receives fascial support from the transversalis fascia, the continuous fascial layer of the anterior abdominal wall that fuses with the peritoneum at the brim of the true pelvic cavity but then continues over the muscles of the pelvic floor as the parietal pelvic fascia. The piriformis muscle arises from the ventral aspect of the second, third, and fourth sacral vertebrae, and inserts at the posterior aspect of the greater trochanter after passing through the greater sciatic foramen. The coccygeus, which originates primarily from the coccyx, inserts at the ischial spine. The levator ani muscles originate from the pubis, lateral pelvic wall, ischial spine, and sacrum/coccyx to blend with the rectum and the vagina or prostate. Since all the muscles that comprise the pelvic floor are innervated by branches of S2-S5, they are all susceptible to denervation atrophy following sacrectomy.

Perineal hernias are uncommon and usually result from operations such as abdominoperineal resections of the rectum or pelvic exenterations for advanced pelvic malignancies; the incidence ranges from 1% to 3% following these operations. These hernias usually manifest within the first postoperative year with a painful mass in the perineum. Pearl specifically includes “removal of the coccyx as part to the operative procedure” as a possible contributing factor in the development of a postoperative perineal hernia although he provides no support for this statement.

Methods of repair of perineal herniae include primary suture repair, synthetic mesh repair, omental or mesenteric graft repair, or complex myocutaneous flap reconstructions. The choice of repair is predicated on the size of the defect, the presence of infection, the integrity of the local tissues, and knowledge of the anatomy. Our primary choice is the synthetic mesh repair for its ease of performance, applicability to wounds of almost any size, and low incidence of complications.

If Pearl’s concern is correct that resection of the coccyx does increase the risk of perineal herniation, why hasn’t sacrectomy led to previous reports of perineal hernia? We believe that this hernia probably occurs more often, if not universally, following this operation. Recognition of these hernias is difficult due to the large amount of overlying soft tissue in the buttock unless obstructive symptoms develop. Any pain that may arise from these hernias would be difficult to distinguish from postoperative arthritic discomfort.

Why this patient developed a hernia following sacrectomy is unknown. It is speculated that the postoperative bowel obstructions that could have resulted in markedly increased intra-abdominal pressures were contributing factors. This increased intra-abdominal pressure in turn may have exacerbated the weakness of the denervated pelvic floor musculature, leading to attenuation of the fascia in the area devoid of bony support.

Santora et al: If a large portion of the sacrum is excised, a perineal hernia to some degree would be inevitable. If only a small portion or the distal third were removed, it might be possible to approximate the perineal muscles and secure them to the region of the piriforms on each side to create a barrier to posterior displacement of the pelvic contents. In a case such as this or in cases in which a major part of the sacrum is removed, this may not be possible. Under such circumstances, the surgical team might consider prophylactic mesh placement to prevent the possibility of herniation.

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REFERENCES

EDITORIAL DISCUSSION
ORTHOPEDICS: If one encounters a tumor such as this, where resection of the sacrum is indicated, what steps, if any, should be taken during the initial resection procedure to prevent such a complication?

CLOSED RUPTURE OF THE FLEXOR DIGITORUM PROFUNDS TENDON IN THE PALM OF A NON-RHEUMATOID PATIENT
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A closed traumatic injury to the flexor tendons of the hand most commonly results in avulsion of the flexor digitorum profundus tendon from its insertion in the distal phalanx. The substance of the tendon or muscle generally is not the site of mechanical failure under load. Intratendinous ruptures in rheumatoid patients due to weakening by invasion of tenosynovium or by abrasion on arthritic spurs occasionally are seen, but intratendinous ruptures of flexor tendons in non-rheumatoid patients are unusual. They are inevitably associated with pathological
penetrating wound in his left hand. The normal cascade of his fingers at rest was altered by the extended posture of the little finger (Fig 1). He was unable to actively flex the distal interphalangeal joint of his little finger although he had full passive motion and good active extension. Active flexion of the proximal interphalangeal joint was weak but intact. The patient had mild tenderness to deep palpation at the base of the palm. Radiographic examination showed no evidence of fracture or dislocation. The neurovascular examination was normal.

The patient underwent surgery for exploration and repair of the flexor digitorum profundus tendon of the left small finger. Exploration at the distal insertion of the profundus tendon revealed an intact tendon at that level. Further exploration proximally in the palm demonstrated an intact flexor digitorum superficialis to the little finger, but an intratendinous rupture of the profundus tendon at the origin of the lumbrical muscle was evident (Fig 2). The distal stump of the tendon had some muscle fibers of the lumbrical still attached.

The transverse carpal ligament was released to better visualize the proximal tendon (Fig 3). The proximal stump had not retracted much due to the intact lumbrical muscle. No anatomic abnormalities could be discerned in the floor of the carpal canal or in the region of the rupture. There was no abundant tenosynovitis to suggest chronic inflammation, nor were there sharp bony irregularities that could have caused an attritional rupture. Histologic examination of the debrided tendon ends revealed no pathologic abnormality.

