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Letters

Intrathoracic Extension of Retroperitoneal Disease: Is It Purely Lymphatic Spread or Do Some Potential Interfascial Pathways Exist?

The pictorial essay by Dr. Aizenstein et al. [1] about the spreading pathways of retroperitoneal diseases was well prepared, and the glossary and high-quality drawings address some of the controversial points on this subject. Our contribution relates to the pathways of transdiaphragmatic spread. We do not think the spreading of retroperitoneal diseases can be explained only by transpleural or transdiaphragmatic lymphatic communications. To our knowledge, no clinical or postmortem anatomic study supporting the transdiaphragmatic continuation of the retroperitoneal spaces with mediastinal or plural spaces has been reported; however, a limited number of case reports deal with this potential anatomic communication (through the interfascial planes) between these compartments [2, 3]. We welcome the author's opinions about direct transdiaphragmatic spread of retroperitoneal diseases, which would, in our view, make this pictorial essay complete.

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Reply

We appreciate the comments of Drs. Akpek and Ilgit concerning our pictorial essay [1]. In our essay, we discussed several pathways for spread of retroperitoneal disease, including the spread of retroperitoneal fluid within fascial planes (i.e., interfascial spread of disease). The concept of retroperitoneal interfascial disease spread derives from clinical CT findings and the work of Molmenti et al. [2], who showed that the retroperitoneal fascia is composed of laminated, potentially expansile fibrous sheaths that can serve as conduits for spread of fluid throughout the abdominal and pelvic retroperitoneum. In our article, we showed examples of retroperitoneal fluid extending both medially across the midline and inferiorly into the pelvis within these interfascial planes.

Drs. Akpek and Ilgıt augment our discussion of interfascial disease spread by suggesting that interfascial disease may occasionally extend superiorly into the mediastinum. Two clinical reports, including one by Akpek et al. [3], describe the mediastinal extension of retroperitoneal urinomas [3, 4]. The intrathoracic extension of retroperitoneal urine is uncommon and most often involves the pleural cavity rather than the mediastinum [4]. In these cases, the most likely mechanism for the passage of urine from the perirenal space into the pleural cavity is via transdiaphragmatic lymphatic channels. An analogous mode of metastatic disease spread—transdiaphragmatic lymphatic spread of bronchogenic carcinoma from the thorax into the perirenal space—was discussed in our article [1]. Although mediastinal extension of pancreatic fluid collection is well known, direct transdiaphragmatic extension of retroperitoneal urinoma into the mediastinum is rare.

The exact pathway for direct spread of retroperitoneal fluid into the mediastinum is not well understood. Molmenti et al. [2] performed detailed cadaveric retroperitoneal dissections and showed that the retromesenteric interfascial plane (i.e., the potential space between the anterior pararenal and perinephric spaces) may traverse the midline and extend inferiorly into the pelvic retroperitoneum. In addition, these same studies showed that the retromesenteric plane extends superiorly to the level of the dome of the diaphragm, posterior to the esophagus and phrenicoesophogeal ligament [2]. On the basis of these findings, we concur with Drs. Akpek and Ilgit that the most likely explanation for mediastinal, as opposed to pleural, extension of retroperitoneal fluid collections is via the interfascial planes. Specifically, we surmise that some clinical retroperitoneal fluid collections (e.g., urinomas or pancreatic fluid collections) spread superiorly within the retromesenteric interfascial plane to reach the undersurface of the diaphragm and gain access to the mediastinum via the aortic or esophageal hiatus.

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Helical CT Angiography in Gastrointestinal Bleeding

The article by Ettore et al. [1] on the usefulness of helical CT angiography for the diagnosis of gastrointestinal hemorrhage of obscure origin does not discuss the possibility of cessation of bleeding after injection of intraarterial iodinated contrast media.

The authors stated that after intraaortic injection of iodinated contrast media for helical CT angiography, all patients underwent conventional angiography. Though not clearly stated, we assume that the conventional angiographic studies were performed subsequently to avoid an additional invasive catheterization procedure. During the course of an angiographic study, the cessation of active gastrointestinal bleeding is not rare. Thus, the possibility of vasoconstriction and cessation of bleeding from an active site after contrast injection is important to keep in mind [2].

In a case of jejunal leiomyoma, the source of bleeding was revealed by conventional angiographic study, but a few minutes later the lack of identification of the site of bleeding prevented us from attempting any trial of embolization. Control angiographic and radionuclide studies during the next 36 hr confirmed the cessation of bleeding, and no bleeding was observed during the surgical excision of the tumor. Thus, helical CT angiography should not be considered more sensitive than conventional angiography for the two cases of ulcerative colitis and Meckel's diverticulitis in the study group of Ettore et al. [1], and the helical CT and conventional angiographic results cannot be based on this kind of methodology.

