Diagnose einer traumatischen Bauchwandhernie 14 Jahre nach einem Sturz mit LWK-Fraktur

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Abstract

Traumatische Bauchwandhernien können für Patienten und Versicherungen, insbesondere wenn sie beim Unfall nicht diagnostiziert werden, erhebliche Bedeutung haben.
In diesem Fallbericht erlitt ein Gärtner einen Sturz von einem Carport im Jahre 1994 und wurde sofort ins Krankenhaus aufgenommen. Es wurde eine Fraktur des zweiten LWK diagnostiziert und operativ stabilisiert. Postoperative CT und MRT Untersuchungen zeigten korrekte Heilung der Fraktur. Die Schmerzen im Kreuzbein, linkes Bein und linker Unterbauch sowie die plötzlichen Schmerzattacken in den Leisten mit Bevorzugung der linken Seite persistierten. Verschiedene Neurologen sahen als Ursache der Schmerzen eine Veränderung der LWS mit Radikulopathie. Als Folge der neurologischen Untersuchung wurde bei dem Patienten eine Verblockung des vierten und fünften LWK durchgeführt. Leider kam es dadurch nicht zu einer Beseitigung der Schmerzen oder der neurologischen Befunde. Es kam in der Folge zu einer Verstärkung der Beschwerden, die dazu führte, dass der Patient nicht mehr die Kupplung im Wagen betätigen konnte, keine Treppen mehr steigen konnte und auch keine längeren Gehstrecken mehr bewältigen konnte. Im Jahre 2008 stellte Professor Gresser, Internistin und Rheumatologin in Sauerlach, bei dem Patienten die Diagnose traumatische Bauchwandhernie mit Nerveneinklemmung. Diese Diagnose wurde durch den intra-operativen Befund und die histologische Untersuchung der Gewebes bestätigt. Durch eine außerordentlich zeitaufwendige, anatomisch-orientierte und gewebeschonende Operationstechnik konnten die Schäden der Bauchwand und des Nervenkompressionssyndroms beseitigt werden. Der Patient hatte unmittelbar nach der Operation keine Schmerzen mehr im Kreuzbein und eine fast vollständige Beseitigung der bisherigen Schmerzen. Er konnte wieder Treppensteigen, ungehindert Autofahren und auch wieder länger gehen.
Schlussfolgerung: Patienten mit unklaren chronischen Schmerzen in der unteren Bauchwand, Leiste, Hüfte oder Oberschenkel können an den Folgen einer traumatischen Bauchwandhernie und/oder Nerveneinklemmungssyndrom leiden. Ehe man aufwendige orthopädische Operation wie künstliche Hüfte oder Wirbelsäulenverblockung in Erwägung zieht, ist es empfehlenswert vorher eine traumatische Bauchwandhernie, Sportlerhernie, oder verdeckte (occult) Hernie auszuschließen. Eine operative Behandlung der Hernie sollte in diesem Fall erfolgen, um dem Patienten einen Verlust an Lebensqualität und um eine Verschlechterung mit Ausweitung auf andere Bereiche der Körpers, z.B., Hüftkopfveränderung, Veränderung der Lendenwirbelsäule, zu ersparen. Man sollte sich auf keinen Fall von den akuten Beschwerden einer LWS-Fraktur abbringen lassen, nach weiteren Ursachen von Schmerzen zu suchen.

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TRAUMATIC ABDOMINAL WALL HERNIA DIAGNOSED 14 YEARS AFTER A BAD FALL WITH LUMBAR SPINE FRACTURE

