Acquired Neurogenic Abdominal Wall Weakness Simulating Abdominal Hernia

Anat Kesler MD[1], Ronit Galili-Mosberg MD[2] and Natan Gadoth MD[1]

[1] Department of Neurology, Meir General Hospital, Kfar Saba, Israel
[2] Department of Neurology, Rabin Medical Center (Beilinson Campus), Petah Tiqva, Israel
Affiliated to Sackler Faculty of Medicine, Tel Aviv University, Ramat Aviv, Israel

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Abdominal hernia is the result of a defect in the supporting structures through which a contained organ or tissue may protrude [1]. Damage to the intercostal or upper lumbar nerves, which supply the abdominal wall musculature, may mimic a hernia by its appearance as an abdominal wall bulging. We describe four patients in whom abdominal wall bulging appeared after acquired denervation of the abdominal wall. In three of the cases, surgical repair of "an abdominal hernia" was offered but deferred, and the "bulge" subsequently disappeared.

Patient Descriptions

Patient 1

A 58 year old healthy female presented with lower extremity weakness that had been slowly progressing over the preceding 2 years. The weakness was associated with numbness and tingling in the left thigh radiating to the leg and later to the foot.

About six months prior to admission, she experienced episodes of knee quivering, sudden falls and gait difficulties. On admission to the department of neurology, general physical examination was normal. Neurologic evaluation disclosed moderate spastic paraparesis with a sensory level to touch, pain and temperature, but intact vibration and position sense at the level of T11. Thoracic spine computerized tomography revealed an intradural, extramedullary hyperdense mass at the T9–T10 level that was thought to represent a meningioma. The histopathologically confirmed meningioma was completely resected through a T8–T10 laminectomy that was followed by a marked and rapid postoperative subjective and objective neurologic improvement. About 4 weeks after discharge she noticed a slow-growing painless "eggplant-size" bulge in the right abdomen, which became more prominent when she sat up or rose from the supine position. Abdominal ultrasound ordered by the family physician was normal. On consulting the neurosurgeon who had performed the laminectomy she was told that the "bulge" was unrelated to the recent spinal surgery. Two general surgeons diagnosed an abdominal hernia and offered surgical repair. However, neurologic follow-up at our outpatient clinic led to the conclusion that the localized abdominal wall weakness was secondary to segmental denervation of the abdominal wall muscles acquired during surgery. During the next 3 months, the "bulge" spontaneously decreased in size and then disappeared. At the latest follow-up visit, she reported being completely recovered and had resumed a normal life.

Patient 2

An 11 month old Bedouin male infant was admitted for severe dehydration, fever, vomiting and diarrhea. His parents reported that he had been in perfect health until the age of 6 months when he suddenly became paralyzed. They refrained from consulting a doctor at that time. On admission, the infant was severely dehydrated and intravenous replacement therapy was immediately initiated. There was a soft and painless "ballooning" of the right abdominal wall, without abdominal organomegaly or palpated masses. Neurologic examination disclosed marked asymmetric flaccid areflectic quadriparesis with intact cranial nerve functions, sensation, sphincter tone and social development. He had not been immunized against poliomyelitis, and the diagnosis of paralytic poliomyelitis was confirmed by the appropriate serologic tests. Following an uneventful recovery from his acute illness the child was followed for a number of years and no change in abdominal wall shape was noted. The parents refused to give permission for an electromyographic study.

Patient 3

This 82 year old male had suffered from bronchial asthma for several years and was using steroids intermittently. He presented to his family physician for acute back pain, which was resistant to adequate doses of non-steroidal anti-inflammatory drugs. Ten days later he noticed a vesicular rash at the site of the most intense pain, which corresponded to T9–12 left dorsal dermatomes. A clinical diagnosis of herpes zoster was considered and treatment with oral acyclovir and local capsaicin was initiated but failed to prevent the development of severe post-herpetic neuralgia. About a month later, he noticed a bulge in the lower lateral abdomen on rising from a supine position. A surgeon recommended surgery for an abdominal hernia but the patient refused. Four months later, the bulge completely disappeared.

Patient 4

This 62 year old male has suffered from non-insulin-dependent diabetes mellitus for the last 11 years that was reasonably controlled with oral hypogylcemics. A week prior to his admission he experienced severe left-sided back pain radiating in a band form to the skin of the breast and somewhat below the nipple. The family physician suggested a diagnosis of herpes zoster and prescribed acyclovir. This did not affect the pain and when the "shingles" failed

to show he was admitted to the department of neurology. The physical examination was unremarkable. The neurologic examination was remarkable for "shinning" of both tibias, "glove and stocking" hypoesthesia, symmetric diminished tendon reflexes and a band of decreased tactile sensation with local allodynia corresponding to the T4–T8 dermatomes. The relevant laboratory data included fasting blood glucose of 145 mg/dl and mild hypercholesterolemia. There was no glycosuria or ketosis. The rest of the routine laboratory data were normal. Nerve conduction studies disclosed the presence of mild sensory-motor neuropathy compatible with demyelinative diabetic polyneuropathy. Needle electromyography of the left T6–T7 intercostal muscle disclosed increased insertional activity with fibrillation potentials and bizarre repetitive discharges compatible with acute denervation. This was not present on the right side. A lumbar puncture disclosed acellular clear fluid under normal opening pressure. The protein was 180 mg/dl and glucose 100 mg/dl. Amitriptyline 25 mg 3 times a day brought a dramatic relief of the pain with no change in the neurologic signs. He was readmitted to the department of surgery 3 weeks later due to a soft increasing bulge at the site of his previously painful area. The surgeons were assured by the neurology consultant that this was not a true hernia but the result of diabetic radiculitis. The bulge slowly disappeared during the following 3 months. Two years later he was seen again because of acute right femoral neuropathy.