We appreciate the idea that helical CT can be a possible diagnostic tool for identification of gastrointestinal bleeding of unknown origin, but the methodology of the studies must be planned in a different manner.

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Reply

We are grateful to Drs. Dicle and Göktay for their comments and interest in our paper [1]. With regard to the moment of the conventional angiography, we stated in our paper that the catheterization procedure was performed for the helical CT angiography; immediately afterward, the patient was transferred to the angiographic unit, where iodinated contrast medium for conventional angiography was injected into the catheter that was still in place. In our cases of helical CT, the catheter is positioned in the aorta, which naturally has a large caliber and therefore is scarcely affected by an eventual vasoconstriction effect provoked by the contrast medium.

Naturally, we cannot comment on the personal experience of our colleagues; for any diagnostic procedure, the identification of a bleeding site is dependent on the presence of an active bleeding site, and interruption of hemorrhaging can be caused by various events, not necessarily the vasoconstriction effect of the contrast medium.

With regard to the question of the sensitivity of our method, the term "sensitivity" implies a statistical study with a randomized population. Our paper should be considered a preliminary report of an original diagnostic method, and our intention was not to present a comparative study with angiography.

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Needle Characteristics Related to Headaches After Myelograms

The incidence of post–dural puncture headaches after myelograms may be related to tip morphology, as noted recently by Prager et al. [1]. However, many other technical factors also affect the incidence of post–dural puncture headaches, including age and gender of patient, bevel position, approach (midline versus paramedian), needle size, and concentration of injected material [2]. Prager et al. made no mention of the bevel position of the Quincke tip needles (Becton-Dickinson, Franklin Lakes, NJ) when they entered the thecal sac. Our incidence of post–dural puncture headaches after myelograms was significantly reduced when our routine practice was changed to orient the bevel parallel to the longitudinal fibers of the dural sac [2]. Further, Prager et al. did not use control subjects to monitor the influences of gender, approach, or concentration of myelographic contrast material. Any of these influences could introduce significant bias into their results given their small number of patients. A multivariate analysis of data would have been more appropriate and helpful.

Reducing the incidence of post–dural puncture headaches after myelography requires careful attention to technique. Needle-tip morphology may be important as Prager et al. [1] suggest, but needle size is also critically important as is well known from the anesthesia literature [2]. The 22-gauge needles that Prager et al. used have a significantly greater incidence of post–dural puncture headaches associated with them than do 25-gauge needles [2]. Twenty-five-gauge pencil-point needles can be used for myelography, even when using viscous (300 mg I/ml) contrast media [3]. We routinely use 25-gauge needles for more than 90% of our myelograms and have a 100% technical success rate in patients with nonfused spines. Although 25-gauge needle-tip morphology may play a role in the incidence of post–dural puncture headaches in patients who have been given anesthesia [4], the question that needs to be answered is whether 25-gauge pencil-point needles reduce the incidence of post–dural puncture headaches after myelography sufficiently to justify their increased costs when data are corrected for all confounding variables.

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Reply

We appreciate the comments by Drs. Suojanen and Punzak on our recent article regarding needle type and spinal headaches [1]. We agree that many factors may affect the incidence of spinal headaches after myelography; however, the purpose of our study was to study the influence of needle-tip morphology. Some of these other factors, also mentioned and controlled in our study, include age, gender, and needle size. Although we did not discuss concentration of contrast material, different concentrations were used for different levels. We noted no significant difference in the incidence of spinal headaches regardless of the level studied. Furthermore, one neuroradiologist did perform lumbar punctures with the bevel parallel to the longitudinal fibers of the dural sac, whereas the other neuroradiologist did not. No significant difference in the incidence of headaches was found between the two examiners.

We recognize the importance of needle size as discussed in our paper. In addition, we agree that further studies including 25-gauge needles with pencil-point tips versus cutting edges may add to our understanding of factors that affect the incidence of headaches after myelography.

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Color Doppler Sonography of Renal Artery Aneurysm

We read with interest the recent paper "Renal Vein Aneurysm: Diagnosis with Color Doppler Sonography" [1]. We have encountered a similar case with some unique features in which color Doppler sonography was also instrumental in making the diagnosis.

A 60-year-old woman was referred to our department for new-onset microhematuria.



Fig. 1.—60-year-old woman with renal artery aneurysm who presented with new-onset microhematuria.
A, Excretory urogram reveals multilobed cystic mass within left kidney without impairment of function.
B, CT scan shows contrast-enhancing renal mass.
C, Color Doppler sonogram shows flow throughout mass.

An excretory urogram was ordered. The examination revealed several large calcified masses within or next to the left kidney. The masses did not displace the kidney nor interfere with its excretory function (Fig. 1A). A CT scan with contrast enhancement was ordered as the next test. The initial interpretation was that of a densely calcified, enhancing mass of the left kidney (Fig. 1B). The primary diagnosis was a partially calcified renal cell carcinoma, although infectious causes such as tuberculosis or ecchinococcal cyst were considered. Finally, the possibility of a large distal renal artery aneurysm was suggested, and sonography was ordered just before surgery.