The tendon was repaired end-to-end with a modified Kessler stitch using 4-0 Dacron suture. A 6-0 nylon running epitenon suture also was used. The hand was immediately placed in a resting dorsal plaster splint. Limited passive range of motion exercises were begun with a therapist 5 days after surgery, and active motion was initiated 5 weeks later.

At his 4-month follow-up examination, he was able to actively flex the small finger, bringing the fingertip to the distal palmar crease. He had −15° to 90° of motion at the metacarpophalangeal joint, 0° to 90° at the proximal interphalangeal joint, and 0° to 60° of motion at the distal interphalangeal joint.

states including infection, previous injury, chronic repetitive injury, bony irregularities, or after a fracture.3

Folmar et al4 reviewed 12 flexor tendon ruptures in non-rheumatoid patients and found that all were associated with an underlying pathologic condition. Boyes et al3 in their report of 80 flexor tendon ruptures, found 3 intratendinous ruptures in the palm; only 2 out of the 3 were not associated with underlying pathology.

An intratendinous rupture of the flexor profundus of the small finger has been reported that was associated with an anomalous tendinous fusion between the deep flexor of the ring and small fingers.4 Only isolated cases of closed intratendinous ruptures of the flexor profundus tendon in the palm in patients with no causative pathology have been reported in the literature.5,7

This study reports a case of a closed injury causing rupture of the flexor digitorum profundus tendon to the small finger just distal to the origin of the lumbrical muscle in a patient with no predisposing factors.

**CASE REPORT**

An unemployed, 34-year-old, right-hand dominant man presented to the emergency room with the inability to actively flex the distal interphalangeal joint of his left small finger. Five days earlier, he had been involved in a motor vehicle accident. During the accident, the patient, who was driving, grabbed the door handle with his left hand as his body was thrown to the right. The weight of his body pulled his hand free of the door handle. He experienced immediate sharp pain in the ulnar side of his palm and soon afterward noted swelling in his hand. No other injuries were sustained.

Over the next 2 days, the pain and swelling subsided. However, he discovered that he could no longer actively flex the distal joint of the left small finger. He was in good health, with no significant past medical history and was not taking any medications.

On examination, there was no sign of a
DISCUSSION

Closed flexor tendon injuries most commonly occur when an extension force is applied to a finger while the flexor digitorum profundus muscle is maximally contracting. The profundus tendon is avulsed from its insertion in the distal phalanx. This is a relatively common injury, particularly in athletes, and the ring finger is the most frequently involved digit. McMaster demonstrated that the insertion is the weakest point in the musculotendinous unit. Therefore, the normal profundus tendon ruptures at its bony insertion and not in the substance of the tendon.

Luch and Madden noted that the tensile forces transmitted through the tendon exceed the shearing stress of the bone, and an avulsion fracture generally occurs. Leddy categorized the tendon avulsion based on the fragment size and the extent of retraction of the proximal stump.

Closed flexor tendon ruptures in non-rheumatoid patients are rare. James in 1949 and others subsequently reported flexor tendon ruptures secondary to Kienbock’s disease. Boyes et al. mention a case of flexor tendon ruptures due to a roughened hook of the hamate. Osteoarthritis of the pisotriquetral joint has been implicated in flexor tendon rupture. Scaphoid fracture nonunion has been reported as a cause of flexor tendon rupture. Scaphotrapezial osteoarthritis has been reported to result in flexor carpi radialis rupture.

Delayed rupture of flexor tendons due to a lunate fracture also has been known to occur. Ruptures secondary to hamate hook fractures also have been reported. A closed rupture of the flexor digitorum profundus tendon to the little finger was reported by de Roos and Zeeman in a patient who had an anomalous tendinous fusion of the deep flexors of the ring and little fingers up to the lumbral insertion.

Intratendinous flexor tendon rupture in patients without predisposing conditions is extremely rare. Boyes et al. were the first to mention flexor tendon rupture at the level of the palm in patients with no known underlying pathology. In their series of 80 cases of flexor tendon ruptures, 2 such cases were found. More recently, Kumar and James reported a case of a flexor digitorum profundus tendon rupture in the palm of an otherwise healthy patient. The patient was an active 44-year-old man working on a diesel engine who forcefully retracted his hand while the little finger was hooked in a small opening of the engine. The patient felt a snap in his palm as he withdrew his hand. At surgery, his profundus tendon was found to have ruptured at the origin of the lumbral muscle.

Imbriglia and Goldstein reported seven patients who had surgically demonstrated intratendinous ruptures of the flexor digitorum profundus tendon involving the little finger and who had no predisposing pathological conditions. Four of these ruptures occurred in the palm just distal to the lumbral origin. Two ruptures occurred distal to the A2 pulley, and one occurred in the carpal canal. All but one of these patients were employed in manual labor requiring repetitive motion combined with power grip on a routine basis. The authors noted that the ability of flexor tendons to elongate after being subjected to a constant force over time is extremely diminished.

