

FIGURE 441e-55 Krabbe's disease (Chap. 432e). Axial and coronal T2-weighted MRIs (**A, B**) show increased T2 signal involving predominantly the posterior white matter bilaterally (*arrows*) with sparing of the subcortical U-fibers (*arrowheads*). MR spectroscopy of the left parietal white matter (**C**) shows markedly decreased N-acetylaspartate (*large arrow*) and increased lactate/lipids (*small arrow*), consistent with severe neuronal injury.

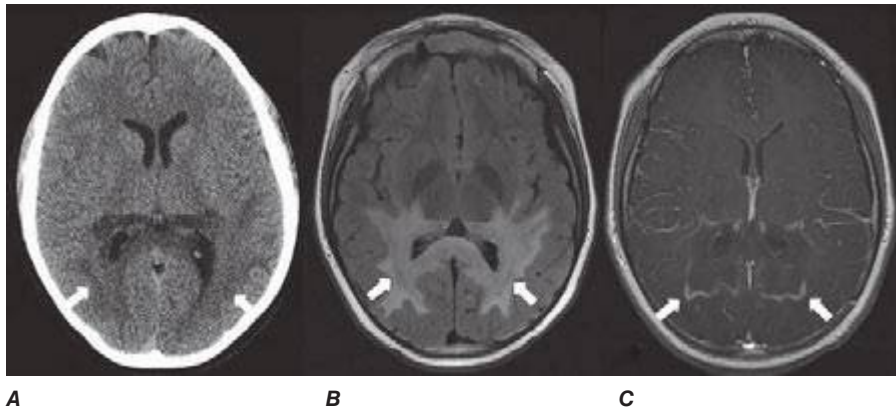


FIGURE 441e-56 X-linked adrenoleukodystrophy (Chap. 459). Axial unenhanced CT (**A**) demonstrates areas of attenuation involving the posterior white matter bilaterally (*arrows*). Axial T2 FLAIR MRI (**B**) displays increased T2 signal consistent with edema (*arrows*). Axial T1-weighted image postgadolinium (**C**) shows peripheral enhancement of the parietal lesions bilaterally (*arrows*). These findings are typical of adrenoleukodystrophy.

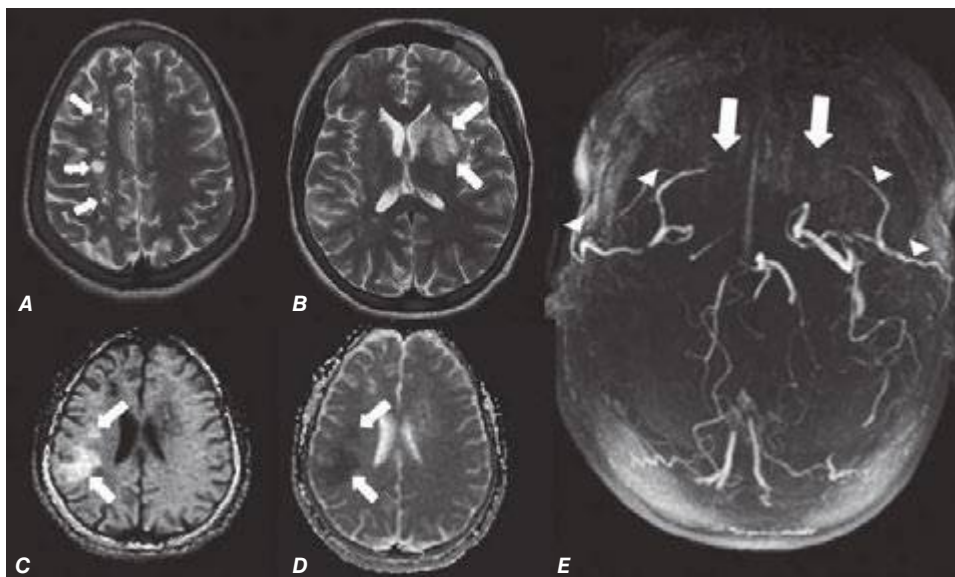


FIGURE 441e-57 Sickle cell disease and moyamoya disease (Chap. 446). Axial T2-weighted MRI (**A, B**) shows multiple small areas of encephalomalacia from prior infarcts in the watershed zones between the anterior and middle cerebral artery territories (*small arrows*). There is also an area of edema involving the left basal ganglia from an evolving subacute infarct (*arrow*). An axial diffusion-weighted image (**C**) with corresponding ADC map (**D**) shows an area of restricted diffusion in the right frontoparietal region, consistent with an acute infarct (*arrows*). Time-of-flight MR angiography (**E**) shows absence of flow in the distal internal carotid arteries and proximal middle cerebral arteries (*arrows*) due to moyamoya disease. Also note that this patient is status post–bilateral encephalo-duro-arterio-synangiosis (EDAS) (*arrowheads*), a surgical procedure to create indirect anastomosis between branches of the external carotid artery with distal branches of the middle cerebral artery.