Gray-scale imaging was limited because of the dense calcification of much of the hilar-based mass. However, color Doppler sonography was used by imaging around the calcific shadowing. Color Doppler sonography showed that the entire mass was vascular (Fig. 1C), and duplex Doppler sonography revealed the arterial waveform morphology.

The kidney was removed at surgery, and a 7.2-cm, multilobed intraparenchymal renal artery aneurysm was found. The aneurysm extended from the renal hilum through the parenchyma, with cystic cortical protrusions. As predicted, the aneurysm was extensively calcified.

Renal artery aneurysms are uncommon, with an incidence of between 0.09% and 0.3% of the population [2]. Most are less than 3 cm in size and are found during angiography for hypertension or hematuria [3]. The cause includes arteriosclerosis, fibromuscular hyperplasia, and neurofibromatosis [2]. Thirty percent of patients have multiple aneurysms in the same kidney, and 20% of aneurysms are bilateral [2]. Renal pseudoaneurysms may occur after trauma or iatrogenic injury and may present with delayed hematuria [4].

The most serious complication is rupture; therefore, elective repair or removal is indicated in cases in which the aneurysm is more than 1.5 cm in size [2]. Other indications for surgery include aneurysms that are symptomatic, cause hypertension, or may impair renal function because of thromboembolism.

This case reveals that a renal artery aneurysm can be quite large and can have a clinical presentation and imaging characteristics that are indistinguishable from those of renal cell carcinoma. In this instance, the addition of color and duplex Doppler sonography in the workup made a significant contribution toward making the diagnosis before surgery.

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The Importance of Being Where? Balancing the Perspective on Practice Coverage

In addressing the prospect of round-the-clock coverage of radiology practices, we have made the tacit assumption that such coverage is the right thing to do [1–3]. The arguments for it have become familiar: competitive pres-

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sure to "outcover" the practice down the street, the need to be perceived as a proactive rather than a passive player in the patient care team, turf protection to keep studies away from nonradiologists. And if those seem too self-serving, improved patient care always provides an unassailable haven.

This new paradigm has spawned a novel work niche. The "night hawk" radiologist is a graveyard shift jack-of-all-trades employee who inherits the undesirable hours. Occasionally, I've questioned the propriety of offering such second-tier positions, because we would have scoffed at such an offer at the conclusion of our own training. Usually I receive a lesson on supply-and-demand theory and how it is "just business." I'm reminded of the scene from *The Godfather* in which rival mobsters gun down Don Corleone and then explain to his son that it was nothing personal, "just business."

An alternative to such hirelings is to foist additional work hours onto current staff. The implications of such a move really hit home when my wife phoned me at the hospital late one Saturday afternoon to say that our son (who has phenylketonuria and struggles with athletics) got the game winning hit in his Little League baseball game and was carried off the field on the players' shoulders. I was bitterly disappointed about not being there. How many more such moments would be missed with increased evening, night, or weekend duty plus compensatory daytime sleep?

Yes, we are practicing in the midst of a revolution. Yes, change is inevitable. But as we rush to protect wallet and turf, let us be cognizant that the change is not without significant personal cost. What are the long-term ramifications of two-tier radiology, in which practice owners work days and hired hands with no stake in the practice work nights? Alternatively, more hours at work for current staff members inevitably would mean less hours at home with family. How does one figure the bottom line on that transaction?

I don't know the right answer or even if a right answer exists. Ultimately, each radiologist must decide for himself or herself how irreplaceable hours will be spent. But at least let this choice be a decision, not a thoughtless, mob-following reflex that treats our time as a valueless commodity. Perhaps I'm guilty of retrothink in wondering whether this new paradigm comes with too high a price tag. I'm reminded of the insightful old caveat that no one on his or her deathbed ever laments, "If only I had spent more time at the office."

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Langerhans' Cell Histiocytosis of the Synovium

A 53-year-old woman with known Langerhans' cell histiocytosis (Hand-Schüller Christian type) presented with left shoulder pain. Several lytic lesions with sclerotic borders in the humeral head were evident on plain film (Fig. 2A). MR imaging showed the humeral head lesions to be of low signal intensity on T1-weighted images and high signal intensity on T2-weighted images. Marked bone marrow edema surrounding these cystic bone lesions was also noted. On T2-weighted images and short inversion time inversion recovery images, high signal intensity was evident (Fig. 2B) in the synovium of the shoulder joint, and thickening and high signal intensity were seen in the biceps tendon sheath, which showed intense enhancement after gadolinium administration (Fig. 2C). The portions of the humeral lesions that were more cystic appearing showed no enhancement.

