Imaging Manifestations of Kaposi Sarcoma

Carlos S. Restrepo, MD • Santiago Martinez, MD• Julio A. Lemos, MD• Jorge A. Carrillo, MD • Diego F. Lemos, MD • Paulina Ojeda, MD Prakash Koshy, MD

Kaposi sarcoma (KS) is a low-grade vascular tumor that typically manifests as one of four variants: classic KS, endemic (African) KS, iatrogenic (organ transplant–related) KS, or acquired immunodeficiency syndrome (AIDS)–related KS. Several clinical and epidemiologic differences have been noted among these variants. Classic KS and endemic KS rarely require radiologic evaluation due to their usually chronic course and stability of skin compromise. However, iatrogenic KS and AIDS-related KS, the most common forms of the disease, are frequently disseminated or symptomatic and may thus require imaging studies for both diagnosis and staging. KS is the most common tumor among AIDS patients, affecting a high percentage of these individuals, and is considered to be an AIDS-defining illness. Multiple organs can be involved by AIDS-related KS. KS has been linked with human herpes virus type 8 infection and other cofactors. Although pulmonary, gastrointestinal, and skin involvement by KS has previously been described, this tumor can affect multiple organs, generating a wide spectrum of imaging findings and pathologic correlates. It is important for the radiologist to be familiar with this spectrum of imaging manifestations and corresponding pathologic findings.

Abbreviations: AIDS = acquired immunodeficiency syndrome, HHV8 = human herpes virus type 8, HIV = human immunodeficiency virus, KS = Kaposi sarcoma

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1From the Department of Radiology, Louisiana State University Health Sciences Center, New Orleans, La (C.S.R., S.M., J.A.L., D.F.L., P.K.); and the Department of Radiology, Universidad Nacional de Colombia, Hospital Santa Clara, Bogotá, Colombia (J.A.C., P.O.). Presented as an education exhibit at the 2004 RSNA Annual Meeting. Received June 8, 2005; revision requested July 15 and received October 3; accepted October 5. All authors have no financial relationships to disclose. Address correspondence to C.S.R., Department of Radiology, University of Texas Health Science Center at San Antonio, Mail Code 7800, 7703 Floyd Curl Dr, San Antonio, TX 78229 (e-mail: restrepo@uthscsa.edu).

2Current address: Department of Radiology, Duke University Medical Center, Durham, NC.

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Introduction
Kaposi sarcoma (KS), first described by Moritz Kaposi in 1872, is a low-grade mesenchymal tumor that involves the blood and lymphatic vessels, affecting primarily the skin and causing disseminated disease in a variety of organs (Fig 1) (1). Currently, four variants of the disease with different clinical manifestations are recognized: classic (sporadic or Mediterranean) KS, endemic (African) KS, iatrogenic (organ transplant–related) KS, and acquired immunodeficiency syndrome (AIDS)–related (epidemic) KS (2). KS was viewed as an uncommon tumor prior to the 1980s but came to be considered an AIDS-defining illness soon after the onset of the AIDS epidemic.

In this article, we review the causes and mode of transmission of KS. In addition, we discuss and illustrate the clinical, epidemiologic, radiologic, and pathologic features of KS with respect to the variant (with special emphasis on AIDS-related KS) and the area of involvement (head and neck, thorax, liver and spleen, gastrointestinal tract, genitourinary tract, musculoskeletal system).

Etiology and Mode of Transmission
Human herpes virus type 8 (HHV8 or KS-associated herpesvirus) (3) and other cofactors (eg, cytokine-induced growth) have been linked to the development of KS and other diseases (eg, primary effusion lymphoma, multicentric Castleman disease) (4,5). Whereas AIDS-related KS and iatrogenic KS are clearly associated with immunosuppressive states, immunostimulation or immune disregulation may be involved in the pathogenesis of classic KS. The etiologic trigger has not been clearly identified, although HHV8 infection can potentially initiate the processes (6). No definitive evidence of underlying immunodeficiency has been found in endemic KS (7).