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Abstract

Traumatic abdominal wall hernias have significant implications for patients and insurance companies, especially when not been discovered at the time of trauma. We present the case of a gardener who sustained a bad fall during work in 1994 with immediate admission to the hospital for treatment. A fracture of the second lumbar spine body has been diagnosed and stabilized operatively. Postoperative computer tomography and magnetic resonance examinations demonstrated correct healing of the fracture. Neither the pain in the sacral spine, the left leg and left lower abdominal wall nor the sudden pain attacks in the groins with preference of the left groin stopped. Different neurologists considered as cause of the unchanged pain in the lower abdomen and left leg a radiculopathy in the lumbar spine. As a result of the neurological assessment the patient was operated in the lumbar spine (fixation of the fourth and fifth body) in a different hospital in 2007, unfortunately without elimination of the pain and no change of the neurological defects. The complaints increased to an extent that the patient was unable to drive a car, climb stairs or walk a longer distance. In 2008, when he was examined by the rheumatologist and internal medicine specialist, Prof. Dr. Ursula Gresser, in the Praxisklinik Sauerlach, the diagnosis of a traumatic abdominal wall hernia and isolated nerve compression syndrome was made. Prof. Gresser referred the patient to my hernia centre for surgical treatment. The intraoperative findings and histological examination of tissue were consistent with this diagnosis. The difficult meticulous repair of the 14 years old massive defects of the several layers of the abdominal wall and compression of nerves, when crossing these layers, has been made possible in a time demanding open approach with special care for the viable tissue and anatomy. Immediately after the operation the patient had no longer pain in the sacral spine, with a massive decline of pain level in the remaining areas. Without any further pain medication the patient is now able to climb stairs, walk longer distances and drive his car.

Conclusion: Patients suffering from pain and neurological alterations in the lower abdomen, groins and legs, with or without known trauma, may have a traumatic abdominal wall hernia and nerve compression syndrome. Before planning extensive orthopaedic operations in spine and hip, it is rewarding to exclude other causes, e.g., Sportsman hernia, traumatic hernia or occult hernia. A treatment of the hernia is absolutely necessary to avoid loss of quality of life for the patient and further detrimental development to the patient, e.g., destruction of the head of the femur, deterioration of the respiratory activity and lordosis of the spine. One should not get distracted by evident fractures in the spine to look for other causes of pain.

INTRODUCTION

Abdominal wall hernias are usually regarded as a hereditarily conditional illness caused by connective tissue weakness (Koch et al. 2000). Traumatic abdominal wall hernias have been rarely recognized before the general introduction of computer tomography and ultrasound (Faro et al. 1990; Brenneman et al. 1995; Burt et al. 2004). Some authors believe that with modern diagnostic equipment there may be an increased number of diagnosed occult traumatic abdominal wall hernias (Hickey et al. 2002; Kileen et al. 2000). In cases without direct blunt abdominal trauma it may happen that a traumatic abdominal wall hernia is not discovered (Suleiman and Johnston 2001). This may then be a burden for the patient who suffers from the consequences that the diagnosis of the traumatic abdominal wall hernia is delayed and the insurance does not acknowledge the hernia as a result of the trauma.

Several factors are required to recognize the traumatic abdominal hernia as a result of work related trauma (Koch et al. 2000).

We present the case of a patient who sustained a deceleration injury in 1994 by a fall from a car port during work resulting in a fracture of the second lumbar vertebral body and a delay of 14 years in diagnosis of a traumatic abdominal wall hernia.

CASE REPORT

The patient has been backward fallen from a carport roof during work as gardener at the age of 45 years in November 1994; he immediately has been taken to the hospital with the emergency doctor. An unstable compression fracture of the second loin vertebral body stood out in the x-rays and tomography of the lumbar spine. The implantation of a Fixateur interne was carried out in November 1994, the ventral bonechip blocking and laminectomy in December 1994. After the accident and the uncomplicated operation of the second lumbar vertebral body fracture the patient complained of constant pain in the left lower abdomen radiating in the left leg (Table 1).

Table 1. Follow-up after the trauma and operation of the second lumbar vertebral body.