At surgery, marked synovial proliferation and thickening of the biceps tendon sheath were noted. The cystic bony lesions in the humeral head were in a superficial location that was contiguous with the thickened synovium. Pathologic analysis of the synovium and biceps tendon sheath confirmed Langerhans' cell histiocytosis.

Langerhans' cell histiocytosis encompasses a spectrum of disease ranging from isolated bone involvement (eosinophilic granuloma) to disseminated fulminant disease (Leterrer-Siwe disease). Bone lesions are the most common manifestation of Hand-Schüller Christian disease, and extraosseous involvement, including the lungs, skin, thymus, and gastrointestinal tract, is common [1]. To our knowledge, this is the first report of synovial involvement in Langerhans' cell histiocytosis.

Soft-tissue involvement has been described in association with the bone lesions of Langerhans' cell histiocytosis, particularly in the early phase of the bone lesions [2]. This soft-tissue involvement may be ill-defined or masslike on T2-weighted MR images [3].

In the patient reported here, the operative findings were remarkable for the extent of synovial proliferation and involvement of the biceps tendon sheath and less remarkable for the bony lesions. These findings suggest that the primary process was a proliferative arthritis.

The MR imaging appearance in this patient showed some atypical features when compared with prior MR imaging reports of Langerhans' cell histiocytosis. Beltran et al. [3] reported diffuse ill-defined areas of signal hypointensity on T1-weighted images and hyperintensity on T2-weighted images. De Schepper et al. [4] showed diffuse enhancement of the bone lesions after gadolinium administration. Our case is atypical in that the bone lesions showed a central cystic component on T2-weighted images and lack of enhancement of this central portion of the bone lesions with gadolinium administration; these findings suggest that the bone lesions, rather than being primary Langerhans' cell histiocy-

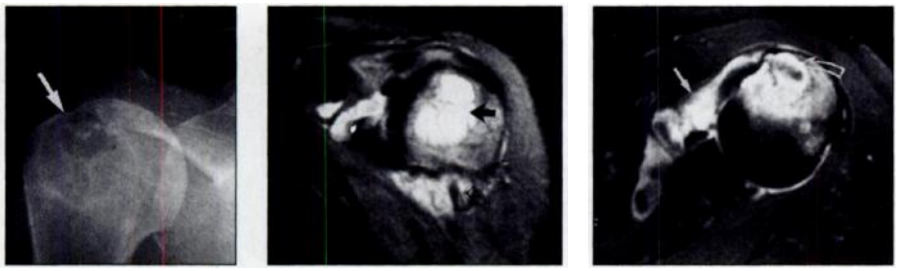


Fig. 2.—53-year-old woman with known Langerhans' cell histiocytosis (Hand-Schüller Christian type) who presented with left shoulder pain.
A, Plain film shows several lytic lesions with sclerotic margins in humeral head (*arrow*).
B, Short inversion time inversion recovery image (TR/TE, 3500/21) shows cystic lesions in humeral head (*solid arrow*) and thickening of biceps tendon sheath with fluid (*open arrow*).
C, Gadolinium-enhanced T1-weighted fat-saturated image (550/15) shows peripheral enhancement of cystic humeral lesions (*open arrow*) with enhancement of synovium (*solid arrow*).

tosis lesions, were in fact associated with the synovial process.

In summary, this patient with a protracted history of Hand-Schüller-Christian disease presented with shoulder pain related to a severe tenosynovitis of the shoulder joint and biceps tendon sheath, with bone lesions of the humeral head most likely attributable to the synovial proliferation.

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MR Imaging and CT Appearances of Aggressive Angiomyxoma

Aggressive angiomyxoma is a rare mesenchymal tumor involving primarily the female pelvis and perineal regions. Clinically, the tumor presents as a slow-growing mass or swelling in the vagina, labia, perineum, or scrotum. Histologically, the tumor is composed of stellate or spindle-shaped cells embedded in a loose myxoid matrix with scattered vessels of varied caliber. Most of the tumors have relatively sharp margins in some areas and infiltration into fat, fibrous tissue, or muscle in other areas. Local excision is the usual treatment. To our knowledge, no metastases or tumor-related deaths have been reported. Complete excision is often impossible because of a locally infiltrative nature, and local recurrence may occur [1, 2]. We report MR imaging and CT findings in two histologically proven cases.

