Stroke differential diagnosis and mimics: Part 2

Shahmir Kamalian, MD; Shervin Kamalian, MD; Daniel J. Boulter, MD; Michael H. Lev, MD; R. Gilberto Gonzalez, MD, PhD; Pamela W. Schaefer, MD

Editor's note: This is the second part of a 2-part article. The first part was published in the November 2015 print issue of Applied Radiology.

In Part 1, the authors reviewed the typical imaging features of ischemic stroke at different ages and considered numerous pathologies that can mimic ischemic stroke such as seizure, migraines, tumors, and toxic-metabolic abnormalities. The MR characteristics of ischemic stroke were compared to imaging mimics based on six patterns to topographic distribution

In this second part, further anatomic localizations of ischemic stroke versus non-ischemic pathology with similar MRI appearances are reviewed. These include white matter distribution, such as multiple sclerosis, scattered punctate foci of abnormality including cardiac emboli or vasculitis leading to stroke versus mimics as diffuse axonal injury or fat-emboli, and a border zone pattern including watershed infarcts versus Posterior Reversible Encephalopathy Syndrome (PRES) as one mimic.

Many of the pathologies simulating ischemic infarction also demonstrate restricted diffusion as occurs with hypoglycemia, carbon monoxide poisoning, and early stages of herpes simplex encephalitis., which makes the differentiation from ischemic infarct even more difficult

White matter abnormality

CADASIL

Cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy (CADASIL) is an inherited disease that affects smooth muscles of the penetrating cerebral and leptomeningeal arteries. The symptoms usually start in the third or fourth decades with migraine headaches, TIA and ischemic strokes. In later decades, they develop dementia. MRI reveals confluent patchy supratentorial white matter T2 hyperintensities and lacunar infarcts in deep gray matter, midbrain and pons.[133-136] The hallmark of CADASIL is involvement of bilateral anterior temporal white matter and external capsules. Acute lesions demonstrate restricted diffusion (Figure 16).

Dr. Shahmir Kamalian *is Assistant Professor, Department of Radiology at University of Massachusetts Medical School, Worcester, MA;* ***Dr. Shervin Kamalian*** *is Radiology Resident, Department of Radiology at Mount Auburn Hospital, Cambridge, MA;* ***Dr. Boulter*** *is Assistant Professor, Department of Radiology at The Ohio State University, Columbus, OH;* ***Dr. Lev*** *is Director of Emergency Imaging and Professor of Radiology,* ***Dr. Gonzalez*** *is Director of Neuroradiology and Professor of Radiology, and* ***Dr. Schaefer*** *is Associate Director of Neuroradiology and Associate Professor of Radiology, all at Massachusetts General Hospital/Harvard Medical School, Boston, MA.*

Susac's syndrome

Susac's syndrome is rare and involves small vessels of the brain, retina and inner ear and presents with focal neurologic deficits as well as visual and hearing loss. Mean age of onset is 32 years and it is more common in women.[137] Numerous small T2 hyperintense foci are seen in the supratentorial white matter, corpus callosum, and less often the deep gray matter.[138] Acute lesions demonstrate restricted diffusion and in approximately 50% enhance (Figure 17).[138] Susac's syndrome can be mistaken for multiple sclerosis due to a similar pattern of white matter involvement.[137] One difference is that the callosal lesions associated with Susac's syndrome tend to involve the full thickness of the corpus callosum while those associated with multiple sclerosis tend to involve only the inferior surface of the corpus callosum. In addition, approximately 25% of cases demonstrate leptomeningeal enhancement which is not seen with multiple sclerosis.

Metronidazole toxicity

Metronidazole is usually used for treatment of anaerobic infections and for preventing recurrence in Crohn's disease. It is generally safe, but it rarely may result in peripheral neuropathies and central neurotoxicity at dosages more than 2 g/day when used for prolonged periods.[139, 140] Central neurotoxicity is characterized by symmetric bilateral T2 hyperintensity in

