Table 1A.**5** (Cont.) Extra-axial lesions in the posterior cranial fossa (infratentorial)

Disease	MRI Findings	Comments
Fibrous dysplasia (Fig. 1A.**115)**	Expansile process involving the skull base with mixed low to intermediate signal on T1WI and variable mixed low, intermediate, high signal on T2WI; usually heterogenous enhancement.	Usually seen in adolescents and young adults. Can result in narrowing of neuroforamina with cranial nerve compression, facial deformities. Mono-ostotic and poly-ostotic forms (endocrine abnormalities may be associated, such as with McCune-Albright syndrome, precocious puberty.)
Paget disease (Fig. 1A.**116)**	Expansile sclerotic/lytic process involving the skull with mixed low to intermediate signal on T1WI and variable mixed low, intermediate, high signal on T2WI; variable heterogenous enhancement. Irregular/indistinct borders between marrow and inner margins of the outer and inner tables of the skull.	Usually seen in older adults. Can result in narrowing of neuroforamina with cranial nerve compression, basilar impression. With or without compression of brainstem.
Inflammatory		
Subdural/epidural abscess–Empyema (Fig. 1A.**72)**	Epidural or subdural collections with low signal on T1WI and high signal on T2WI; thin, linear peripheral zones of Gd-contrast enhancement.	Often results from complications related to sinusitis (usually frontal), meningitis, otitis media, ventricular shunts, or surgery. Can be associated with venous sinus thrombosis and venous cerebral or cerebellar infarcts, cerebritis, brain abscess. Mortality 30%.
Leptomeningeal infection/inflammation (Fig. 1A.**117)**	Single or multiple nodular enhancing lesions and/or focal or diffuse abnormal subarachnoid enhancement. Low to intermediate signal on T1WI and intermediate to high signal on T2WI. Leptomeningeal inflammation often best seen on postcontrast images.	Gd-contrast enhancement in the intracranial subarachnoid space (leptomeninges) usually is associated with significant pathology (inflammation and/or infection versus neoplasm). Inflammation and/or infection of the leptomeninges can result from pyogenic, fungal, or parasitic diseases as well as tuberculosis. Neurosarcoidosis results in granulomatous disease in the leptomeninges producing similar patterns of subarachnoid enhancement.
Eosinophilic granuloma (Fig. 2B.**34)**	Single or mutiple circumscribed soft-tissue lesions in the marrow of the skull associated with focal bony destruction/erosion with extension extracranially, intracranially, or both. Lesions usually have low to intermediate signal on T1WI and mixed intermediate to slightly high signal on T2WI, with Gd-contrast enhancement. With or without enhancement of the adjacent dura.	*Single lesion:* Commonly seen in males more than females (below 20 years). Proliferation of histiocytes in medullary cavity with localized destruction of bone with extension in adjacent soft tissues. *Multiple lesions:* Associated with syndromes such as: Letterer-Siwe disease (lymphadenopathy hepatosplenomegaly), children below 2 years; Hand-Schüller-Christian disease (lymphadenopathy, exophthalmos, diabetes insipidus) children aged 5–10 years.
Other:		
Vascular		
AVM	Lesions with irregular margins that can be located in the brain parenchyma–pia, dura, or both locations. AVMs contain multiple tortuous tubular flow voids on T1WI and T2WI. The venous portions often show Gd-contrast enhancement. GRE MR images and MRA using TOF or phase contrast techniques show flow signal in patent portions of the vascular malformation. Usually not associated with mass effect except in the case of recent hemorrhage or venous occlusion.	Supratentorial AVMs occur more frequently (80–90%) than infratentorial AVMs (10–20%). Annual risk of hemorrhage. AVMs can be sporadic, congenital, or associated with a history of trauma.
Dural AVM (Fig. 1A.**78)**	Dural AVMs contain multiple tortuous tubular flow voids on T1WI and T2WI. The venous portions often show Gd-contrast enhancement. GRE MR images and MRA using TOF or phase contrast techniques show flow signal in patent portions of the vascular malformation and areas of venous sinus occlusion or recanalization. Usually not associated with mass effect except in cases of recent hemorrhage or venous occlusion.	Dural AVMs are usually acquired lesions resulting from thrombosis or occlusion of an intracranial venous sinus with subsequent recanalization, resulting in direct arterial to venous sinus communications. Location in order of decreasing frequency: transverse, sigmoid venous sinuses; cavernous sinus; straight, superior sagittal sinuses.

