showed that the testis and tunica albuginea were not involved. The histopathologic diagnosis was embryonal cell rhabdomyosarcoma arising from the tail of the epididymis.

The appearance of paratesticular rhabdomyosarcoma on sonography and MR imaging is nonspecific. Care must be taken because similar findings may be seen in other more common paratesticular tumors such as fibromas, leiomyomas, or adenomatoid tumors. Previous studies on intrascrotal extratesticular masses have suggested that a homogeneously echogenic appearance on sonography indicates a benign process [4, 5]. However, the uniformly echogenic appearance of paratesticular rhabdomyosarcoma suggests a more cautious diagnostic approach. Because paratesticular rhabdomyosarcoma is highly aggressive and can metastasize, early recognition is important. The tumor should be included in the differential diagnosis of intrascrotal extratesticular masses.

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MR Findings of Tracheal Involvement in Wegener's Granulomatosis

A 16-year-old girl was referred for symptoms of chronic sinusitis and treatment of severe tracheal narrowing that had caused increasing dyspnea for 16 months. Biopsies from her larynx and nose were nondiagnostic but showed chronic inflammation, raising the possibility of Wegener's granulomatosis. Serum was negative for antineutrophil cytoplasmic antibodies (ANCA). The chest radiograph showed no pulmonary abnormality. Rigid and flexible bronchoscopy revealed diffuse inflammation of the subglottic trachea. T1-weighted MR images of the neck and upper thorax depicted diffuse thickening of the tracheal submucosal tissues and narrowing of the lumen. The tracheal wall increased in signal intensity on T2-weighted images and enhanced strongly after administration of gadopentetate dimeglumine (Figs. 2A–2C). With a presumptive diagnosis of Wegener's granulomatosis, the patient was treated with prednisolone and methotrexate. Tracheal narrowing was treated by a series of bronchoscopic dilatations and laser therapy. Her symptoms of dyspnea progressed, and a chest radiograph obtained 6 months later showed bilateral nodules for the first time (Fig. 2D). Lung tissue obtained at thoracotomy showed extensive necrotizing granulomatous inflammation with giant cell reaction centered around blood vessels, consistent with Wegener's granulomatosis. Her chemotherapy was changed to cyclophosphamide (Cytoxan; Bristol-Myers Squibb, Princeton, NJ). The lung nodules subsequently resolved; however, tracheal stenosis continues to be problematic and is alleviated by intermittent manual dilatation.

Increased awareness of tracheobronchial Wegener's granulomatosis is due in part to development of a highly specific test for Wegener's granulomatosis in the form of antineutrophil cytoplasmic antibodies with diffuse granular cytoplasmic staining (c-ANCA), widespread use of the fiberoptic bronchoscope, and greater success in therapy that has extended patient survival in recent years. Daum et al. [1] reported tracheobronchial abnormalities in 30 (59%) of 51 patients with Wegener's granulomatosis in whom bronchoscopy was performed. Airway involvement in patients with Wegener's granulomatosis may be asymptomatic, but clinical presentation can range from subtle hoarseness to stridor and life-threatening upper airway obstruction. Only 20% of lesions diminish with immunosuppressive therapy alone; 80% are fixed or irreversible because of chronic inflammation [2]. Bronchoscopy is limited in assessment of the submucosal and extrinsic extent of disease. Cross-sectional imaging studies, especially CT and MR imaging, noninvasively help to determine the degree of tracheal narrowing, the length of obstruction, extratracheal involvement, and the presence of enlarged lymph nodes. Maskell et al. [3] analyzed chest CT findings of 30 patients with Wegener's granulomatosis and reported bronchial abnormalities in 12 (40%), eight of whom had bronchial wall thickening and bronchiectasis remote from other pulmonary involvement.