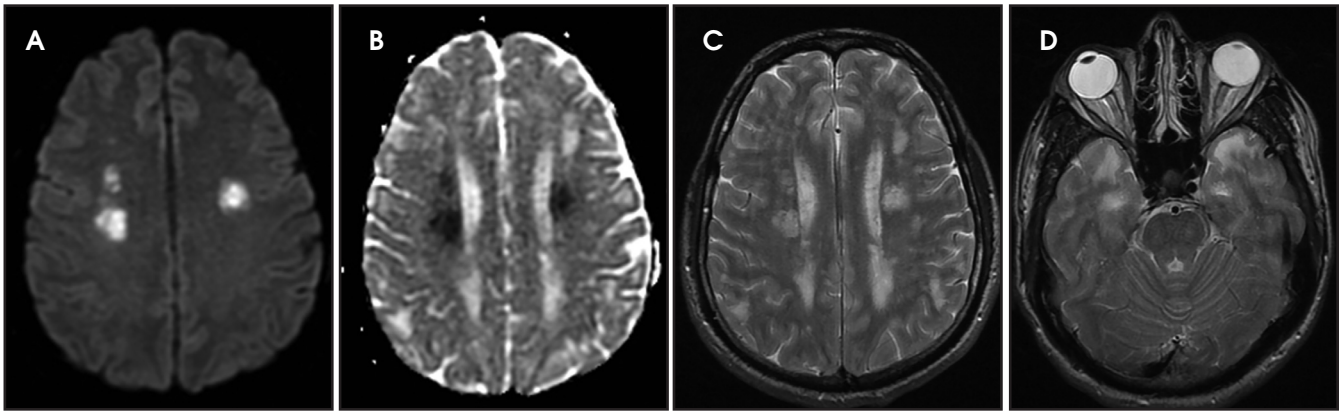

FIGURE 16. *A 40-year-old man presented to ER with abulia and left facial droop. MRI demonstrated multiple foci with restricted and increased diffusion in the bilateral centrum semiovale and corona radiata (A, DWI and B, ADC map) with associated T2 hyperintense foci (C and D, T2WI) in the bilateral periventricular white matter and bilateral anterior temporal lobes. Notch 3 mutation confirmed presence of CADASIL.*

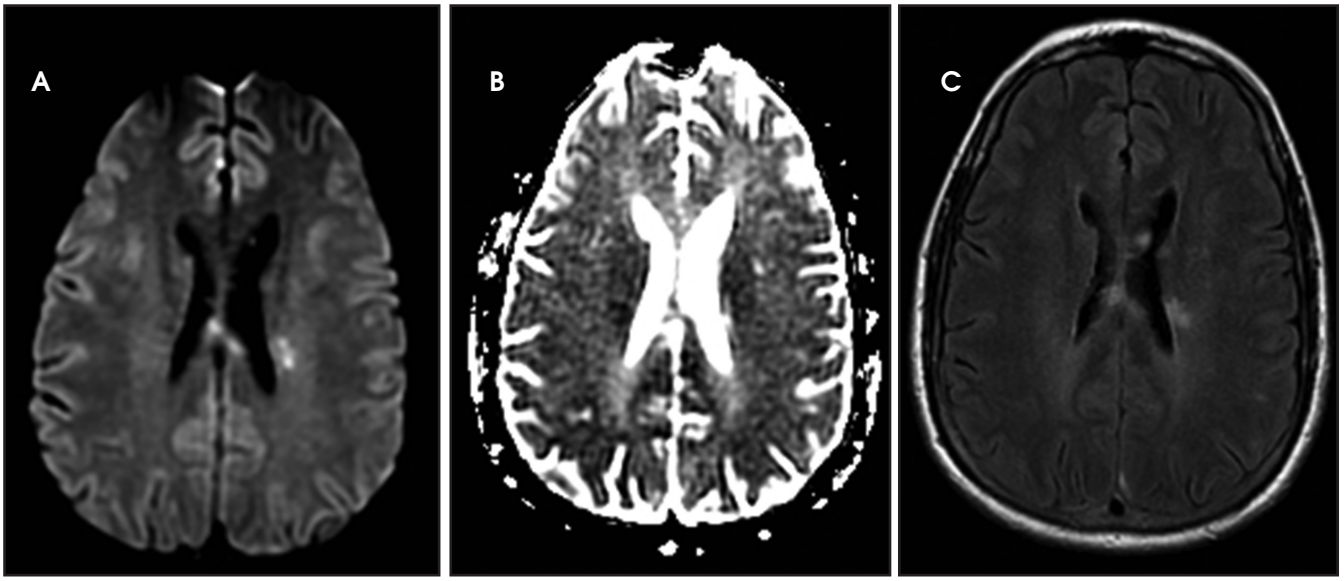

FIGURE 17. *A 39-year-old man with Susac's syndrome presented with headache, apathy and amnesia. MRI demonstrated multiple foci of restricted diffusion (A, DWI and B, ADC map) and T2/FLAIR hyperintensity (C) in the corpus callosum and supratentorial white matter. On further evaluation he was found to have retinal vasculitis and sensorineural hearing loss.*

