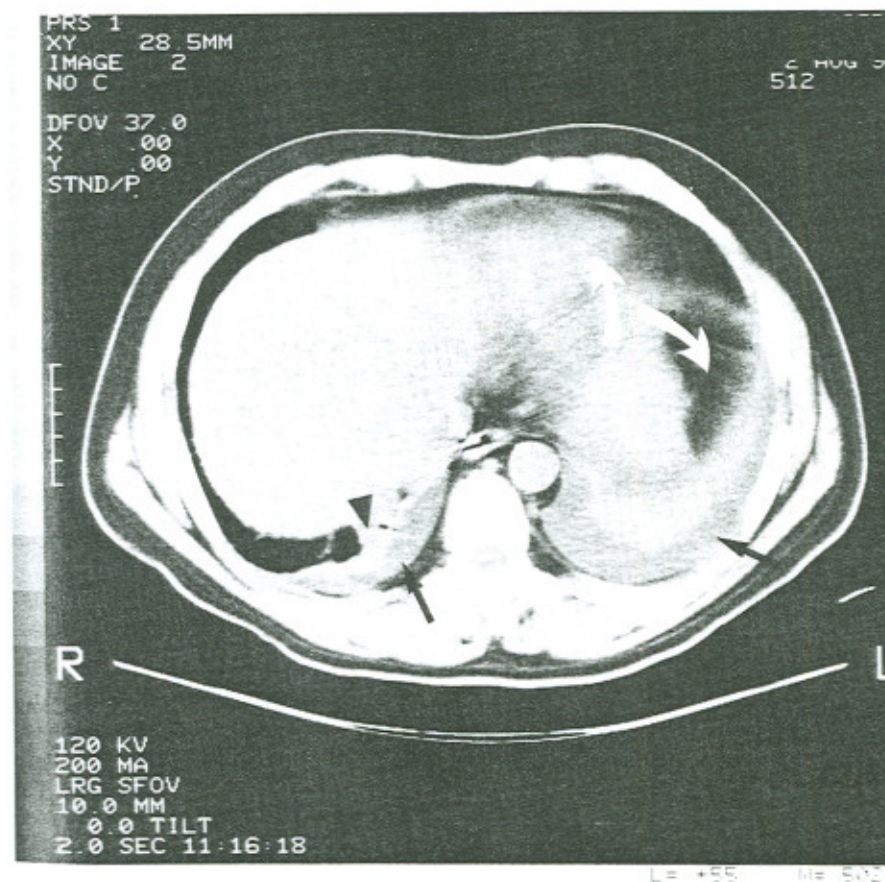


Figure 4b Patient's CT scan of the upper abdomen 2 days after hospital admission. There is bilateral pleural effusion (dark arrows) and lower lobe atelectasis (triangle). The hazy edges of the liver and spleen (white arrows) is due to intra-abdominal edema.