MR imaging is ideal for evaluating tracheal disease because of its multiplanar capability, the superior contrast resolution in depicting inflammatory tissue compared with that seen on CT scans, and the ability to depict surrounding vessels without the need for intravascular contrast material. Although the MR findings are characteristic of tracheal inflammation, they are not specific for Wegener's granulomatosis. Other causes of tracheal wall thickening and luminal narrowing include strictures resulting from intubation, amyloidosis, sarcoidosis,

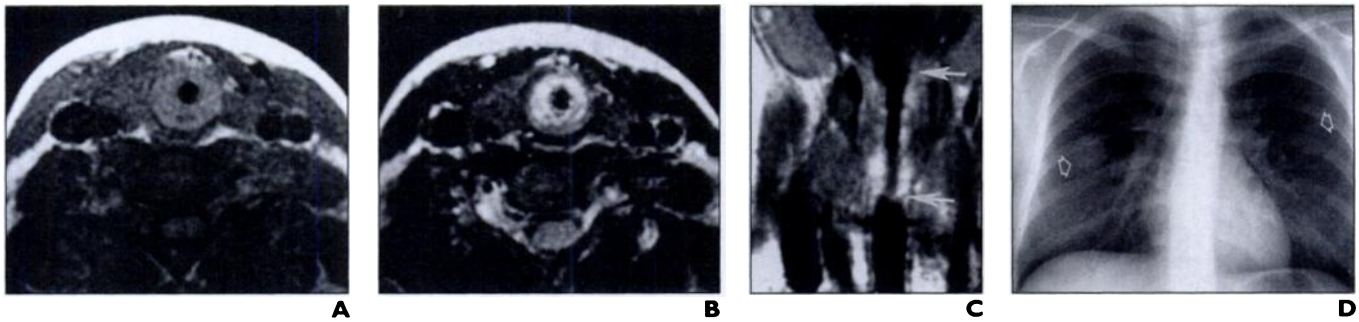

Fig. 2.—16-year-old girl with Wegener's granulomatosis and tracheal stenosis.
A, T1-weighted axial MR image (TR/TE [800/25], two excitations) shows circumferential narrowing of trachea.
B, T2-weighted axial MR image (2200/90, two excitations) shows increase in signal intensity in submucosal tracheal soft tissues.
C, T1-weighted coronal MR image after administration of gadopentetate dimeglumine shows subglottic tracheal narrowing that extends along 3 cm of tracheal wall, with enhancement of submucosal tissue (between *arrows*).
D, Chest radiograph obtained because of increasing dyspnea shows bilateral nodules (*arrows*) proven to represent pulmonary Wegener's granulomatosis.

relapsing polychondritis, tracheobroncheopathia osteochondroplastica, and infectious tracheitis. The decision to treat tracheal Wegener's granulomatosis by chemotherapy or by mechanical dilatation is based in part on evidence of active inflammation [4]. Although c-ANCA titer changes are known to correlate with changes in overall disease activity, the presence or absence of c-ANCA is less accurate in predicting the presence of tracheobronchial disease [1]. Our patient did not test positive for c-ANCA until 12 months after her initial presentation with tracheal stenosis and at least 18 months after her initial symptoms. MR imaging provides a noninvasive method for identifying tracheal inflammation that may help the clinician to decide the most appropriate type of therapy for tracheal narrowing due to Wegener's granulomatosis and provide a noninvasive method for following up patient response.

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Portal and Mesenteric Vein Gas in Diverticulitis: CT Findings

Hepatic portal vein gas is most often associated with extensive bowel necrosis due to mesenteric infarction, with mortality exceeding 75% in such circumstances [1]. We present a case of hepatic portal and mesenteric venous gas associated with colonic diverticulitis. This cause is rare, and to our knowledge, ours is the first such case diagnosed with CT to be reported.

A 41-year-old man presented to the emergency room with a 4-day history of low-grade fever, chills, nausea, vomiting, and diarrhea. The patient's initial temperature was 105.3°F (40.7°C). His abdomen was mildly tender but had no localizing peritoneal signs. Rectal examination showed scant hemoccult positive stool and no palpable masses.

The blood count revealed a WBC of 5000/μl (5×10^9/l) with a differential of 72 segmented polymorphonuclear leukocytes and 21 bands. Serum electrolytes were unremarkable. Acute enteritis was diagnosed. The patient was given IV fluids and cefoxitin and was admitted to the hospital.