dentate nuclei, midbrain, corpus callosum, pons, medulla, and subcortical cerebral white matter and basal ganglia in decreasing order. The gray matter lesions are associated with vasogenic edema and are typically reversible, but the white matter lesions are associated with low ADC due to cytotoxic edema (Figure 18) and may be irreversible. The mechanism of metronidazole induced neurotoxicity is not clear. It is suggested that the conversion of metronidazole to a thiamine analog which inhibits thiamine pyrophosphokinase and malabsorption may play role in metronidazole induced toxicity.[141-144]

Methotrexate toxicity

Methotrexate neurotoxicity occurs in 1 to 3% of treated patients, especially after administration of intrathecal or intermediate and high doses of intravenous or oral methotrexate[145] and can be acute, subacute or chronic. The subacute form occurs within 5–14 days and may mimic stroke or TIA, but generally symptoms completely resolve within days. Patients present with seizures and neurologic deficits such as aphasia, hemiparesis and ataxia. The MRI findings include transient restricted diffusion and/or T2 hyperintensity in the periventricular white matter, particularly the corona radiata and centrum semiovale (Figure 19). The mechanism of restricted diffusion in methotrexate induced subacute encephalopathy may be related to excitotoxic injury rather than demyelination.[146-148]

Heroin leukoencephalopathy

Heroin leukoencephalopathy is caused by inhalation of heroin vapors, a practice known by a Chinese phrase "chasing the dragon" and has a mortality rate of 23%. MRI findings are pathognomic and include symmetric and confluent bilateral T2-hyperintensity within the cerebral and cerebellar white