Case 1 is a 67-year-old man who presented with prostatic hyperplasia. A pelvic sonogram revealed a homogeneously hypoechoic mass in the retrovesical fossa. CT showed a pelvic mass displacing surrounding visceral structures without apparent sign of invasion. The tumor extended caudally into the right ischiorectal fossa crossing the levator ani muscle. Before contrast enhancement, the tumor was almost homogeneous, with CT attenuation similar to that of water. After injection of contrast agent, the tumor showed gradual heterogeneous enhancement. On T2-weighted MR images, the tumor showed a mixed appearance of hyper- and hypointensity (Fig. 3A). On T1-weighted images, most of the tumor was hypointense in relation to muscle; after administration of gadopentetate dimeglumine, the tumor showed irregular enhancement. The low-signal-intensity components seen on T2-weighted images enhanced 1 min after injection of contrast agent, whereas the high-signal-intensity components seen on T2-weighted images showed gradual and steady enhancement 10 min after injection of contrast agent (Fig. 3B). A soft myxoid tumor was resected completely without evidence of invasion.

Case 2 is a 27-year-old woman who presented with a soft mass in the left vulva. Partial resection was performed, and a diagnosis of aggressive angiomyxoma was made. The tumor extended from the left ischiorectal fossa through the levator ani muscle into the peritoneal cavity, involving the rectum. The internal attenuation and enhancement patterns evident on CT and MR imaging were similar to those of case 1 (Fig. 4).

Aggressive angiomyxomas have been described as masses of low attenuation or soft-tissue attenuation on CT [3, 4]. One report described the tumor as cystic on sonography and CT [4]. Most tumors extended from the pelvic space into one of the ischiorectal fossae crossing the levator ani muscle. We did not find a description of the enhancement pattern on CT and MR imaging.

From MR imaging and CT findings, regions that enhanced immediately (which were of low signal intensity on T2-weighted MR images) probably represented angiomatous components, whereas regions that enhanced gradually (which were high signal intensity on T2-weighted images) probably represented myxoid components. Therefore, the typical study showed an irregular enhancement pattern after contrast administration. Care must be taken because findings

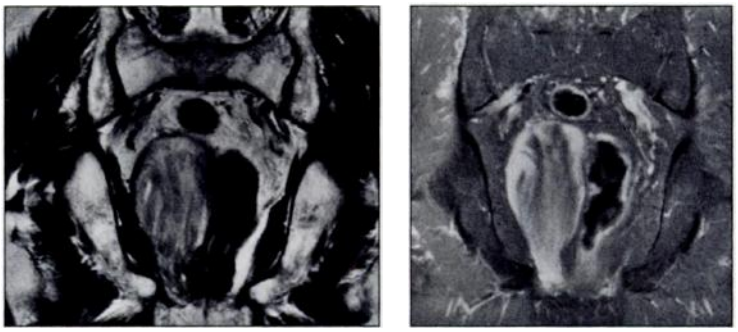


Fig. 3.—67-year-old man with prostatic hyperplasia.
A and **B**, Coronal T2-weighted spin-echo MR image (TR/TE, 4800/128) (**A**) and T1-weighted spin-echo MR image (845/12) with fat suppression after injection of gadopentetate dimeglumine (**B**) show tumor extending to ischiorectal fossa. Mixed appearance of hyper- and hypointensity, like marbled soft cream, is seen.

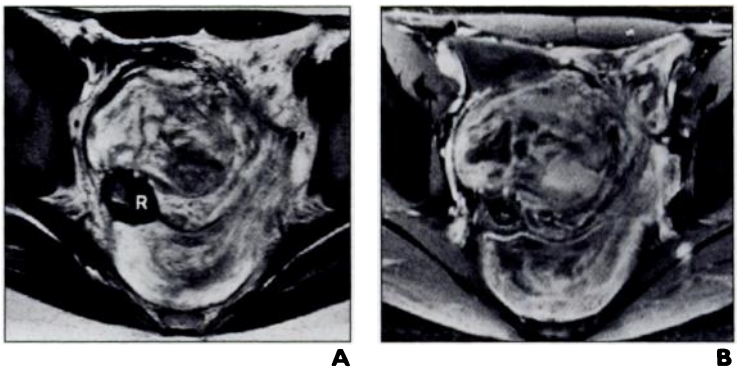


Fig. 4.—27-year-old woman with soft mass in left vulva.
A and **B**, Tumor, surrounding rectum (R, **A**), shows mixed appearance of hyper- and hypointensity on T2-weighted spin-echo MR image (4250/128) (**A**). On T1-weighted spin-echo MR image (570/6), low-intensity strands on T2-weighted image are enhanced 1 min after injection of gadopentetate dimeglumine (**B**), whereas other strands are enhanced 10 min after injection (not shown).

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similar to those of a cyst may be found on unenhanced CT and T1-weighted MR images. These findings resemble those of other myxoid tumors, such as myxoid malignant fibrous histiocytoma, myxoid liposarcoma, myxoid schwannoma, or neurofibroma.

MR imaging and CT, including dynamic study, showed the angiomatous and myxomatous natures of this tumor well. Aggressive angiomyxoma should be included in the differential diagnosis of myxoid masses involving the ischiorectal and perirectal spaces, especially in young women.