An enhanced CT scan of the abdomen and pelvis showed multiple linear foci of gas in the left portal vein branches, main portal vein, and inferior mesenteric vein (Figs. 3A–3C). Diverticulosis involved the mid and distal portions of the sigmoid colon, and mild pericolonic inflammatory changes were seen (Fig. 3D). A small air collection adjacent to the sigmoid colon suggested a localized diverticular perforation.

Exploratory laparotomy revealed minimal findings of diverticulitis at the rectosigmoid junction. A 3.0- to 4.0-cm area of induration without any obvious pus could be identified in the sigmoid mesocolon. A Hartmann's procedure and an end colostomy were performed. *Bacteroides fragilis* grew from one blood culture. The patient was discharged on the seventh postoperative day without further complications.

Pathologic evaluation revealed acute suppurative diverticulitis with pericolonic microabscess and phlegmon. Although a direct colovenous fistula could not be identified, such a fistula was believed to represent the most probable cause for the extensive mesenteric and portal vein gas.

A review of the literature revealed only seven patients with documented hepatic portal vein gas due to colonic diverticulitis, all involving the sigmoid colon [2, 3]. Five of the patients, including ours, survived. In the others, portal vein or mesenteric vein gas was discovered with radiography, barium or meglumine diatrizoate (Gastrografin; Mallinckrodt Medical, St. Louis, MO) enemas, or, in one patient [2], sonography. An eighth reported case was complicated by a large pelvic abscess [4]. To our knowledge, this case was the first to be documented showing the CT findings of portal and mesenteric vein gas due to diverticulitis.

Ischemic bowel necrosis has been documented in about 72% of cases of portal vein gas [1]. However, portal vein gas has also been associated with such entities as bowel distention, perforated ulcer, acute hemorrhagic pancreatitis, corrosive ingestion, and inflammatory bowel disease after barium enema. In these clinical circumstances, mucosal damage without necrosis is the usual pathologic cause. Improved survival has been reported in this subset of patients. Although portal vein gas can be seen on plain ra-

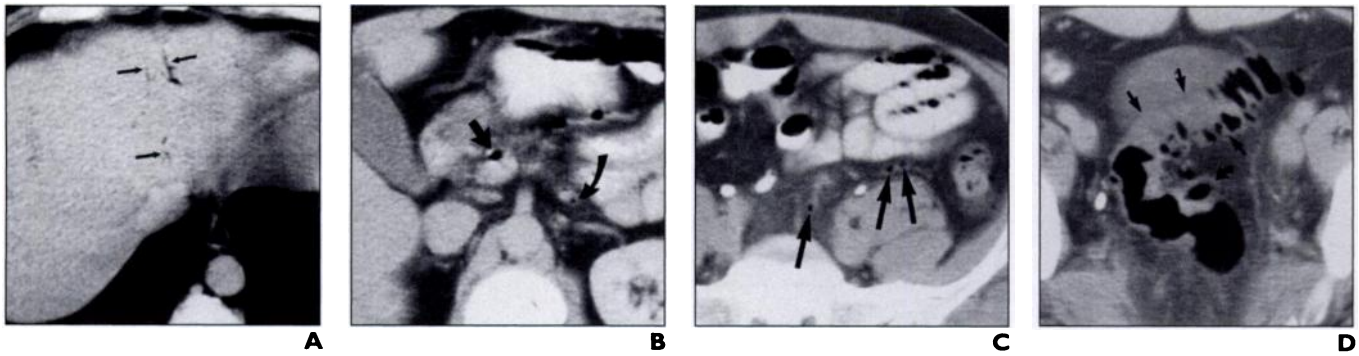

Fig. 3.—Enhanced CT scans of abdomen and pelvis in 41-year-old man with hepatic portal and mesenteric venous gas.
A, Linear streaks of gas in left portal vein branches (*arrows*).
B, Gas in inferior mesenteric vein (*curved arrow*) and superior mesenteric vein (*straight arrow*) near splenoportal confluence.
C, Gas in inferior mesenteric vein branches draining sigmoid colon (*arrows*).
D, Mural thickening and diverticulosis (*straight arrows*). Mild inflammatory changes are present in sigmoid mesocolon. Small eccentric air collection is compatible with localized diverticular perforation (*curved arrow*).