The mode of transmission of HHV8 is not completely understood. Adult homosexual contact seems likely to be an important route of transmission in North America, whereas mother-to-child and child-to-child routes may be responsible for certain pediatric cases in Africa and southern Europe (4,5). Although HHV8 infection may occur from the organ transplant donor to the recipient, it has been suggested that reactivation is the primary mechanism responsible for iatrogenic KS (8).

Variants of KS
Classic (Sporadic) KS
Classic (sporadic) KS mostly affects individuals of eastern European or Mediterranean origin and Ashkenazi Jews between 50 and 80 years old, with a male-female ratio of 10–15:1. A lower risk noted in second-generation American Jews raises the question of whether factors other than HHV8 infection are involved (6). Two clinical forms of the disease are recognized. A common and asymptomatic cutaneous type manifests clinically as red, purple, or brown patches, plaques, or nodular skin lesions that develop over 10–15 years; has a prolonged survival rate; and compromises the skin of the lower limbs (ankles and soles). If left untreated, the tumor can become ulcerated, and venous stasis with lymphedema may develop. A second form of the disease follows a fulminant course, with rapid development of disseminated mucocutaneous and visceral lesions (9). In one retrospective review, biopsy-proved extracutaneous involvement was found in up to 18.4% of cases (10). Involved anatomic structures include the gastrointestinal tract, lymph nodes, liver, lungs, kidneys, and spleen (9). Higher incidences of lymphoproliferative disorders (ie, lymphoma, chronic lymphocytic leukemia, and so on) and of other primary malignancies (ie, bladder and colon carcinoma) have also been reported (6,10).

Endemic (African) KS
Endemic KS is a relatively common neoplasm that primarily affects men in East and Central Africa in the 4th decade of life (male-female ratio, 13–17:1). Endemic KS accounts for 9% of all cancers among men in Uganda (2,5). Four clinically distinct types of endemic KS have been recognized: benign nodular, aggressive localized, disseminated florid, and pediatric lymphadeno-
pathic. The clinical setting is similar to that of classic KS in most instances (ie, localized disease in >50% of cases). Aggressive cases are likely to penetrate the skin and affect the bone. In children of sub-Saharan Africa (eg, Zambia, Uganda), an aggressive lymphadenopathic form manifests with both a lower male-female ratio (3:1) and a lower prevalence, characterized by a more disseminated pattern, with lymphadenopathies and rapid fatal outcome occurring within 3 years in most patients (9). As in classic KS, associated lymphoproliferative disorders have also been described (11).

Iatrogenic (Organ Transplant-related) KS

The development of neoplasms is a well-known complication of solid organ transplantation that is related to chronic drug-induced immunosuppression (2,5). Frequently reported tumors include skin cancer (eg, squamous and basal cell carcinomas), iatrogenic KS, lymphoma (especially non-Hodgkin lymphoma), and visceral carcinomas (12). Diverse incidences of iatrogenic KS have been reported. In a study of 8724 de novo malignancies in recipients of organ allografts, iatrogenic KS had a prevalence of 5.7%, with a mean development time of 21 months (13). Approximately 60% of patients developed nonvisceral KS confined to the skin, conjunctiva, or oropharyngeal mucosa, whereas 40% had visceral disease (ie, involvement of the gastrointestinal tract, lungs, lymph nodes, and so on) (13).

Radiographic findings have been reported in the lungs and in diverse allograft organs. In a case of iatrogenic KS affecting the transplanted kidney, ureter, and urinary bladder, contrast material-enhanced computed tomography (CT) showed heterogeneous hypoechoic masses within the urinary bladder and distal ureter; associated hydroureterosis, which exhibited nodular enhancement; and additional masses in the renal pelvis of the allograft (14). In another case, kidney transplant–related iatrogenic KS manifested at CT as an infiltrative lesion involving the allograft sinus and hilum, with mild hydroureterosis and effacement of the fat surrounding the iliac vessels (15). In a case of pulmonary iatrogenic KS in a renal allograft patient, chest radiography showed bilateral opacities with right hilar adenopathy. Further characterization with CT revealed bilateral ill-defined nodules, alveolar opacities, bilateral pleural effusions, and right hilar adenopathy. The differential diagnosis includes lymphoma, Pneumocystis jiroveci pneumonia, and fungal or mycobacterial infections (16). Multiple hypoattenuating areas are seen at CT in both the liver and the spleen in patients with liver allograft iatrogenic KS and splenic involvement (17).