Date	Specialty	Diagnosis or treatment
November 94	Surgery department 1	Diagnosis of a compression fracture of the second lumbar vertebral body after a fall from a car port that same day.
November 94	Surgery department 2	Uncomplicated operative stabilization with a fixateur interne and laminectomy
November 94	Neurologist 1	Radicular lesion in L4-S1 left side with paresthesia L5-S1
December 94	Surgery department 2	Ventral blocking in L1-L2
1994-1996	Surgery department 2	Constant pain in the lumbar spine, radiating to the sacral spine, left leg and groin with sensitivity disturbances. Physiotherapy, medical pain treatment, pain clinic.
May 95	Neurologist 2	No sign of polyneuropathy, but multiradicular chronic neurogenic alteration left side more than the right side L3-S1, active denervation in the Rectus femoris muscle
October 95	Surgery department 2	Removal of the fixateur interne
1995-1996	Consultant of the insurance	Back pain could be caused by arthrotic changes in the lumbar spine, psychological disturbance or by instability in L1-2. In case of a radicular pain syndrome in L4-S1 this may not be related to the trauma.
January 96	Neurologist 3	Pain during walking, numbness left foot to left knee, painful attacks in both groins
Januar 96	Neurologist 4	Suspected spinal stenosis; unclear pain syndrome L5-S1
February 96	MRT	No spinal stenosis, no prolapse of intervertebral disc, no instability in L1-2
1996	Pain clinic	Orthopaedic shoe, walk and gait training,
March 96	Neurologist 3	Left sided radicular pain syndrome L4-L5-S1
September 96	Surgery department 3 Special trauma clinic	Expert report: no instability in L1-2, pain in the lumbar area caused by a bend in the spine with consecutive malposition and strain in the spine muscles
September 96	Neurologist 4 Special trauma clinic	Expert report: radicular damage L3-S1; trauma related alterations are sensitivity disturbance left leg with pain radiating into the left leg
August 07	Surgery department 2	Deterioration of the existing pain status in the left leg with weakness of the left leg. Patient is transferred to a different clinic for operation.
August 07	MRT	Hypertrophic spondylarthrosis with spinal stenosis in L 3-4 and L 4-5
September 07	Neurologist 5	Radicular lesion in L2-S1 both sides: all pain of the patient is likely caused by these lesions
September 07	Surgery department 5	Dorsal laminectomy and decompression with stabilization L4-5
October 07	Surgery department 5	Ventral spondylodesis L4-5
October 07	Surgery department 5	Persisting pain despite correct radiological status of the operated area
January 08	Neurologist 5	Pain status (clinical examination and electromyography) as before the last operation with lesions in the segments L2-3 and L5-S1 left side, only improvement in the iliopsoas muscle right side (L2-3)
June 08	Internal medicine and rheumatologist – Sauerlach	The patient no longer can walk a longer distance, is unable to climb stairs, cannot disengage the clutch when driving, and has strong pain in the sacral spine. Pain in the left lower abdomen and groin on pressure. Diagnosis: Traumatic abdominal wall hernia with isolated nerve compression
June 08	Ambulatory surgery clinic – Sauerlach	Operative repair of traumatic abdominal wall hernia left lower abdomen and partial nerve resection.
June 08	Pathology	Nerve with scared fibrosis, tissue fibrosis, irregular structure of fascia of the abdominal wall – findings are compatible with traumatic abdominal wall hernia and nerve compression
June 08 – September 08	Ambulatory surgery clinic – Sauerlach	Patient is able to climb stairs, disengage the clutch when driving a car, can walk again and has lost the pain in the sacral spine immediately after the operation. The pain has lost 50% of its intensity on a visual analogue scale within two months with further constant decline without any medical pain treatment. Ongoing physiotherapy to correct malfunction of muscles, tendons, joints caused by delayed diagnosis of traumatic abdominal hernia.

Several neurological and neurophysiologic examinations by different neurologists took place in 1994 to 1996. The neurologists considered the pain to be caused by a chronic neurogenic alteration in L3-L5-S1 (radicular pain syndrome). In 1996, a spinal stenosis as result of the vertebral fracture could be excluded by magnetic resonance imaging, which also demonstrated consolidation of the second vertebral body after operative reconstruction.

Parallel the patient has been treated in a special pain clinic in 1995 and 1996: physical therapy, pain mastering therapy, learning of relaxation methods, attitude training, walk training, temporary medical therapy of the neuropathy and a rise of the outer edge of the left prefoot was undertaken because of a pathological rolling movement of the left foot and limping of the left leg. All treatment remained unsuccessful. It is noteworthy that the case records contained on several occasions notes of sudden pain attacks especially in the left groin in 1996.