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Rupture of the Right Atrium–Superior Vena Cava Junction from Blunt Thoracic Trauma: Helical CT Diagnosis

A 56-year-old man was admitted to the emergency department after a high-speed motor vehicle crash during which he had been wearing his safety waist and shoulder belt. The patient had cranial trauma with initial loss of consciousness, facial trauma with many wounds, anterior thoracic trauma, abdominal contusion, and a deep injury to the right knee. The patient was fully conscious with a Glasgow Coma Scale score of 15 when he was referred to the emergency department. However, hemodynamic instability was noted and was controlled by volemic expansion.

Thoracic helical CT with contrast enhancement showed a hemomediastinum, a hemopericardium, and extravasation of contrast medium from the right atrium–superior vena cava junction (Fig. 5). Also noted were right lung lower lobe contusion and pleural effusion. Abdominal CT showed hepatic contusion without hemoperitoneum.



Fig. 5.—Chest CT scan shows leak of contrast medium close to superior vena cava (*arrow*), hemomediastinum, and pleural effusion.

Thoracotomy confirmed the vascular tear, which was subsequently repaired. The patient's postoperative course was uneventful, and he was discharged 3 weeks after surgery.

Blunt traumatic ruptures of the superior vena cava and the right heart are rare and are not usually diagnosed preoperatively. The literature is scarce on such a traumatic condition because most of these patients die before being admitted to the hospital. Three case reports have been described [1, 2]; two of the patients had presurgical explorations (CT in one case, cross-sectional echocardiography in another). None of these patients had angiography and two survived after surgery.

The pathogenic mechanism combines deceleration cardiac injuries associated with abdominal compression that leads to a huge venous pressure wave into the right heart and the superior vena cava. Therapeutic management of these injuries consists of intensive care before surgical repair by simple suture.

Several studies [3–5] have compared conventional CT sensitivity with angiography, and in all instances, angiography remained the gold standard. However, a recent study [6] that compared helical CT and angiography in the evaluation of thoracic aortic rupture showed that high-quality helical CT contributed equally as well as aortography except for the detection of small tears.

Our belief is that helical CT may be the immediate imaging procedure to evaluate mediastinal vascular ruptures. Large series of mediastinal vascular ruptures are needed to appreciate the high sensitivity of helical CT, but we believe that indications for angiography would decrease as in our case, in which angiography was not performed because of obvious lesions on helical CT images. Optimal polytrauma management needs close cooperation between the radiology team, the intensive care unit medical team, and the surgical team.

In summary, mediastinal vascular injuries are rare but severe and need quick management. Speed and high-image quality make helical CT the optimal exploration tool for diagnosis and therapeutic decisions in patients with polytrauma, especially when mediastinal injury is suspected.

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Migration of Translumbar Inferior Vena Cava Catheter into the Right Ureter

A 53-year-old woman with a long-standing history of Crohn's disease and short bowel syndrome requiring total parenteral nutrition presented to her primary care physician with a complaint of white-colored urine. Past medical history included extensive thrombosis of the patient's upper extremity, neck, and iliofemoral veins attributed to extended use of multiple previous central lines. For this reason, a translumbar inferior vena cava catheter had been placed approximately 1 month ear-

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lier for long-term administration of triphosphopyridine nucleotide.

Two days before presentation, the patient experienced an abrupt onset of polyuria of white-colored urine, which coincided temporally with the infusion of triphosphopyridine nucleotide. The patient was evaluated by her primary care physician. Laboratory examination showed mild metabolic acidosis with electrolyte abnormalities of hyponatremia and hypokalemia. A urine sample that had been collected by the patient was white in color and viscous. The patient was referred to our department for a contrast study of the translumbar catheter to determine its position. A scout film of the abdomen showed the translumbar catheter with the tip located at the level of the L3 vertebral body. The catheter tip was perpendicular to the expected course of the inferior vena cava. No apparent cephalad course of the catheter was evident within the inferior vena cava. The catheter was injected with approximately 15 ml of iodinated contrast material, and immediate opacification of the right ureter and the entire right renal collecting system was seen (Fig. 6A). The tip of the catheter was located in the midportion of the right ureter. No evidence of opacification of the inferior vena cava or other vascular structure and of extravasation of contrast material into the soft tissues around the ureter was seen. Focal medial deviation of the ureter at the point of catheter entry and mild luminal irregularity in this region was revealed. Helical CT of the abdomen was performed to further evaluate the position of the catheter relative to retroperitoneal structures and to exclude a periureteral fluid collection. The tip of the catheter was revealed just lateral to the infrarenal inferior vena cava (Fig. 6B). Opacification of the right renal collecting system was again seen. No evidence of extravasation of contrast material or periureteral fluid collection was found.

