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Traumatic Lumbar Plexopathy by Seat Belt Injury

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Study Design: A case report.
Objectives: To report and discuss an extremely uncommon cause of lumbar plexopathy seat belt injury.
Summary of Literature Review: For patients who undergo traffic accidents, most cases of seat belt injury cause trauma to the lower torso. Seat belt injury is associated with variable clinical problems such as vascular injury, intestinal injury (perforation), vertebral injury (flexion-distraction injury), chest wall injury, diaphragmatic rupture/hernia, bladder rupture, lumbosacral plexopathy, and other related conditions.
Materials and Methods: A 38-year-old male truck driver (traffic accident victim) who suffered monoplegia of his right leg due to lumbar plexus injury without spinal column involvement. Injury to a lumbar plexus and the internal vasculatures originated from direct compression to internal abdominal organs (the iliopsoas muscle and internal vasculatures anterior to the lumbar vertebrae) caused by the seat belt. We have illustrated an extremely uncommon cause of a neurologic deficit from a traffic accident through this case.
Results: Under the impression of traumatic lumbar plexopathy, we managed it conservatively, and the patient showed signs of recovery from neurologic deficit.
Conclusions: We need to review the lumbar plexus pathway, in patients with atypical motor weakness and sensory loss of the lower extremities which are not unaccompanied by demonstrable spinal lesions. Therefore, close history taking, physical examination and comprehension of injury mechanism are important in the diagnosis.

Key words: Traumatic, Lumbar plexopathy, Seat belt injury

We present a case of 38–year–old man, a truck driver, who had suffered monoplegia of his right leg due to lumbar plexus injury without spinal column involvement after a traffic accident. This case also presented combining superior mesenteric artery rupture, pseudo–aneurysm of a common iliac artery, and fibular fracture. Injury to lumbar plexus and internal vasculatures originated from direct compression to internal abdominal organs (the iliopsoas muscle and internal vasculatures anterior to the lumbar vertebrae) by seat belt. This case illustrated an extremely uncommon cause of unexplained neurologic deficit after traffic accident.

Introduction

The seat belt injury described as a trauma affecting mostly the lower torsos of patients who wore a seat belt restraint during car accidents. Seat belt injury is associated with variable clinical problems.1) Lumbar plexopathy is defined as signs and symptoms due to dysfunction of the nerve plexus formed from the ventral division of the lumbar nerve roots. When a patient has sciatica or radicular symptoms, the clinician usually looks to the spine for the origin of symptoms because extra-spinal causes are less commonly considered in the differential diagnosis.2) We experienced the case of a 38–year–old man, restrained driver involved in a car accident, who had a lumbar plexopathy without involving any spinal lesions, combined with a branch rupture of superior mesenteric artery, pseudo–aneurysm of a mesenteric artery.
common iliac artery, and fibular fracture. The purpose of this report is to illustrate an extremely uncommon cause of lumbar plexopathy and discuss how it can be originated from seat belt injury.

**Case Report**

A 38-year-old male truck driver, wearing a seatbelt, involved rear-ends collision. The patient was rushed to the emergency department of hospital due to abdominal pain and neurologic impairment of his right leg. The patient had no special medical history other than getting an operation for herniated lumbar disc 5 years ago.

Neurologic examination showed obvious motor impairment in the right leg. The power of right hip, knee, and ankle joint movements were grade 0/5. He had markedly decreased touch sensation of the entire right leg except inguinal area and lateral aspect of hip area. The left leg showed normal muscle tone, power and sensation. On the x-ray of lumbar spine and pelvis, there were no definite abnormal lesions. On the x-ray of left lower leg, it showed fracture of mid shaft of fibula.

Other physical examination showed direct tenderness on

![Fig. 1](image-url)
whole abdomen area. The abdomen computed tomography (CT) scan showed a large amount of hemoperitoneum with active bleeding at a branch of superior mesenteric artery and short- segmental filling defect at the proximal right common iliac artery (Fig. 1-A, B). Tachycardia (124 bpm) and low blood pressure (69/52 mmHg) were measured. Follow-up laboratory test indicated that his hemoglobin dropped to 8.5 g/dL. At the intervention for embolization, we found a 2 cm-sized pseudoaneurysm as the cause of filling defect of right proximal common iliac artery (Fig. 1-C, D).

After the successful embolization, the lumbar spine magnetic resonance image (MRI) scan was performed to evaluate the cause of neurologic deficit of his right leg. It didn’t show any abnormal findings of spinal lesion that might explain the cause of neurologic deficit (Fig. 2-A). On the contrary, the axial view of MRI scan showed a signal change and swelling in the intramuscular and perimuscular area of right psoas and iliacus muscles, compared to the left side (Fig. 2-B). These findings were consistent with the diagnosis of psoas muscle injury and strongly correlated with the traumatic lumbar plexopathy. Under the impression of traumatic lumbar plexopathy, we managed it conservatively.