Epidemic (AIDS-related) KS

By 1989, 15% of all reported AIDS patients in the United States had AIDS-related KS (18). The lifetime prevalence may be as high as 50% among homosexual male AIDS patients (19). Less frequently, AIDS-related KS has also been reported among intravenous drug users, hemophiliac patients, and women (18,20–23). The higher prevalence of AIDS-related KS among homosexual men has received considerable attention, and several hypotheses have been offered to explain this phenomenon. The high prevalence of HHV8 infection among homosexual men correlates with the higher number of partners in this population. Furthermore, HHV8 infection is an independent risk factor for AIDS-related KS secondary to sexual transmission of the virus, probably by means of receptive anal intercourse. Studies evaluating numerous other cofactors such as inhaled nitrites, coinfection (intestinal parasites, hepatitis B, cytomegalovirus), and fecal-oral contact showed no consistent association of these cofactors with the development of AIDS-related KS (5). Recently, the prevalence of AIDS-related KS has declined, presumably due to widespread use of highly active antiretroviral therapy (24–26). The overall risk of developing AIDS-related KS is 20,000 times greater in patients with AIDS than in the general population and 300 times greater than in other immunosuppressed patients (18). AIDS-related KS usually develops in the setting of a low CD4 lymphocyte count (<150–200 cells/mm3) (27,28). Prognosis is better in white homosexual men and, interestingly, worse among black female intravenous drug users (29). Factors that are predictive of a shorter survival time include prior or coexistent opportunistic disease, presence of systemic symptoms (eg, unexplained fever lasting more than 2 weeks, unexplained weight loss of 10% or more, unexplained diarrhea and night sweats), and a low absolute CD4 lymphocyte count (<100–300 cells/mm3) (30,31).

In an autopsy series by Niedt and Schinella (32), the most common sites for visceral involvement by AIDS-related KS were the lymph nodes (72% of cases), lung (51%), gastrointestinal tract (48%), liver (34%), and spleen (27%). An autopsy series by Lemlich et al (33) showed that, at the time of death, only 25% of patients had evidence of cutaneous disease alone and up to 29% had visceral compromise without skin lesions. Concomitant infections are the cause of death in nearly 80% of cases. These infections include cytomegalovirus, candidiasis, Mycobacterium avium
intracellular infection, P jiroveci pneumonia, bacterial pneumonia, and herpes simplex viral infection (33).

**Areas of Involvement**

**Head and Neck**
Before the emergence of AIDS, head and neck KS was an uncommon neoplasm (34). Soon after the onset of the AIDS epidemic, however, head and neck AIDS-related KS became one of the most common manifestations of the disease (35–38). Cutaneous involvement (ie, involvement of the face, scalp, and neck skin) is present in 66% of cases; mucosal forms (ie, intraoral, laryngeopharyngeal, and, less frequently, nasal) are seen in 56%; and enlarged lymph nodes are present in up to 13%. Rarely, other sites (eg, conjunctiva, lacrimal gland, parotid gland, masseter muscle, tonsil, and so on) may be affected (38–41). AIDS-related KS is usually asymptomatic; however, symptoms

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**Figure 2.** Disseminated AIDS-related KS in a 39-year-old man with neck and head compromise. (a) Head CT scan shows left frontal soft-tissue thickening (arrow), a finding that is consistent with cutaneous KS. (b) CT scan obtained inferior to a shows an ill-defined heterogeneous mass at the nasopharynx (white arrows). Note the areas of enhancement within the lesion (black arrows), a characteristic feature of KS. (c) CT scan of the neck exhibits enlarged hyperattenuating lymph nodes (arrows) with diffuse thickening and increased attenuation of the subcutaneous soft tissues, findings that are consistent with swelling due to lymphatic obstruction. Biopsy of the nasopharyngeal mass was performed. (d) Photomicrograph (immunohistochemical staining) shows diffuse positivity for vimentin. The sample was also positive for CD31.
may appear when mucosal lesions become ulcerated or produce local mass effect (37,38).