The catheter was removed, and MR venography was performed to evaluate the central venous system to plan for future venous access options. This procedure revealed complete thrombosis of the inferior vena cava below the level of the renal veins. A large collateral vessel was noted in the retroperitoneum adjacent to the right psoas muscle (Fig. 6C). In retrospect, this enlarged collateral was identified on the unenhanced CT scan as a large, unopacified tubular structure coursing through the right hemipelvis (Fig. 6D). Under sonographic guidance, this vessel was localized and punctured using an 18-gauge needle. A 7.0-French silastic catheter (Cook, Bloomington, IN) was advanced over a wire, and the line was tunneled to the anterior chest wall.

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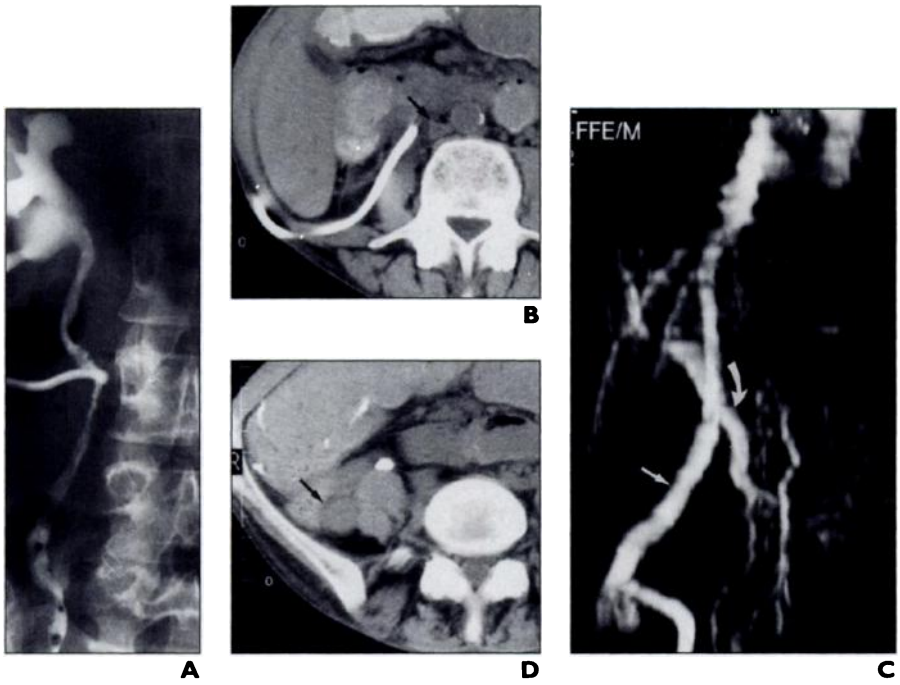


Fig. 6.—53-year-old woman 1 month after placement of translumbar inferior vena cava catheter for long-term administration of triphosphopyridine nucleotide.
A, Spot film of translumbar catheter (after injection of contrast material into catheter) shows opacification of right ureter and collecting system. Note medial deviation at point of catheter entry.
B, Unenhanced helical CT scan confirms position of translumbar catheter. Inferior vena cava is seen medial to catheter tip (*arrow*).
C, Maximum-intensity-projection image from MR venography shows occlusion of infrarenal inferior vena cava caudal to entry of large-caliber venous collateral (*straight arrow*) originating in right hemipelvis. Note superimposition of portal confluence over patent suprarenal inferior vena cava (*curved arrow*).
D, Unenhanced helical CT scan at level of upper pelvis reveals large venous collateral (*arrow*) adjacent to right psoas muscle. Contrast material is seen in right ureter.

CT and MR Imaging of Nasoethmoid Schwannoma with Intracranial Extension

Nerve sheath tumors occur in the head and neck region more frequently than in any other part of the body, affecting mainly the eighth cranial nerve [1]. However, nasoethmoid schwannoma is a rare tumor. In 1993, Higo et al. [2] reviewed the medical literature and found 160 cases of schwannoma of the nasal cavity and the paranasal sinuses with only a few cases with intracranial extension.

A 38-year-old man presented with a 1-year history of right-sided nasal stuffiness and nosebleeds, which were gradually increasing in frequency. A CT scan revealed a homogeneous soft-tissue density mass located in the right upper nasal cavity, ethmoid sinus, frontal sinus, and anterior cranial fossa. A coronal CT scan with bone window setting (Fig. 7A) revealed that the roof of the ethmoid sinus was remodeled and the cribriform plate was interrupted.

On February 29, 1996, the patient was admitted to our hospital. MR imaging showed that the mass was located in the right nasal cavity, ethmoid sinus, and frontal sinus and extended into the anterior cranial fossa (Figs. 7B–7D). The border of the mass was well defined and compressed the frontal lobe. The tumor was differentiated from the retained secretions in the frontal sinus and showed different signal intensity on all pulse sequences before and after IV administration of contrast material.