In patients engaged in heavy manual work, flexor tendons may be less tolerant of a sudden traction force. The largest series of intratendinous flexor tendon ruptures in the palm was reported by Naam, who described 12 cases involving the little and ring fingers. He recommends primary repair in cases seen in the first 2 weeks after injury, interposition tendon grafts for late cases, and distal interphalangeal joint fusion as an alternative. He also noted that if the site of rupture is uncertain, the first incision should be made at the proximal interphalangeal joint level. If the tendon is intact at that location, the next incision should be in the palm.

Although rare, flexor tendon rupture at the level of the palm must be considered in patients who present with a history and examination suggestive of the more common “jersey finger.” Unlike the typical flexor digitorum profundus avulsion that most commonly occurs in the ring finger, intratendinous flexor tendon ruptures seem to occur exclusively in the small finger.

The patient described here had tenderness in his palm. However, cases of flexor tendon avulsion from the distal phalanx that retract into the palm can present with similar findings. Our patient reported that at the time of injury, he felt a sharp pain at the base of his palm rather than in his finger. This kind of history should heighten the surgeon’s suspicion that an intratendinous rupture may have occurred more proximally. In patients with no underlying pathology to predispose them to tendon ruptures, this is certainly an unusual occurrence.

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EDITORIAL DISCUSSION

ORTHOPEDICS: Can you explain why ruptures tend to occur in this area, i.e., distal to the lumbrical?

Yang et al: It is not clear why the tendon tends to rupture at the origin of the lumbrical. Possibly, this area of the flexor digitorum, which leaves on the hamate hook, represents a relatively avascular portion of the tendon. Perhaps the origin of the lumbrical muscle, where muscle fibers interdigitate with the longitudinally oriented fibers of the flexor tendon, is a mechanical weak point, the next weakest link after the flexor digitorum profundus insertion.

ORTHOPEDICS: What is the explanation for the weakness of flexion of the proximal interphalangeal joint? Could it be due to some form of increased lumbral tension?

Yang et al: The weakness of flexion at the proximal interphalangeal joint is certainly due to increased lumbral tension. The tension previously shared by the distal flexor digitorum profundus and the lumbral is entirely borne by the lumbral after rupture of the tendon. This increased tension on the intrinsics exerts a greater extension force on the proximal interphalangeal joint, making flexion more difficult.

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ELECTROMECHANICAL DISSOCIATION AND PARADOXICAL EMBOLIZATION DURING HIP SURGERY

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Pulmonary embolization of venous thrombi is a known complication of hip surgery. When 25% or more of the pulmonary vascular bed is occluded by emboli, a sudden increase in pulmonary artery and right ventricular pressure occurs. This may lead to hemodynamic decompensation and electromechanical dissociation (EMD). In addition, if right atrial pressure exceeds left atrial pressure, right-to-left shunting across a patent foramen ovale (PFO) may occur. This report describes what is believed to be the first case of a patient with large intracardiac thrombi and paradoxical embolization in progress through a patent foramen ovale during intramedullary nail placement of the left femoral head.

CASE REPORT

A 67-year-old man presented with debilitating pain in the left hip of 1 week's duration. The patient's past medical history was significant for hypertension and prostate carcinoma, for which he underwent bilateral orchiectomy 19 months prior to this presentation. He subsequently was treated with radiotherapy due to extensive bony involvement. At the time of his last radiotherapy treatment 2 months prior to admission, a bone scan revealed abnormal uptake in the left femur, suggesting active metastatic involvement. Lower extremity venous Doppler images were negative for deep venous thrombosis. A Richards reconstruction of the left hip with interlocking nail placement was planned prior to administering radiotherapy to the region. However, prior to the scheduled surgery, the patient was hospitalized with a 1-week history of debilitating pain in the left hip that was not relieved by analgesics.

While undergoing reaming of the femur, a mild drop in both arterial oxygen saturation (98% to 94%) and systolic blood pressure (160/70 to 120/60 mm Hg) were noted. These parameters were corrected by anesthesia intervention. The procedure continued, and after obtaining AP and lateral radiographs, a nail placement was attempted. However, the patient experienced sudden cardiovascular collapse, and the procedure was aborted due to inability to obtain a pulse or blood pressure. Cardiopulmonary resuscitation (CPR) was promptly initiated, and a transesophageal echocardiogram (TEE) was performed in the operating suite 15 to 20 minutes after the initiation of CPR. A four-chamber view demonstrated effective chest compressions with no pericardial effusion. Marked dilatation of the right atrial and ventricular chambers was noted. Complete cardiac standstill was evident with cessation of chest compression.

Several multilobulated, serpiginous masses floating freely in the right atrium, right ventricle, right ventricular outflow tract, and main pulmonary artery were demonstrated (Fig 1). One of these thrombi was clearly seen strad-