Pharyngeal AIDS-related KS may manifest radiographically as diverse nodular lesions, usually without ulceration. CT is helpful in assessing the involvement of deep tissue planes and the extent of nodal disease. Findings include nodular or polypoid intraluminal protrusions, distortion of the valleculae and pyriform sinuses, infiltration of deep-tissue planes, and adenopathies (Fig 2) (42).

At CT and magnetic resonance (MR) imaging, AIDS-related KS is characterized by relatively strong tumoral enhancement after contrast material administration (Fig 3), a finding that may suggest the diagnosis in the appropriate clinical setting (ie, typical skin lesions), even though this finding is considered nonspecific (43).

**Thorax**

Thoracic disease is found in about 45% of patients with cutaneous AIDS-related KS (44,45). In another series of patients with bronchopulmonary AIDS-related KS, 15.5% did not exhibit mucocutaneous involvement (46). Manifestations include parenchymal (Fig 4), tracheal (Figs 5, 6), lymphatic, pleural (Fig 7), and chest wall abnormalities. Common clinical symptoms include chronic cough, dyspnea, fever, and hemoptysis (47). The typical purplish endoscopic appearance

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**Figure 3.** Disseminated AIDS-related KS in a 36-year-old man. (a) Photograph shows nodular violaceous involvement of the tongue base and soft and hard palates. (b) CT scan shows nodularity of the right lingual tonsil and vallecula, partial obliteration of the right pyriform sinus (black arrows), a pedunculated mass arising in the right lateral aspect of the epiglottis (white arrow), and enhancing lymph nodes (*). (c) Contrast-enhanced fat-saturated T1-weighted MR image shows the intensely enhanced mass (arrows).

**Figure 4.** Pulmonary KS. Photograph shows pulmonary KS. Arrows indicate violaceous patchy areas in the lungs that correspond to tumor.
of tracheobronchial KS is helpful in developing the differential diagnosis. About 20% of deaths are related to complications of the disease itself (upper airway obstruction, hemorrhage, or parenchymal destruction), but the majority of deaths are related to other factors (eg, concomitant infection) (48). A low CD4 lymphocyte count (<100 cells/mm³) generally accompanies thoracic involvement by AIDS-related KS (30,46,49).

Cardiac compromise may be either primary or secondary. Although autopsy series have shown AIDS-related KS to be present in up 28% of the AIDS population, compromise is rarely extensive enough to cause dysfunction and clinical manifestations (50,51).

At radiography, AIDS–related KS may manifest as an isolated finding or in association with an opportunistic infection. In the first scenario, radiography may demonstrate middle to lower lung zone reticular opacities and parenchymal nodules with a bronchovascular distribution that may progress to consolidation, peribronchial cuffing, Kerley B lines, pleural collections, and hilar or mediastinal adenopathies (Figs 8–10) (46,49,52).
Figures 9, 10. (9) Thoracic AIDS-related KS in a 45-year-old man. (a) Chest radiograph demonstrates multiple bilateral 3–5-mm micronodules in a peribronchovascular distribution. (b) High-resolution lung CT scan shows innumerable bilateral, poorly defined peribronchovascular micronodules, some of which exhibit coalescence. (c) CT scan (soft-tissue windowing) depicts enlarged lymph nodes in the axillae and mediastinum (thin arrows). Note also the bilateral pleural fluid collections as well as some nodularity (thick arrows). Skin compromise is also identified in the left hemithorax (arrowhead). Histopathologic findings were consistent with KS. (10) Disseminated AIDS-related KS in a 36-year-old man with thoracic involvement. (a) Chest radiograph shows ill-defined nodular confluent opacities in the left upper lobe. (b) Chest CT scan demonstrates multiple nodules around the bronchus for the apicoposterior segment of the left upper lobe (black arrow). Other small nodules are also identified in the posterior segment of the right upper lobe (white arrows). (c) CT scan (soft-tissue windowing) demonstrates enlarged enhancing lymph nodes (arrows) in the left hilum and occupying the azygosophageal recess. Bronchoalveolar lavage was negative. Results of transbronchial biopsy confirmed the diagnosis.
Normal chest radiographic findings may occasionally be present (44–47,49,52). On the other hand, when AIDS-related KS is associated with an opportunistic infection (eg, \textit{P} jiroveci infection), radiography demonstrates granular opacities with a more diffuse or apical distribution and, in some cases, cavitations (46).