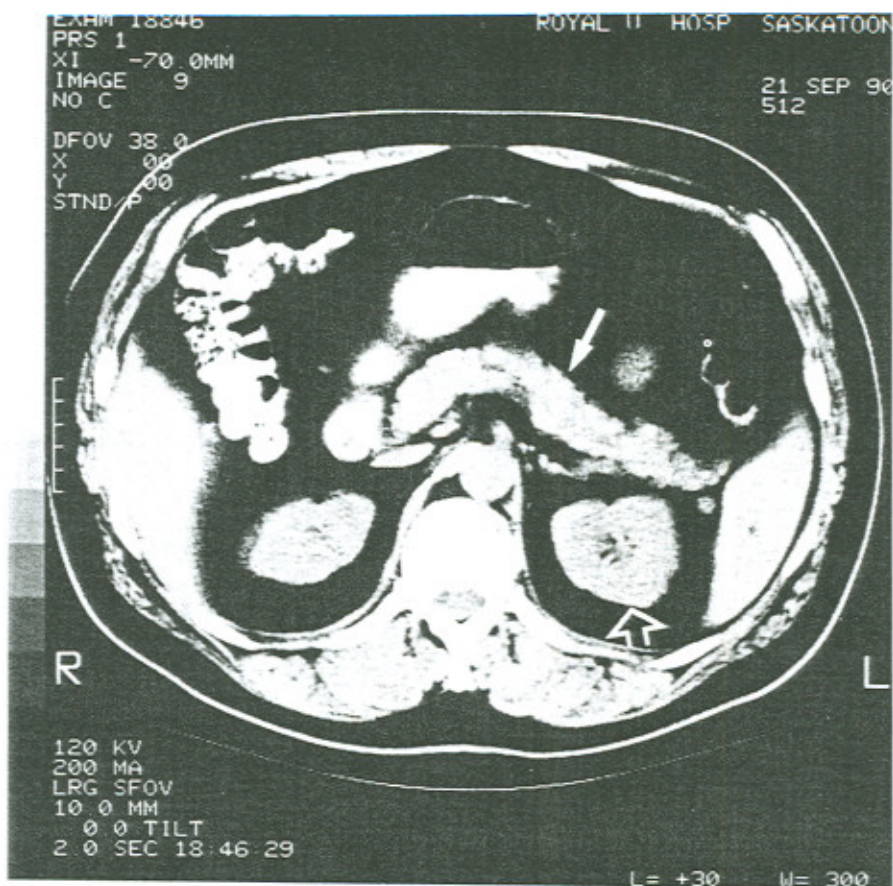


Figure 5a Normal CT scan of upper abdomen. The pancreas (white arrow) and kidneys (open arrow) are sharply defined.

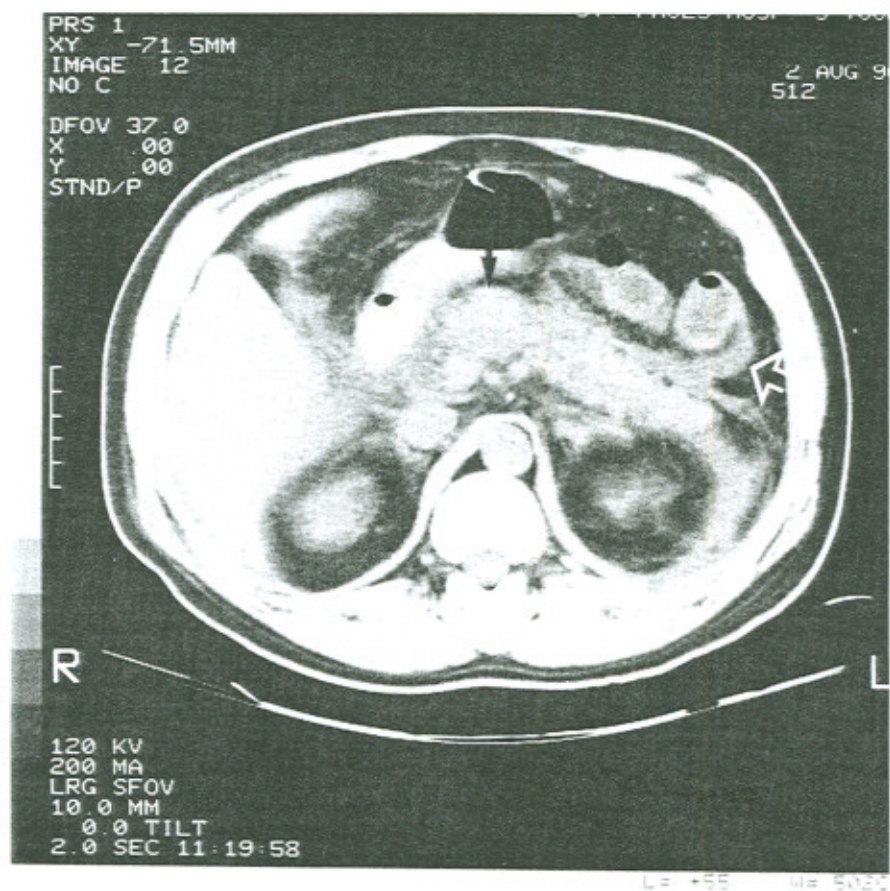


Figure 5b Patient's CT scan of upper abdomen 2 days after hospital admission. There is diffuse enlargement of the pancreas (dark, solid arrow). The tissue planes surrounding the pancreas (white, open arrow) are obscured by edema spreading into the perirenal fat.