In September 1996, the bended lumbar spine causing a malposition and increased strain on the spine muscles has been acknowledged to be cause of pain in the lumbar spine according to a surgical expert report. There was no sign of instability in the first and second lumbar vertebral body. With regard to the pain radiating to the left leg the surgical expert referred to a neurological expert report done in the September 1996, who considered the pain to be caused by a radicular damage at the level L3-S1 left side. As consequence of this assessment the sensitivity disturbances in the left leg and the patient's pain radiating to the left leg were recognized to be a result of the trauma (1994) by the insurance company.

In 2007, the patient felt a deterioration of symptoms. The neurologists considered a radiculopathy in the lumbar spine in the segments L2-S1 to be the cause of the unchanged pain in the lower abdomen and left leg. As a result of the neurological assessment an operative fixation of the fourth and fifth lumbar vertebral body was performed in a different hospital in 2007. Unfortunately for the patient the pain and the neurological defects remained unchanged.

As the complaints increased to an extent that he was unable to drive a car, climb stairs or walk a longer distance, the patient contacted Prof. Dr. Ursula Gresser, University of Munich and specialist for Internal Medicine and Rheumatology, at the Praxisklinik Sauerlach in 2008. He complained of constant pain in the sacral spine, left lower abdomen, radiating to the groin and left leg. When turning over in the bed during sleep he got awake by the pain every time. The strength in the left leg declined increasingly so that he could hardly climb stairs, drive a car or walk a longer distance. At the examination a muscular atrophy of the left leg stood out. There was a clear pressure pain in the left groin and left lower abdomen, which continued to radiate in the leg during bending of the hip. The patient could hardly stand on the left leg. Mobility in hip and knee were uneventful. The diagnosis was: abdominal wall hernia and nerve compression syndrome as a result of the trauma in 1994.

The patient was then referred to the surgeon specialist for hernia where the diagnosis was confirmed. A familiar predisposition for hernia and preexisting hernia before the traumatic incident were ruled out.

The intraoperative findings and histological examination of tissue were consistent with this diagnosis. The difficult meticulous repair of the 14 years old massive defects of the several layers of the abdominal wall and compression of nerves, when crossing these layers, has been made possible in a time demanding open approach with mesh, a special care for the viable tissue and the respect for anatomy. Immediately after the operation the patient had no longer pain in the sacral spine, with a massive decline of pain level in the remaining areas. Without any further pain medication the patient is now able to climb stairs, walk longer distances and drive his car.

Discussion

The acknowledgment of inguinal hernia or abdominal wall hernia as result of a work related trauma is a rare incident (Koch et al. 2000). This is mainly due to the fact that abdominal wall hernias are in most instances caused by a hereditary preexisting weakness of the connective tissue (Mollowitz 1993). Since the first description by Selby in 1906, there are not many reports on traumatic abdominal wall hernias (Brett et al. 2008). Although several investigators were unable to find an association to work related injuries in most abdominal wall hernias (Pathak und Poston 2006; Smith et al. (1996) they conceded that it may possible that traumatic incidents were not recorded by the treating physicians when contacted for a hernia probably due to a disregard of the significance of such an incidence (Schofield 2000; Pathak and Poston 2006).

There are different ways to describe a hernia in German and English literature which may have an impact on the assessment of traumatic work related abdominal wall hernia. In their list of prerequisites for acknowledgment as work related traumatic abdominal wall hernia Koch et al. (2000) used the definition of a hernia as reported by Saegesser in 1972: the hernia consists of a protrusion of the peritoneum in or through a defect in the abdominal wall. According to Saegesser the true hernia always contains a hernia sac of peritoneum with contents. Whereas German literature does not cover the subject intensively, there are quite a few reports of different types of abdominal wall hernias in the English literature (Lane et al. 2003), including handlebar hernia (Kubalak 1994). According to Brenneman et al. (1995) a traumatic hernia is defined as a full-thickness disruption of the abdominal wall musculature/fascia. There should be a timely association to a trauma – without penetration of the skin or preexisting hernia – but not necessarily a hernia sac of peritoneum (Damschen et al. 1994).