CT is at least slightly better than radiography in identifying patients with thoracic disease and in developing the differential diagnosis for thoracic complications of AIDS (53,54). A characteristic CT finding in AIDS-related KS is the presence of bilateral and symmetric ill-defined nodules in a peribronchovascular distribution (flame-shaped lesions), usually exceeding 1 cm in diameter (55,56). Ground-glass opacities may be seen surrounding the nodules (“halo sign”) (57,58). Other common findings include peribronchovascular and interlobular septal thickening, fissural nodularity, mediastinal adenopathies (eg, axillary, mediastinal, and hilar) (Figs 8–10), and pleural abnormalities (eg, bilateral pleural collections and, rarely, pleural implants) (Fig 11) (45,46,58,59).

The presence of pleural collections has been associated with shortened survival times (30). Alveolar hemorrhage is frequently diagnosed during bronchoalveolar lavage in human immunodeficiency virus (HIV)—positive patients and is related to extravasation of erythrocytes into the alveolar spaces (Fig 12) (60). Other less common findings include asymmetrically distributed nodules and larger masses. Lytic lesions of the sternum and thoracic spine and soft-tissue masses or infiltration of the skin and subcutaneous fat are well-recognized abnormalities associated with thoracic wall KS, being seen in up to 53% of cases (59).
Other imaging modalities, such as scintigraphy with sequential thallium and gallium scanning, have also been used. Gallium uptake is usually negative in KS but positive in infection and lymphoma, whereas thallium uptake is positive in KS and lymphoma (61). This combined imaging is rarely necessary but may be helpful when coexisting opportunistic infection is suspected (62). No intrapulmonary accumulation of indium 111–labeled polyclonal human immunoglobulin is seen in patients with AIDS-related KS (63). Little information is available regarding the use of MR imaging in thoracic AIDS-related KS. Findings include hyperintense areas on T1-weighted images, markedly reduced signal intensity on T2-weighted images, and strong tumoral enhancement after the injection of gadopentetate dimeglumine (64).

The differential diagnosis includes lymphoma, bronchogenic carcinoma, infection, and bacillary angiomatosis (44,54,65–68). A diagnosis of infection (eg, mycobacterial or bacterial infection) is favored by the presence of a tree-in-bud pattern or nodules less than 1 cm in diameter with a centrilobular distribution (69). 
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P jiroveci pneumonia does not usually manifest with hilar adenopathies or pleural effusion but may exhibit cavitation (Fig 13) (70). Nodule size is not helpful in differentiating KS from lymphoma or bronchogenic carcinoma, and biopsy may be necessary (69). Bacillary angiomatosis may mimic AIDS-related KS, with violaceous plaques at bronchoscopy, solitary or multiple pulmonary nodules, and mediastinal lymphadenopathies that demonstrate intense enhancement at contrast-enhanced CT. Bacillary angiomatosis should be suspected in heterosexual patients with a diagnosis of KS (62,66).

