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Osteoporotic Vertebral Compression Fractures Presenting With An Acute Ileus --A Case report

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We report a 76 year-old female of T11 and T12 vertebral compression fracture, presenting with abdominal pain and acute ileus but minimal back symptom within 24 hours of onset. The treatment was focused on the abdominal condition, exploratory laparotomy was done with normal intra-abdominal organ, except diffuse moderate distention of intestines. Gallium-67 scan was arranged to differentiate recent from old fracture. The mechanism of thoracolumbar compression fractures presenting with an acute ileus is possible due to the retroperitoneal hemorrhage or structure change irritating the sympathetic chains which innervate the intestines. Treatment of acute ileus were reviewed and discussed.

Key words: vertebral compression fracture, acute ileus,low back pain, osteoporosis, pseudo-obstruction

INTRODUCTION

Osteoporotic vertebral compression fractures (VCFs) usually presenting with back pain, the pain is intense and deep at the site of fracture, and may radiate anteriorly along the affected nerve root. The characters of pain is relieved by rest and aggravated by activities. The VCFs accomplished with acute ileus within 24 hours is rare.

CASE REPORT

A 76 year-old female patient suffered from mild back pain when she sat up from the bed without traumatic evidence in the evening. Initially, the back pain was tolerable, but abdominal pain developed in the early next morning followed with abdominal distension. The family took her to the local hospital with chief complaint of abdominal pain. The symptoms were aggravated when she was changing positions and

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relieved when she was resting. Under the impression of acute abdomen with ileus, surgical intervention was suggested and the patient was transferred to the emergency room(ER) of Chang Gung Memorial Hospital. She had symptoms and signs of nausea, diffuse abdominal pain and tenderness. KUB showed dilation of small intestines and colons with gas (Fig.1) due to paralytic ileus . The lateral view of thoracolumbar spine showed T11 and T12 compression fracture (Fig.2). As the abdominal pain is more predominant than the low back pain, the treatment was focused on the abdominal condition. However the abdominal pain and tenderness persisted and aggravated despite conservative treatment. Hence, exploratory laparotomy was performed immediately on the day of admission to ER. The intraoperative findings were diffusely moderate distention of small intestines and colons, absence of ascites, and normal intrabdominal organs. After detail intraoperative examination, rectal tube was inserted for decompression and abdominal incision wound was closed. The postoperative diagnosis was pseudo-obstruction.

Tracing back her history, she had suffered from two episodes of stroke with minor neurological deficit of right hemiparesis. Unfortunately, she suffered from the third episode of stroke the next day immediately after the exploratory laparotomy with sequelae of right hemiplegia and aphasia. The patient was transferred to the rehabilitation department when her neurological condition was stable. The patient's conscious level was E4VAM5. Reviewing the history and plain film of radiography, the osteoporotic vertebral compression fractures presenting with an acute ileus was diagnosed. Gallium-67 scan was arranged in order to differentiate recent from old fracture. It revealed increasing up-take of radioactivity at the mid back (Fig.3a and Fig.3b) which corresponded to low thoracic spine level. This suggest a recent fracture and compatible with the findings of T11 and T12 compression fracture in plain film radiography. Tayler's brace was prescribed. Because of poor cooperation to rehabilitation program and economic problem, she was discharged from hospital. The patient had been followed up for three months after the operation. The abdominal condition was rather well without recurrence of ileus.

DISCUSSION

Vertebral compression fractures (VCFs) can be diagnosed based on radiological findings and/or clinical symptoms. Radiographic findings such as crushed or wedge-shape deformed vertebrae are used to diagnose VCFs. However, greater than 15% of vertebral height loss noted on the X-ray is used as an objective criteria for the diagnosis of VCFs[1]. The clinical manifestations of VCFs encompass a spectrum ranging from acute lower back pain with neurological deficits to a asymptomatic state. In the study of Cooper et al. sixteen percent of the diagnosed VCF's cases were made incidentally when radiographic investigation was arranged due to other unrelated disorders or as part of the screening examination for the high risk patients[2]. The low back pain in acute VCFs is intense and deep at the site of fracture with or without pain radiating anteriorly along the affected nerve root dermatome. The pain can be aggravated by activities involving changing of back posture such as

bending or arising and relieved by lying down, or physical modality [3]. The duration of pain usually lasts for two weeks to three months.

Thirty patients of VCFs were studied by Patel et al. Low back pain radiated to the flanks and anteriorly along the affected dermatomes was noted in 66% of the cases. Pain radiated to leg was uncommon (6%). Majority of the VCFs occurred spontaneously (46%), including whilst in bed (30%), or after trivial strain (36%). The associated symptoms were nausea (26%), abdominal pain (20%) and chest pain (13%)[4]. No paralytic ileus had been reported in this report.