The general accepted definitions for a traumatic abdominal wall hernia are according to Clain (1964): immediate occurrence of the hernia after the trauma, no penetration of the skin, pain in the involved area, consultation of a doctor within 24 hours, and no visible preexisting hernia. Malangoni and Condon (1983) added: missing of a peritoneal sac. These criteria were accepted in further studies on traumatic abdominal

wall hernias (Pathak und Poston 2006; Smith et al. 1996; Schofield 2000). However, it is noteworthy to say that all these criteria may not be demonstrated anytime (Brett et al. 2008).

Wood (1988) classified the traumatic abdominal wall hernias in three types:

1. High-energy trauma caused by motor vehicle accident or fall with an extended defect of the fascia and often coexisting injuries to intraabdominal organs in relation to the hernia
2. low-energy trauma, e.g., handlebar hernia
3. Fall or deceleration injury with an intraabdominal interior hernia.

An important criterion of a traumatic abdominal hernia is that the patient was forced to stop work and seeks medical consultation within 24 hours after the traumatic incident (Koch et al. 2000). The patient in the current presentation fulfilled the necessary criteria, although the fracture of the second lumbar vertebral body overshadowed all other complaints. The delay of diagnosis has been observed in other cases of traumatic abdominal wall hernias. Netto et al. reported that an abdominal wall hernia became symptomatic eight months after the trauma (Netto et al. 2006). Del Frari and colleagues treated a left sided abdominal wall hernia eight months after rupture of the symphysis (Del Frari et al. 2008). There is a historic report of a traumatic Sphighelian hernia which has been discovered by the patient and proven intraoperatively eight months after a direct trauma to the abdominal wall (Brandtner 1933).

It is known that the diagnosis of a traumatic abdominal wall hernia is not always easy. In case of hematoma or a direct trauma to the involved area this may help to establish the diagnosis (Koch et al. 2000). Symptoms and complaints could be unspecific and may vary (Singh et al. 2004). Chronic pain related to abdominal wall injuries may deteriorate in case of sudden and twisting movements or even – as in the presented case – when rolling over in the bed (Williams und Foster 1995). It seems reasonable to consider the abdominal wall as cause of pain in case of chronic pain, which is not associated with eating or bowel function, but with change of posture (Suleiman und Johnston 2001). A painful trigger point in the abdominal wall radiating diffuse may be caused by an irritation of a sensitive superficial nerve or a myofascial irritation (Suleiman und Johnston 2001). This painful trigger reaction in the area of the inguinal ligament has been observed in this patient early after the trauma. The patient himself recognized sudden sharp painful attacks, especially in the left groin. It remains unclear why these complaints were not further investigated.

Yet, not all patients may have clinical signs of a traumatic abdominal wall hernia. Only 30% of patients with a traumatic abdominal wall hernia showed clinical signs in a recent study; diagnosis of abdominal wall hernia was made possible by computer tomography (Netto et al. 2006). Koch et al. (2000) postulated that in unclear situations the patient should be examined by sonography, computer tomography or magnetic resonance imaging which may demonstrate bleeding or hematoma in the abdominal wall. The authors considered a laceration of the hernia sac as a definite prove for the existence of a traumatic abdominal wall hernia, which is in contrast to the Anglo-American literature (Malangoni and Condon 1983).

Pathak and Poston (2006) even considered the demonstration of a hernia sac as possible disposition for hernia formation in that patient, which supports the demand to study the pretrauma case records by Schofield (2000). In the presented case the patient had no recorded suspected inguinal or abdominal wall hernia within the years before the trauma according to the health insurance company.

Computer tomography and magnetic resonance imaging may be useful tools for diagnosis of abdominal wall hernia (Rose et al. 1994), but not always in acute situations (Koch et al. 2000), which may explain why the traumatic abdominal wall hernia has not been discovered in the presented patient. Another important aspect is that the clinician should inform the radiologist of a suspected traumatic abdominal wall hernia as it is not unusual that these defects were not described in the radiological reports (Suleiman und Johnston 2001). Even better would have been an ultrasound examination of the abdominal wall which is associated with a 100% sensitivity and specificity provided the examiner is experienced (Robinson et al. 2006; Bradley et al. 2003). There are no case records available demonstrating sonographic examinations of the abdominal wall in the presented patient.