Liver and Spleen

AIDS-related KS of the liver is the most common intrahepatic neoplasm in patients with AIDS and was found in 34% of cases in the autopsy series by Niedt and Schinella (32). Cutaneous compromise is present in most cases and is helpful in making the diagnosis when other processes such as metastatic disease, fungal microabscesses, or multiple hemangiomas are being considered. Nonspecific findings include hepatomegaly and elevated serum alkaline phosphatase levels. At gross examination, the liver appears with multiple purple-brown spongiform nodules in the periportal connective tissue. Splenic compromise is less common than hepatic compromise (71–74).

Ultrasonography (US) may show hepatomegaly with multifocal hyperechoic nodules (5–12 mm in diameter) adjacent to portal veins,
parenchymal bands, and heterogeneous parenchymal infiltration (Fig 14). Baseline CT findings include hypoattenuating nodules and irregular enlargement of the hilum and peripheral portal branches. After the injection of iodine-based contrast material, more hypoattenuating nodular lesions and periportal tissue can be visualized, most of which exhibit enhancement on delayed scans (4–7-minute delay). Nodular lesions may appear iso- or hyperattenuating on delayed images and may be indistinguishable from multiple hemangiomas (Fig 15) (71,75–77). Similar findings have been described in the spleen (Figs 16, 17) (74).

**Gastrointestinal Tract**

Gastrointestinal AIDS-related KS compromise is the most common visceral involvement in disseminated disease, being seen in up to 50% of patients. Concomitant cutaneous compromise is more common, but primary cases have also been reported (33). AIDS-related KS can affect any level of the gastrointestinal tract from the oropharynx to the rectum, including the gallbladder. The duodenum is the most frequently affected site. The red-purple endoscopic appearance of lesions is helpful in developing the differential diagnosis, which may include lymphoma, opportunistic infections, hematogenous metastasis, polyps, and Crohn disease. Endoscopic biopsy may be negative because of the submucosal location of the tumor. Early disease is frequently asympto-
matic, but complications (ie, bleeding, intestinal and biliary obstruction, intussusception, perforations, diarrhea, and protein-losing enteropathy) may occur as the lesions enlarge (78).

Double-contrast barium studies may not help detect early flat lesions. Larger lesions are detected as masses with or without central ulceration (“bull’s-eye” or “target” lesions) (75). Polypoid submucosal masses 0.5–3 cm in diameter and irregular fold thickening may be detected at

CT (Figs 18, 19). After the intravenous injection of contrast material, hyperattenuating lymphadenopathies of the porta hepatis or peripancreatic, retroperitoneal, mesenteric, inguinal, or pelvic hyperattenuating lymphadenopathies may be seen in approximately 80% of cases of disseminated KS (79). Bleeding may be a life-threatening complication, often requiring angiographic diagnosis and treatment (80).

**Figures 16, 17.** (16) Disseminated AIDS-related KS in a 41-year-old man with abdominal compromise. (a) Arterial phase abdominal CT scan shows an ill-defined, wedge-shaped hypoattenuating lesion (arrow) in the spleen. Note also the enhancing adenopathies in the minor curvature of the stomach. (b) On an equilibrium phase CT scan, the splenic lesion has become isodense, a pattern that is sometimes seen in hemangiomas. Biopsy showed KS. (17) Splenic KS in a 50-year-old HIV-positive man. Abdominal CT scan shows multiple subcentimeter hypoattenuating nodules in the spleen. Imaging-guided cytologic analysis revealed KS. (Courtesy of Diego Aguirre, MD, Department of Radiology, Fundación Santa Fe de Bogotá, Bogotá, Colombia.)

**Figure 18.** KS in a 44-year-old man with AIDS who presented with fever and diarrhea. Abdominal CT scan shows circumferential wall thickening of the cecum (arrows) that is not associated with enlarged lymph nodes or adjacent fat stranding. A flat lesion was visualized at colonoscopy. Subsequent biopsy helped confirm KS. (Courtesy of Diego Aguirre, MD, Department of Radiology, Fundación Santa Fe de Bogotá, Bogotá, Colombia.)