Seventy patients who met specific criteria of acute traumatic (burst-type) thoracolumbar fracture (T10-L2) without neurological compromise were studied by Juan et al.[5]. Among them, there were only four (6%) of these patients developed intestinal dysfunction which manifested as vomiting, abdominal distention and diminished bowel sounds. One of these patients had an abdominal roentgenogram which revealed gaseous distention in large and small intestines, indicative of a non-obstructive ileus. Such abdominal roentgenogram findings were noted in our case report, too. In all four cases the dysfunction developed within 24 hours post-trauma, which was similar to our case here, but without abdominal operation. All four cases intestinal dysfunction persisted for less than seven days, with an average of four days. Two of the patients who developed intestinal dysfunction had significant injuries other than the vertebral fracture. Our patient had similar manifestations with more prominent abdominal symptoms than back symptoms on the fracture site, but the VCFs is non-traumatic in origin. From the literature review, non-traumatic origin VCFs case with acute ileus is very rare. Only one case was reported by Treadwell, et al. This elderly female patient was a case of systemic lupus erythematous with long-term corticosteroid therapy.She had thoracolumbar compression fracture presented with acute abdomen and minimal low back pain. No intra-abdominal abnormality was found from abdominal studies and exploratory laparotomy[6]

The mechanism of thoracolumbar compression fractures presenting with an acute ileus is poorly understood. The retroperitoneal hemorrhage around the fracture site has been suggested as one possible cause [7,8]. This bleeding and/or acute inflammation around the fracture site may disturb the autonomic nervous system by irritating the sympathetic chains innervating the intestines. The relationship between the level of thoracolumbar fracture and ileus had been stated by Ramamurti[9]. The preganglionic fibers of the sympathetic nervous system lying near the T12, L1, L2 regions are involved in the neurological control of intestines. It is quite possible that disruption at this region by vertebral fracture could affect the intestinal innervation. The most localizable sensations (such as pain, distention, etc.), referable to the gastrointestinal tract, appear to be mediated through the sympathetic afferents[10]. With the bleeding after thoracolumbar fracture, inflammation and structure change which occurred in front of the vertebral body will irritate the sympathetic trunk through afferent or efferent fibers and induce the vicious cycle. Then abdominal pain, distention and paralytic ileus developed.

The treatment of acute pseudo-obstruction of the colon may involve conservative medical

management, colonoscopy, or surgery. Conservative medical management includes intravenous fluid replacement, correction of metabolic disorders, nasogastric suction, nothing by mouth, discontinuation of narcotics, and treatment of any underlying predisposing conditions. Rectal tubes, sigmoidoscopy, or enemas are not of benefit. If there is no improvement in the clinical status or cecal size, or persisted abnormal cecal size greater than 12 cm from follow-up X-ray , then colonic decompression must be performed. The success rate of colonoscopic decompression is higher if the colonoscope passed to or beyond the hepatic flexure. If colonoscopy reveals any evidence of ischemia, the procedure must be stopped immediately. A resection is then performed with either exteriorization or primary anastomosis of the bowel. In the setting of unsuccessful colonoscopic decompression of the colon with a cecal size greater than 12 cm, a cecostomy must be performed. Ponsky described a technique in which a cecostomy tube was placed in a percutaneous fashion under endoscopic guidance. The technique is similar to the one described for percutaneous gastrostomy[11].

VCFs is one possible cause of the acute ileus by sympathetic chains irritation. Conservative treatment can achieve good response by careful clinical judgement and early proper diagnosis.

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骨質疏鬆椎骨壓迫性骨折合併急性麻痺性腸塞－病例報告

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骨質疏鬆椎骨壓迫性骨折，常以背痛表現，特徵為強度極強，且位於深部骨折處，有時因刺激神經根，而有腹痛的表現，但以急性麻痺性腸塞表現卻相當罕見。

本文報告一位七十六歲之女性，於某日傍晚起床時發現輕微背痛，隨著第二天凌晨產生腹脹、噁心想吐、瀰漫性腹部疼痛與壓痛，腹痛亦受翻身或移位之影響。X-光片顯示骨質疏鬆併第十一、十二胸椎壓迫性骨折與腸塞。因腹部症狀嚴重，在急性腹症與腸塞之診斷下，進行探索性開腹術，除大、小腸中度膨脹外，無不正常發現，不幸地術後第二天發生左腦中風，並於病況穩定後轉復健科，在病史與X-光照片重新檢閱後，疑為骨質疏鬆椎骨壓迫性骨折合併急性麻痺性腸塞。為證明骨折為新近病灶，安排Gallium-67核醫掃瞄檢查，於低位胸椎處有放射性物質吸收增加之現象，與X-光片病灶位置吻合。家屬因病患失語、年老及經濟問題，而拒絕復健治療。在術後三個月追蹤時，病患可輪椅活動，腹部狀況相當平穩。

推究骨質疏鬆椎骨壓迫性骨折合併急性麻痺性腸塞之可能機轉為：(一)以時間而言,局部急性出血或急性骨折導致結構改變，而刺激支配大、小腸之交感神經。(二)以病灶位置而言,支配大、小腸之交感節前神經位置靠近第十二胸椎與第一、二腰椎前方，故附近脊椎骨折，可導致急性腸塞。

由於此種病例在臨床上易被誤診，而接受不必要之手術治療，因此特別提出以供參考。

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