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Table 10D.**1** Etiology of AVN of the hip

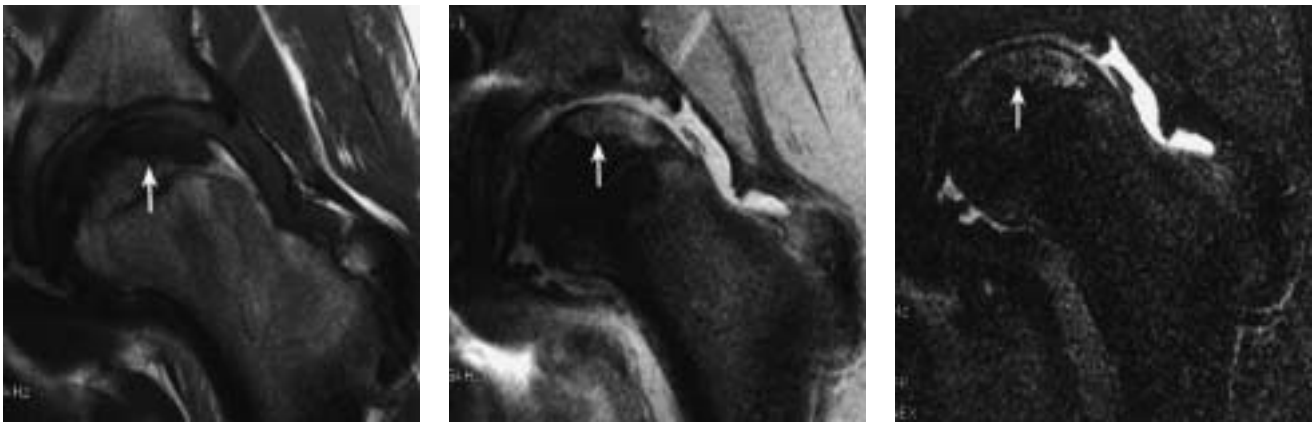
Disease	Comments
Femoral head dislocation	Posterior dislocations (85%) are invariably associated with either rupture of the ligament of head of femur or avulsion fracture of the femoral head. At a later stage sequelae of a femoral head fracture may mimic AVN. Development of AVN following hip dislocation is caused by compromise of the blood supply deriving from the circumflex femoral artery branches rather than the disruption of the foveal artery within the ligament of head of femur.
Femoral neck fracture	Intracapsular femoral neck fractures result in extensive interruption of blood flow to the femoral head. The only remaining blood supply may be within the ligament of head of femur, provided it was functional prior to fracture. The prevalence of AVN depends on the re-establishment of adequate blood flow from the remaining intact vasculature and the ingrowth of new vessels into the femoral head. Infarcts typically are located in the anterolateral weightbearing segment, but are often more extensive and may even involve the entire femoral head.
Sickle cell disease	Sludging of sickled erythrocytes within the sinusoidal vascular bed results in functional occlusion. If the process becomes sufficiently extensive, infarction occurs. Bilateral involvement and association with metadiaphyseal osteonecrosis (bone infarcts) is common.
Corticosteroid therapy and Cushing disease	Mechanisms implicated in the development of AVN of the femoral head include microscopic fat emboli, steroid-induced osteoporosis with subsequent microfractures, compromise of the sinusoidal blood flow by an increasing fat cell mass, and elevation in intraosseous marrow pressure within the femoral head.
Alcoholism	The alcoholic fatty liver may be associated with chronic fat emboli which may lodge in bone. Similar mechanisms to those for hypercortisolism have also been proposed. Metadiaphyseal infarcts are rare.
Pancreatitis	Fat necrosis in bone presumably results from circulating lipases. Metadiaphyseal infarcts are more common than AVN of the femoral head. Alcoholism may be associated.
Gaucher disease	Sinusoidal blood flow obstruction caused by lipid (glucocerebroside) containing histiocytes packing the sinusoids. Metadiaphyseal infarcts are common.
Caisson disease	Dysbaric AVN results from gas (nitrogen) embolization after rapid decompression. Metadiaphyseal infarcts are common.
Pregnancy	Venous stasis with increased intramedullary pressure secondary to impaired venous drainage caused by the enlarged uterus appears to be the etiologic factor. Transient osteoporosis of the hip, commonly occurring in the third trimester of pregnancy and predominantly affecting the left hip, has to be differentiated.
Radiation therapy	The effect of radiation is dose-dependent and mediated through damage to the vascular bed in the femoral head.
Collagen vascular disease	In rheumatoid arthritis and especially systemic lupus erythematosus AVN of the femoral head is caused by either corticosteroid therapy or vasculitis interrupting the arterial blood supply or both.
Renal transplantation	Corticosteroids and immunosuppressive agents used to control rejection appear to be the major etiologic factors. Underlying metabolic bone changes associated with chronic renal disease may be a contributing factor.
Chemotherapy	Patients with lymphoproliferative disorders are particularly at risk for developing AVN. The marrow packing effect of these disorders associated with tumor necrosis are thought to be responsible in the absence of corticosteroid therapy.
Gout and hyperuricemia	A mechanism for apparent association between gout and AVN is not known. Gout may be secondary to alcoholism or treated lymphoproliferative disease, two conditions with a known predisposition for AVN.
Arthritic hip	Synovitis and joint effusion may result in increased intra-articular pressure leading to an increased intraosseous pressure with venous stasis.
Idiopathic	Early conversion of hematopoietic to fatty marrow in the proximal femur and a prominent physeal scar separating the epiphysis from the metaphysis in the femoral head have both been implicated as risk factors for the development of AVN.