Discussion

In contrast to regional abdominal wall bulging, a true abdominal hernia is the result of protrusion of some of the abdominal contents beyond the normal confines of the abdominal wall. It consists of the sac, the contents of the sac, and its coverings formed from the layers of the abdominal wall through which the sac protrudes.

Abdominal wall bulging due to segmental denervation is rare because of its rich innervation [2]. The anterior and lateral abdominal wall consists of fibers of external and internal oblique, transversus, rectus and pyramidalis muscles. The oblique and transversus muscles are innervated by the lower six thoracic nerves and the iliohypogastric and ilio-inguinal nerves. The lower six thoracic nerves also supply the rectus abdominis. The literature is rather limited in descriptions of segmental-acquired neurogenic abdominal wall weakness mimicking abdominal hernia.

There are only a few reports of abdominal wall bulging caused by diabetic truncal neuropathy. Similar to patient 4, Parry and Floberg [3] described two patients with long-standing type I diabetes who presented with segmental unilateral protrusion of the abdominal wall thought to be an abdominal hernia and which spontaneously disappeared after 2–4 months. Boulton et al. [4] reported one patient with abdominal wall bulging and sensory symptoms who underwent an extensive and invasive work-up to exclude intra-abdominal pathology. The sensory symptoms resolved within 2 months with no change in the extent of bulging. There were no other neuropathic manifestations. Additional reports state that this condition is underestimated and frequently overlooked [5–7].

Diabetic neurogenic abdominal wall bulging seems to be a self-limited condition with resolution within a few months. However, Read and colleagues [8] described a patient with diabetic truncal neuropathy in whom surgery was required for repair of the bulging. There is still disagreement about the pathophysiology of diabetic truncal neuropathy, although it is believed to be ischemic by analogy with other painful diabetic neuropathies. Even the exact site of the lesion is unclear, and monoradiculopathy, polyradiculopathy and mononeuropathy have all been suggested.

Billet et al. [9] described lower abdominal wall bulging in a patient with a prolapsed L1–L2 intervertebral disc. On surgery, the L1 root was decompressed and the bulging had almost disappeared 10 months later. This patient is similar to our patient 1 in whom damage to intercostal nerves was acquired during the resection of spinal meningioma.

Inflammatory polyradiculopathies rarely cause abdominal wall bulging. Healy and co-workers [10] reported on "abdominal wall herniation" due to herpes zoster of the T11–T12 roots that completely resolved after 6 months. Gottschau and Trojaborg [11] described a woman with herpes zoster of the T9–T11 dermatomes complicated by abdominal muscle paralysis. Eighteen months later, paralysis of the rectus abdominis was still present. The authors state that abdominal wall bulging due to herpes zoster radiculopathy is rare, between 0 and 6% of all cases.

Daffner et al. [12] described a patient with Lyme's disease whose major complaint was a dramatic abdominal distension. The extensive work-up for known causes of abdominal distension was unrevealing. Nerve conduction and electromyographic studies supported the tentative diagnosis of polyradiculoneuropathy as a manifestation of neuroborreliosis. Following a course of antibiotics the distension and discomfort gradually subsided. This presentation of Lyme's disease polyradiculoneuropathy is different from the typical symptoms of peripheral neuropathy in neuroborreliosis.

Coleman and Ingram [13] reported on a patient with unilateral abdominal wall bulging due to focal amyotrophy and weakness of abdominal musculature associated with an extensive syringomyelia in the lower cervical and upper thoracic cord. The bulging was attributed to asymmetric loss of anterior horn cells in the thoracic cord due to the expanding syringomyelic cyst. This atypical presentation of a syringomyelia was further investigated, and in muscle fibers overlying the abdominal bulging there was electromyographic evidence of acute denervation and partial reinnervation. This patient is somewhat similar to our patient 2 in whom the bulging was the result of paralytic poliomyelitis.

The present case reports and the few briefly reviewed here should increase awareness to these entities. The correct diagnosis using sound clinical judgment and intercostal as well as paraspinal muscle electromyography may avoid unnecessary surgery for this benign and usually self-limiting condition.

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Correspondence: Dr. N. Gadoth, Dept. of Neurology, Meir General Hospital, Kfar Saba 44281, Israel.
Phone: (972-9) 747-2153
Fax: (972-9) 746-1460
email: gadoth@post.tau.ac.il