**Figure 19.** Disseminated AIDS-related KS in a 36-year-old man with rectal compromise. Abdominal CT scan shows a thickened, hypervascular rectal wall (arrows) with involvement of surrounding structures, including the prostate gland. The diagnosis was confirmed with endoscopic biopsy.
Genitourinary Tract
Genitourinary AIDS-related KS most commonly involves the skin of the penis (81). Complications such as meatal obstruction (82,83) and gangrene (84) rarely occur, and imaging studies are not usually needed. Although renal AIDS-related KS has been documented in autopsy series, it does not usually exhibit clinical or radiologic manifestations (77,78). Hydronephrosis and obstructive uropathy may result from enlargement of retroperitoneal lymph nodes (77). Involvement of other genitourinary sites (eg, adrenal glands, bladder, scrotum, seminal vesicles, testes) is exceedingly rare.

Musculoskeletal System
Musculoskeletal involvement is uncommon and occurs secondary to local extension from the skin, although primary cases have also been reported. Conventional radiography may show erosion to frank destruction of bone as well as periosteal reaction, which may be better characterized at CT (Fig 20). Soft-tissue masses have also been described (Fig 21). MR imaging depicts bone marrow abnormalities and soft-tissue masses (Figs 22–24) (85–88). Differentiation from bacillary...
Figure 22. Disseminated AIDS-related KS in a 35-year-old man with a history of the disease. (a) Abdominal CT scan demonstrates a round, hypoattenuating bone lesion (arrow) in the right aspect of the L1 vertebral body. (b–d) Sagittal MR images of the lumbar spine show the lesion to be isointense with a T1-weighted sequence (b), hyperintense with a T2-weighted sequence (c), and diffusely enhancing with a fat-saturated T1-weighted sequence performed after the intravenous injection of gadopentetate dimeglumine (d). In addition, similar lesions are seen in L2 and L5. Findings at bone biopsy were consistent with KS.

Figure 23. Disseminated AIDS-related KS in a 41-year-old man with chronic back pain. (a) Abdominal CT scan demonstrates an ill-defined, hypoattenuating osseous lesion (arrow) in the posterior aspect of the right iliac bone. The lesion was barely visible at T1-weighted MR imaging. (b) MR image obtained after the intravenous administration of gadopentetate dimeglumine shows areas of intense enhancement (arrows).
Figure 24. Disseminated AIDS-related KS in a 45-year-old man who presented with diffuse swelling of the left lower extremity. The patient had a chronic history of skin and thoracic KS with poor response to chemotherapy. (a, b) Anteroposterior (a) and lateral (b) radiographs show diffuse nodular thickening of soft tissues with multiple areas of osteopenia (arrows in a). (c) Bone scintigrams demonstrate irregular radiotracer uptake in the distal left foot. ANT = anteroposterior, LAT = lateral, PLA = posterolateral. (d–f) Sagittal MR images show the distal first metatarsal bone and the proximal and distal phalanges of the great toe. These structures are hypointense on the T1-weighted image (d), are hyperintense on the T2-weighted image (arrows in e), and demonstrate enhancement on the contrast-enhanced image (arrows in f). Abnormalities were also identified in multiple phalanges and metatarsal bones as well as in the cuboid bone. (g) MR image shows diffuse thickening and enhancement of the soft tissues (*) surrounding the metatarsal bones. Histopathologic analysis performed after transmetatarsal amputation showed AIDS-related KS without evidence of osteomyelitis.
angiomatosis, which may also exhibit skin lesions, bone erosions, and soft-tissue masses and is also treated with antibiotics, is not easy and may therefore require biopsy (89). Scintigraphy with sequential thallium and gallium scanning may be helpful, since, as mentioned earlier, gallium uptake is usually negative in KS but positive in infection and lymphoma (88).

Conclusions

Classic KS and endemic KS are variants of KS that rarely require radiologic evaluation due to their usually chronic course and stability of skin compromise. However, iatrogenic KS and AIDS-related KS, the most common forms of the disease, are frequently disseminated or symptomatic and may thus require imaging studies for both diagnosis and staging. It is important for the radiologist to be familiar with the spectrum of imaging manifestations of KS in various affected organs.

References


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