Avascular Necrosis of the Femoral Head

Avascular necrosis (AVN) of the femoral head is caused by an interruption of blood flow to the femoral head. It is frequently associated with osteonecrosis (bone infarcts) in other parts of the skeleton. A variety of disorders are associated with a high incidence of AVN of the femoral head and are summarized in Table 10D.**1**.

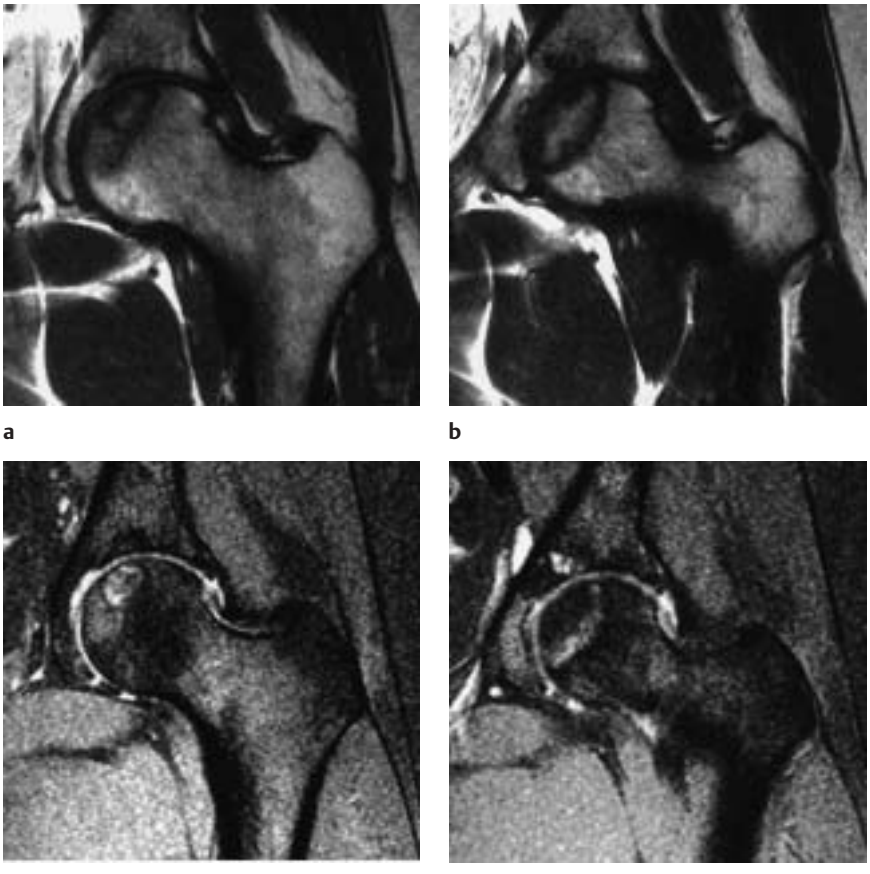
MRI represents a very sensitive method for early diagnosis of AVN of the femoral head (Figs. 10D.**3–5**). Early MRI abnormalities depend on the alterations of bone marrow fat cells dying within 2 to 5 days after the ischemic event and the development of a hyperemia response in viable tissue and adjacent to the infarct. The increased vascularity at the interface between viable and necrotic tissue subsequently progresses to inflammation, granulation-tissue formation, fibrosis, and eventually new bone formation. Variations of MRI patterns in AVN are indicative of individual differences in extent, distribution, and age of the infarct as well as the host response. Focal homogeneous or inhomogeneous abnormalities predominate, with at least one component of diminished signal intensity on T1WI. Abnormal femoral head AVN patterns on T1WI include: 1) a well-defined homogeneous area of low signal intensity adjacent to the articular surface; 2) a band of low signal intensity crossing the femoral head; 3) heterogeneous loss of signal intensity in the femoral head containing foci of normal signal intensity; and 4) a ring of low signal intensity