A biopsy of the tumor was performed, and pathologic examination revealed schwannoma. The patient subsequently underwent surgery. No dural defects were found, and the

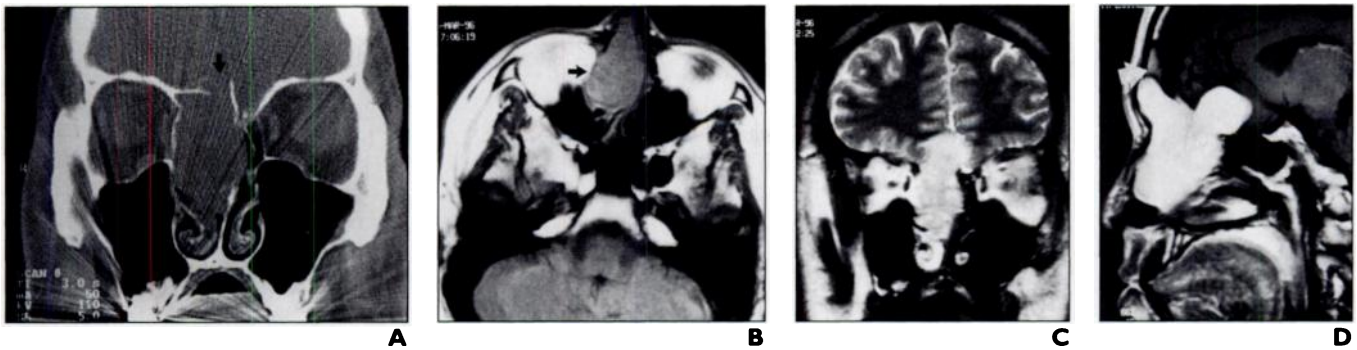


Fig. 7.—28-year-old man with nasoethmoid schwannoma with intracranial extension.
A, Coronal CT scan with bone window setting shows that roof of ethmoid sinus is remodeled and cribriform plate is interrupted (*arrow*). Nasal septum and lamina perpendicularis are deviated to left.
B, Tumor (*arrow*) is of intermediate signal intensity on unenhanced T1-weighted image (TR/TE, 660/15).
C, On T2-weighted coronal image (4000/90), tumor is of inhomogeneous high signal intensity. Note that no signal intensity changes are evident in frontal lobe compressed by tumor.
D, Sagittal T1-weighted image (661/15) after IV administration of gadopentetate dimeglumine reveals intracranial extension of tumor into anterior cranial fossa. In frontal sinus, tumor is differentiated from retained secretions without enhancement (*arrow*).

tumor was separated easily from the dura and excised successfully.

The diagnosis of schwannoma was confirmed by histopathologic examination of the specimen, which revealed compactly arranged spindle cells oriented parallel to one another that formed interlacing bundles and nuclear palisading of Antoni A type with some myxoid areas of Antoni B type. The tumor cells were positive for immunohistologic staining of S100 protein.

Apart from the olfactory nerve, any of the nerves supplying the paranasal sinuses may give rise to the tumor [1]. In our case, the origin of the tumor is obscure and the tumor presumably arose from the ethmoid sinus and grew into the anterior cranial fossa. However, the nerve from which the tumor arose is uncertain.

Nasal or paranasal schwannomas with intracranial extension are very rare. To our knowledge, only two cases have been reported. Zovickian et al. [3] reported a case of intranasal schwannoma with extension into the intracranial compartment, and Enion et al. [4] reported a case of nasoethmoid schwannoma.

In our case, determination that the mass was a schwannoma was difficult by only CT and MR imaging. Whereas MR imaging could clearly delineate the anatomic extent of the tumor, CT offered better resolution than MR imaging of a remodeled ethmoid sinus or interruption of the cribriform plate. CT and MR imaging were useful for determining the location and extension of the tumor.

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Author's Correction

Errors in "Case 4: malrotation of the small bowel" [1] were brought to my attention recently. In Figure 4A, the legend should read "CT scan through mid abdomen with IV administration of contrast agent reveals superior mesenteric artery (*arrowhead*) lying to patient's right of superior mesenteric vein (*arrow*)." In addition, on page 256 in the reference to Weingberger et al. [2], the text should read that five patients had the superior mesenteric vein to the left of the superior mesenteric artery (rather than "right" as printed) and hence had a reversed relationship of the superior mesenteric artery and superior mesenteric vein from what is normal.

I apologize for any confusion to the readers.

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Author's Correction

In the recently published article "Routine addition of an automated biopsy device to fine-needle aspiration of the lungs: a prospective assessment" (*AJR* **1997**;169:661–666), Dr. Stephen W. Miller was inadvertently excluded as an author of the study. Dr. Stephen W. Miller, who is affiliated with the Department of Radiology at Massachusetts General Hospital, Boston, MA, should have been listed as the ninth author, between Elkan F. Halpern and the senior author, Theresa C. McLoud

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