Overnight following intervention, the patient was in a stable condition. The next day power of his right hip, knee, and ankle joint movements were measured with grade 2–3/5 and he showed reaction to pin prick test which indicated signs of recovery. He transferred to a hospital closer to his residence for further rehabilitations.

**Discussion**

The seat belt syndrome is a recognized complication of seatbelt use in vehicles.¹ Seat belt injury causes direct compression and indirect deceleration vector forces acting on anatomic structures.² Direct forces are triggered by the impact with seat belt. Indirect forces originate from the resolution of deceleration vector into a longitudinal and a transverse component, in

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relation to the vertebral spine. The former direct force exerts a compression on the abdominal contents and on the iliopsoas muscle. The latter indirect force results in traction injury to the lumbar plexus. It may cause various clinical problem such as vascular injury, intestinal injury (perforation), vertebral injury (flexion–distraction injury), chest wall injury, diaphragmatic rupture/hernia, bladder rupture, lumbosacral plexopathy, etc.4)

It characteristically involves seat belt sign. The seat belt sign describes skin bruising or avulsion mostly across the anterior chest or abdominal wall. Its presence should always arouse the suspicion of the clinician to the probable presence of intra-abdominal or spinal injuries. Following such suspicion, CT or MRI scan of the spine and abdominal cavity should be performed to search for covert injuries. Our patient showed a seat belt sign on his abdominal wall area (Fig. 3) as well as he had both vascular injury and lumbar plexopathy without involving any vertebral injury.

The lumbar plexus represents the nerve supply to the pelvis and lower limb. The lumbar plexus is formed from the anterior rami of the T12–L5 nerve. It is formed within the substance of the psoas major muscle anterior to the transverse processes of the L2–5 vertebrae. It gives off a number of terminal branches, which emerge from the lateral border of the psoas muscle. The femoral nerve also emerges from the lateral psoas border before coursing along the groove between the iliacus and psoas muscles.5) The anatomical course from the lower spine and along the lateral pelvic walls predisposes the lumbar plexus to direct and indirect involvement by blunt trauma. Under such anatomy, seat belt injury causes swelling of iliopsoas muscle and compression and traction injury to lumbar plexus. Finally, it is followed by neurologic deficits such as hypoesthesia and motor weakness in the lower extremity.

For differential diagnosis, CT or MRI scan is recommended for its high sensitivity for the detection of muscle swelling and pathway of injured lumbar plexus. An understanding of the clinical and radiological features is essential in making the correct diagnosis.

In patients who develop a traumatic lumbosacral plexopathy, managements are still controversial and unresolved.6) Both conservative and surgical management have been applied. However, there is no clear basis for favoring one over the other. Crosby et al. reported treatment results for 61 patients who had lumbar plexopathy due to iliopsoas hematoma. In the 51 patients treated conservatively, 49% of them had a complete recovery. The other ten patients resulted in 50% total recovery after surgical treatment.6)

We stress the need to review the lumbar plexus pathway in patients with atypical motor weakness and sensory loss of lower extremity unaccompanied by demonstrable spinal lesions. Therefore, close history taking, physical examination and comprehension of injury mechanism are very important in the diagnosis.

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연구 계획: 증례 보고
목적: 요추 신경총병증이라는 흔하지 않은 질환에 대해 살펴보고 이 질환이 안전벨트 손상으로 인해 어떻게 야기될 수 있는지 문헌 고찰과 함께 보고하고자 한다.

선행문헌의 요약: 안전벨트 손상은 자동차 사고 때 안전벨트를 매고 있던 환자의 몸통 하부에 주로 가해지는 외상을 일컫는다. 안전벨트 손상은 혈관 손상, 내장 손상, 척추 손상, 흉곽 손상, 횡격막 파열, 방광 파열 및 요추신경총병증과 같은 여러 임상적 문제와 연관이 있다.

대상 및 방법: 척추 병변이 보이지 않는 요추 신경총병증으로 인해 오른쪽 하지 단마비가 발생한 38세 트럭 운전사가 내원하였다. 안전벨트로 인해 내부 장기(장요근, 요추 전방 혈관계)가 직접적인 압박을 받음으로써 발생한 요추 신경총병증이었다. 이에 급변 증례보고를 통해 교통사고 후 발생하는 흔하지 않은 신경학적 결손에 대해 실증하였다.

결과: 외상성 요추 신경총병증을 생각하고 척추를 보존적 치료하였다. 이후 환자는 신경학적 결손으로부터 회복 증상을 보였다.

결론: 비전형적인 하지 운동 및 감각 소실을 보이는 환자에서 이를 입증할만한 척추 병변을 동반하지 않는 경우 요신경총의 주행에 대해서 검토해 보아야 한다. 결국, 환자의 진단에 있어서 면밀한 병력조사, 신체검진 및 손상 기전의 이해가 매우 중요하다고 할 수 있다.

색인 단어: 외상, 요추 신경총병증, 안전벨트 손상
학회 제목: 안전벨트 손상으로 인한 요추 신경총병증

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