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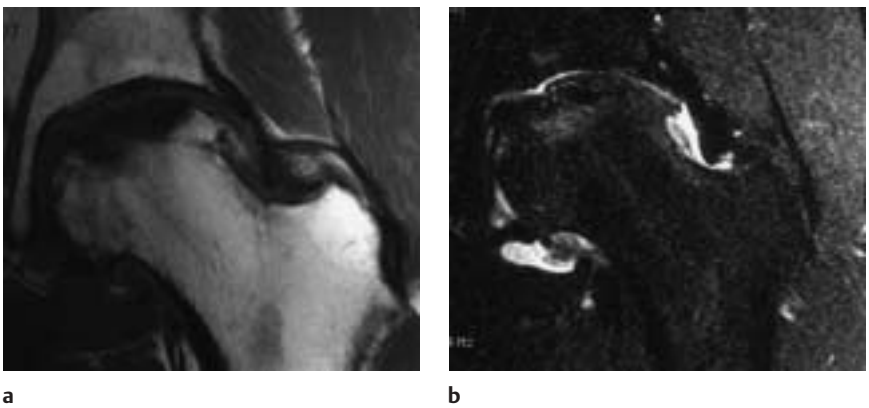
a b c

Fig. 10D.**3** **AVN of the hip.** A well-defined area of low (**a**: T1WI), intermediate (**b**: PDWI), and relatively high (**c**: T2WI, FS) signal intensity (arrow) is seen adjacent to the weight-bearing articular surface of the femoral head (coronal planes).



a b c d

Fig. 10D.**4** **AVN of the hip.** A ring (**a**) or band (**b**) of low signal intensity separates a subarticular region of normal signal intensity from the unaffected marrow in the femoral head (T1WI, coronal). **c, d** A "double line" sign is seen at the interface between ischemic and nonischemic bone, consisting of an outer zone of low signal intensity and an inner zone of high signal intensity (T2WI, coronal).



a b

Fig. 10D.**5** **AVN of the hip.** A collapsed articular surface with fragmentation is seen (**a**: T1WI, coronal; **b**: T2WI, FS, coronal).

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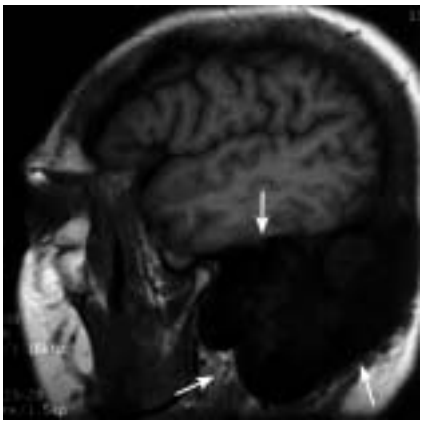


Fig. 1A.**115** **Fibrous dysplasia.** **a** Sagittal T1WI shows a well-circumscribed expansion of the skull with mixed low to intermediate signal (arrows).

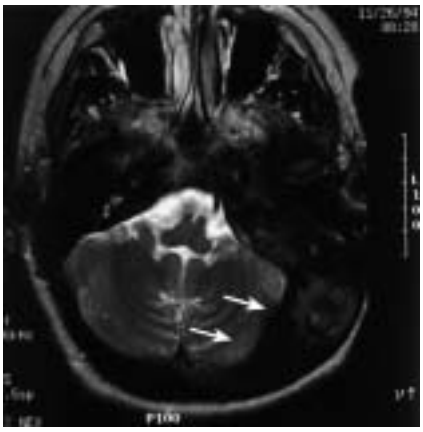


Fig. 1A.**115 b** Axial T2WI shows enlargement of the left petrous and occipital bones with delineated borders, containing mixed low and intermediate signal (arrows).

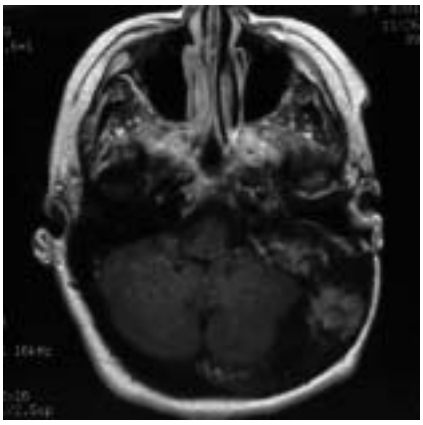


Fig. 1A.**115 c** Postcontrast axial T1WI shows heterogeneous enhancement in portions of the fibrous dysplasia.