Acute pancreatitis occurs when conditions in the pancreas lead to autodigestion of the pancreas by its own enzymes.⁷ Pancreatic proteases, activated within the pancreas rather than within the lumen of the proximal small bowel, digest the pancreas and peripancreatic tissues. The exact mechanism responsible for initiating this process is not clearly understood. However, the frequent association between biliary tract disease and acute pancreatitis has led to the theory that an obstruction of the sphincter of Oddi allows a reflux of bile along the pancreatic duct resulting in enzyme activation.⁸

The dominant presenting patient complaint with cases of acute pancreatitis is pain.⁹ The epigastric pain, if present, can vary from a mild and tolerable discomfort to severe, constant and incapacitating pain.⁷

Pancreatitis and back pain

Acute pancreatitis is one of several painful conditions, along with perforated duodenal ulcer and myocardial infarct, wherein visceral pain is referred to the spine.² The back pain of pancreatitis is typically referred to the region of the tenth to twelfth thoracic vertebrae. From there the pain can radiate to the sub or mid scapular regions and to the left shoulder (see Figure 6).^{2,9,10} The characteristic referral of pain to the back occurring with acute pancreatitis is due, in part, to the neurological phenomenon of referred pain, as well as to the position of the pancreas in the abdomen.

The pancreas lies transversely across the abdomen in a retroperitoneal position against the posterior abdominal wall, behind the stomach and in front of the aorta, at the level of the second lumbar vertebrae.¹³ Pancreatic disease can cause back pain by direct stimulation of nociceptive fibers in the posterior abdominal wall which is well innervated by ventral branches of the intercostal nerves.^{2,13}

The clinical phenomenon of referred pain is explained by the convergence theory. This theory suggests that afferent nerve fibers from muscles, joints, skin and viscera converge onto the same or adjacent cells in the spinal cord.^{2,11} A number of investigators have shown that visceral afferent fibers and small myelinated cutaneous afferent fibers converge onto lamina five cells of the spinal cord.¹²

The pancreas is innervated by the splanchnic nerves. These nerves are made up of preganglionic sympathetic fibers, arising in the spinal cord, that travel through the fifth to tenth thoracic sympathetic ganglia (sympathetic chain) to join at the tenth to twelfth thoracic interspace to become the greater splanchnic nerve.⁹ The lesser splanchnic nerve arises from sympathetic ganglia at the ninth and tenth thoracic levels.⁹ The splanchnic nerves, made up of preganglionic sympathetic fibers, travel through the diaphragm to the celiac plexus from which postganglionic sympathetic fibers travel with the blood supply to the pancreas.⁹ The close relationship between the sympathetic innervation of the pancreas and the segmental innervation of joint, muscle and skin in the lower thoracic spine explains the frequent referral of pain to the lower thoracic region of the back with acute pancreatitis.

Severe and chronic pancreatic pain, like that of carcinoma of the pancreas, is often relieved by an operation to cut the splanchnic nerves to the pancreas.^{2,9,13}



Figure 6 Typical pain referral pattern of acute pancreatitis. (Adapted from Given BA, Simmons ST: *Gastroenterology in Clinical Nursing*. 2nd ed. St. Louis: CV Mosby Co, 1975: 12.)

Diagnosis of acute pancreatitis

The diagnosis of acute pancreatitis is confirmed, when suspected following the history and clinical examination, by analysis of the pancreatic enzyme serum amylase.⁷ Serum amylase is greatly elevated in a patient with acute pancreatitis from a normal value of less than 200 units/L to one of over 1000 units/L.² (Table 1).

Mortality of acute pancreatitis

Acute pancreatitis has a reported mortality rate of 10 percent.⁷ If the condition progresses to the hemorrhagic stage, the prognosis is poor and mortality rate exceeds 50 percent.¹⁴ Therefore, early diagnosis of acute pancreatitis is critical for the initiation of appropriate treatment.

Musculoskeletal conditions mimicking the pain of pancreatitis

Musculoskeletal pain syndromes can present with pain patterns similar to those referred from diseases of the viscera.¹² A number of musculoskeletal conditions can mimic the pain of acute pancreatitis. These include a thoracic posterior joint syndrome or an upper rectus abdominus trigger point. The absence of illness or signs of abdominal disease, and the presence of specific clinical signs implicating a musculoskeletal structure, suggest a musculoskeletal cause for the back pain rather than abdominal disease (see Table 3).