Although some authors (Koch et al. 2000) emphasize the intraoperative visible rupture of the peritoneum as a clear sign of a traumatic hernia, it seems reasonable to expect these signs in large and acute defects in case of clinically evident abdominal wall hernia. In less spectacular cases the typical findings of a traumatic abdominal wall hernia may be fibrotic adhesions and callused, tough scarred connective tissue around the defect of the abdominal wall (Brandtner 1933) and no contents in the defect of abdominal wall (Netto et al. 2006). The intraoperative status of the tissue in the area of the defect in the presented patient could be described in similar words.

Koch et al. (2000) demanded that the histological examination of tissue is performed and demonstrates signs of bleeding with fibrin exudation in acute cases, or fibrosis. The histological report of the tissue taken out during the operation of the presented patient included scared fibrosis with subepineural and intrafascicular myxoid-degenerative oedema as well as fat tissue with focal fibrosis and fascia with irregular fascia structure including nerves surrounded by fibrotic tissue and supported the case of a traumatic abdominal wall hernia and nerve compression syndrome.

The trauma should be strong enough and have an effect directly to the abdominal wall to be suitable to cause such an injury of the abdominal wall; this is usually caused by a direct biomechanical force which may be perforating or blunt. Hurting oneself from lifting too heavy does not suffice for the appreciation of a traumatic abdominal wall hernia (Koch et al. 2000). However, the development of a traumatic abdominal wall hernia is not always associated with a direct im-

pact to the abdominal wall. There are different types of accidents causing abdominal wall hernia, e.g., motor vehicle accident, motor cycle accidents and falls (Lane et al. 2003; Wood et al. 1988). The impact of the trauma may be direct or indirect to the abdominal wall. Netto and colleagues (2006) demonstrated a strong association of lumbar fracture and the type of trauma, e.g., a fall, with the traumatic abdominal wall hernia, caused by an indirect impact of trauma. Indirect tension forces may be responsible for rupture of the attachment of muscles to the bone (Ganchi and Orgill 1996; Del Frari et al. 2008), which is selfexplanatory when looking to the anatomical relations. The transversus abdominis and the iliopsoas muscles, both important for the abdominal wall and posture, have a connection to the lumbar spine (Flament 2006). The obliquus externus muscle approaches the thoracic cage to the pelvis and is important for expiration (Flament 2006). The defect can be remote to the point of maximum impact of the trauma (Nast-Kolb et al. 1998) which may explain why the diagnosis of the traumatic abdominal wall hernia has been delayed (Martinez et al. 1976).

However, it is known that the preferred localization of traumatic abdominal wall hernias is the left lower abdominal wall as is the case in the presented patient (Damschen et al. 1994).

The patient presented in this reports always complained of constant pain in the lower left abdomen and groin since the day of trauma, which has been caused by radiculopathy in the levels of L3-S1 in the lumbar spine according to different neurological assessments. But the sensitivity in the area of pain in this patient is provided by the anterior branches of the first lumbar nerve via the iliohypogastric and ilioinguinal nerve (Flament 2006). Mummenthaler (1998) described a compression syndrome of the ilioinguinal nerve with a mechanical irritation of the nerve in the plane of the transversus abdominis and obliquus internus muscles, which may cause pain in the lumbosacral spine and groins together with a painful restriction of motion in the hip with a change in walking. We have seen several patients with abdominal wall hernias and pain in the sacral spine, which was no longer existent after hernia repair. Mummenthaler recommended a local infiltration or partial resection of the involved nerves in case of persistent pain. We have demonstrated that this is a successful treatment of chronic groin pain in a young sportive patient who developed this pain after a sportive contest (Holzheimer and Gresser 2007). The decision to use mesh for hernia repair remains an individual choice based on the intraoperative findings (Choi et al. 2007). The repair of the abdominal wall defect and treatment of chronic pain, however, is indispensable to avoid serious disadvantages and the loss of quality of life for the patient. Mummenthaler (1998) reported a radiological verified destruction of the femur head in such a condition. The weakness of the abdominal wall may lead to deterioration of the respiratory function (Argenta et al. 1985) or to an impairment of the muscle function stabilizing the spine followed by massive lordosis and pain in spine as was the case in this presented patient (Flament and Rices 1998).

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