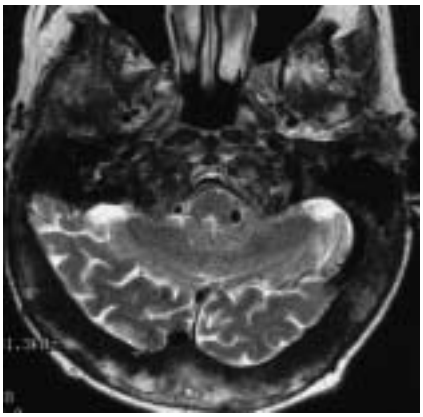


Fig. 1A.**116** **Paget disease.** Axial T2WI shows an expansile sclerotic/lytic process involving the occipital bone with mixed low, intermediate, and high signal. Irregular/indistinct borders are seen between the marrow and outer and inner tables of the skull.

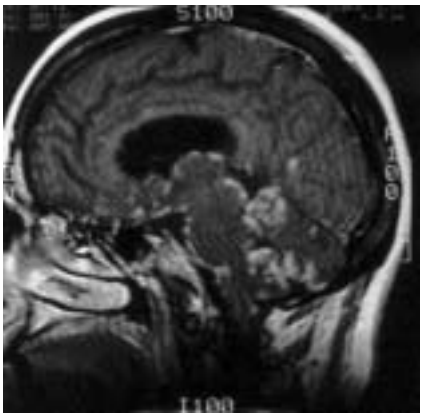


Fig. 1A.**117** **a** **Neurosarcoidosis.** Postcontrast sagittal. T1WI show multiple nodular enhancing lesions in the leptoreninges.

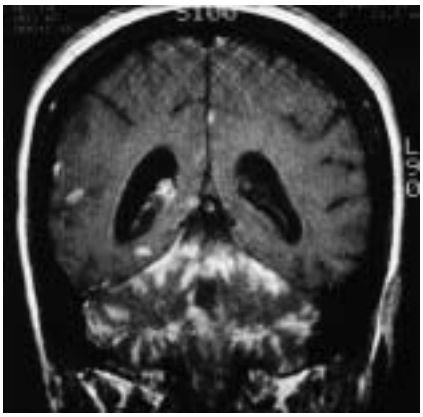


Fig. 1A.**117 b** Postcontrast coronal T1WI show multiple small nodular enhancing lesions in the supratentorial and infratentorial leptomeninges.

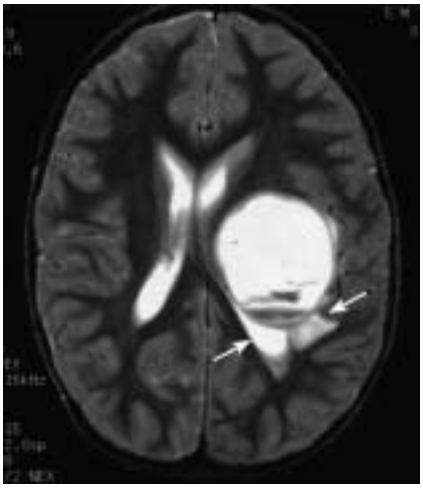


Fig. 1A.**118** **Low-grade astrocytoma.** **a** Axial T2WI shows a cystic lesion with predominantly high signal containing a small fluid level (low signal) (arrows) in the left cerebral hemisphere.

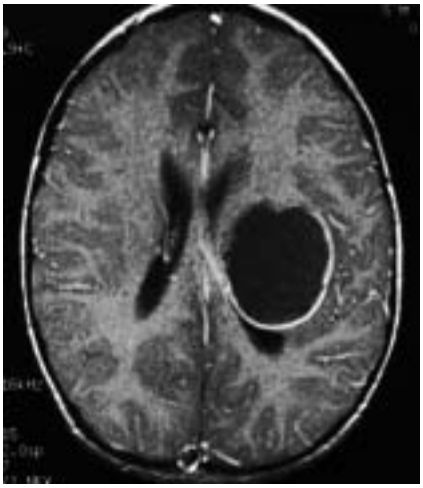


Fig. 1A.**118 b** Postcontrast axial T1WI shows mild peripheral rim enhancement of the cystic lesion.

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