The pain of a thoracic posterior joint syndrome occurs at the same side and spinal level as the affected joint (see Figure 7).¹⁵ There can be referral of pain laterally, along the course of the corresponding rib, and to the spinal segment situated immedi-

Table 3
The clinical features of mechanical back pain
compared with those of abdominal disease

Signs of mechanical spine pain	Signs of back pain due to abdominal disease
Specific restriction of spinal motion	Pallor, fever, cold and clammy skin, jaundice
Local spinal tenderness (unilateral)	Abdominal guarding and tenderness
Pain provocation by specific examination procedures	Lack of pain localizing signs in the spine
	Abnormal vital signs (H.R., B.P., Resp.)
	Abnormal blood values (WCB, chemistry, enzymes)

ately above or below the involved segment, or both. This referral of pain and tenderness to the surrounding spinal segments is due to the multisegmental nature of posterior joint innervation.¹¹ Thoracic spine range of motion is often restricted in one or more planes. The point of maximal tenderness is usually on the side and at the level of the involved posterior joint.

A trigger point in the upper rectus abdominus can refer pain to the back below the scapulae and above the twelfth rib in the T7 to T10 region (see Figure 8).¹⁶ There will be tenderness at the abdominal trigger point and digital pressure at this point often refers pain to the back.

Summary

In this case, the patient's past episodes of back pain were apparently treated with spinal manipulation with symptom improvement. That is why the patient returned to the chiropractic office with another episode of back pain. It is not known whether previous episodes of back pain were due to musculo-skeletal causes, which could explain the symptomatic improvement following chiropractic treatment, or whether the improvement in the symptoms were due to the recurrent, relapsing nature of the chronic cholecystitis. This time the cholecystitis did not resolve but rather progressed to acute pancreatitis.

Patient referral to a hospital is essential in a case of acute pancreatitis to prevent the development of serious complications. Referral will not be delayed if the chiropractor recognizes the signs of acute pancreatitis in a patient presenting with back pain due to acute pancreatitis.

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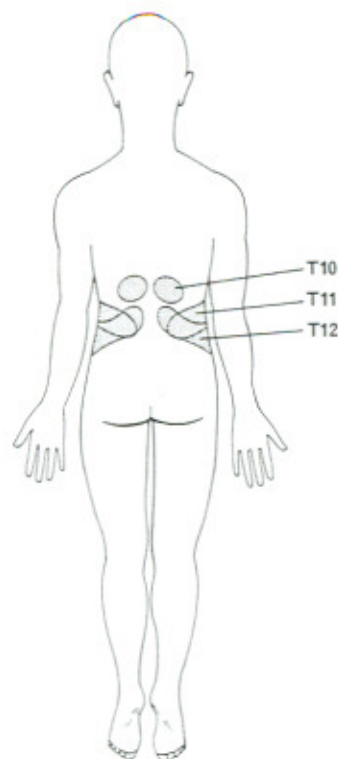


Figure 7 Pain patterns that occur in T10, T11 and T12 posterior joint syndromes. (Adapted from Kellgren JH: The anatomical source of back pain. *Rheum Rehab* 1977; 16:6.)

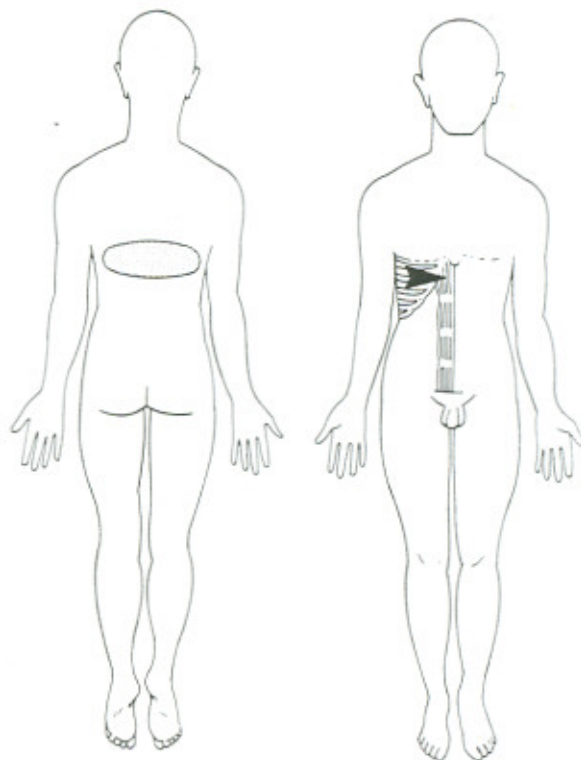


Figure 8 Pain referral pattern that can occur with an upper rectus abdominus trigger point. (Adapted from Travell JG, Simons DG. *Myofascial Pain and Dysfunction: The Trigger Point Manual*. Baltimore: Williams & Wilkins, 1983: 664.)-

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