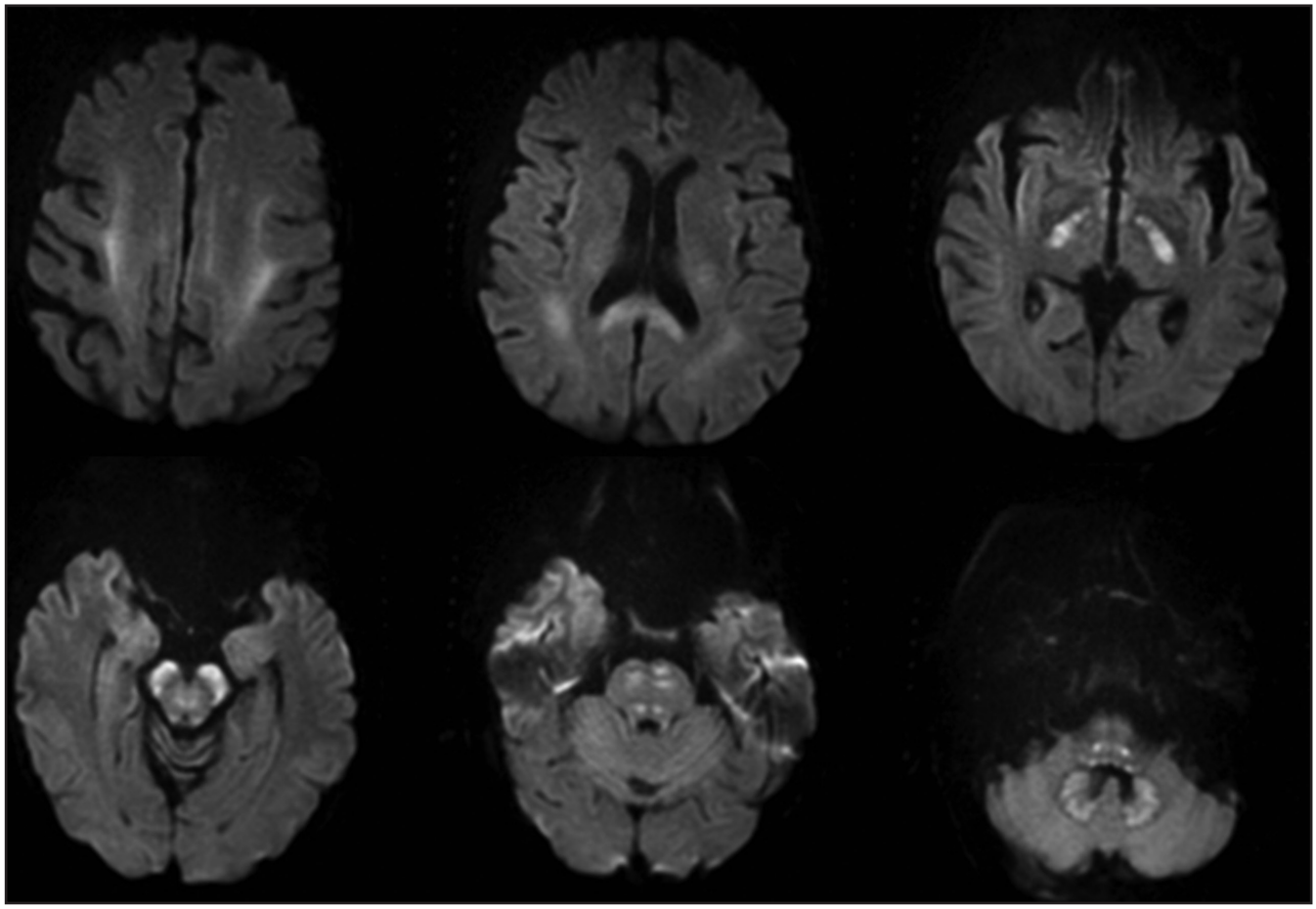

FIGURE 18. *A 68-year-old woman developed seizures after resection of an infected aortic graft, while she was being treated with metronidazole for a presacral abscess. MRI demonstrated symmetric restricted diffusion in the bilateral subcortical white matter, splenium, internal capsule, midbrain, pons and dentate nuclei. The areas of restricted diffusion completely resolved after discontinuation of metronidazole (only DWI images are shown).*

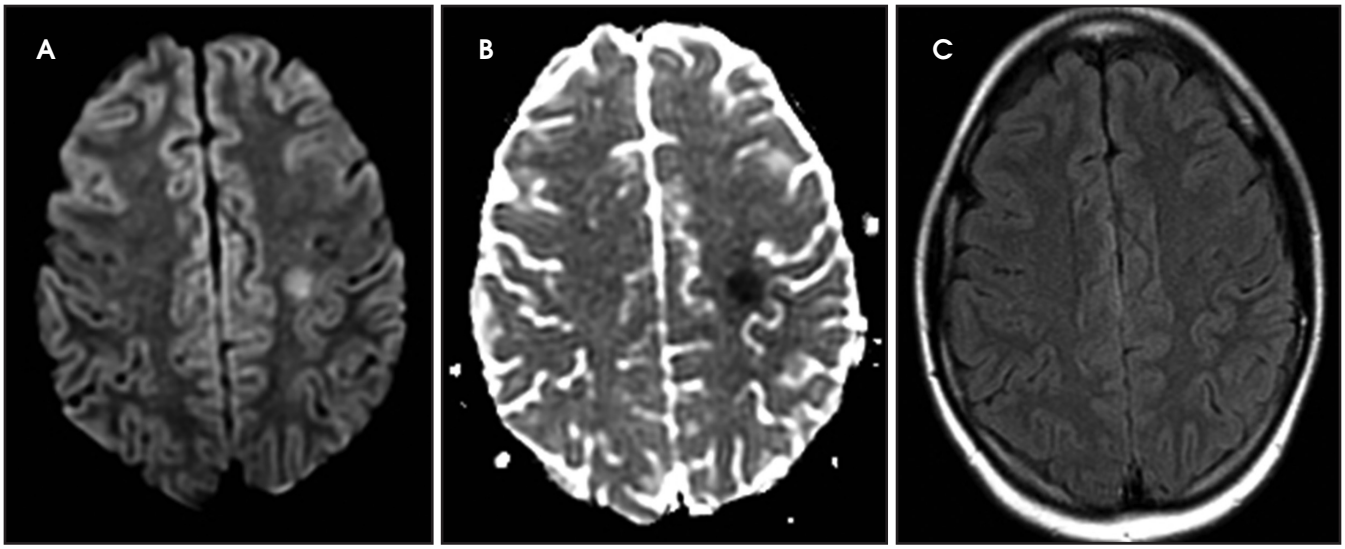

FIGURE 19. *A 26-year-old woman with ALL presented with right arm weakness and transient slurred speech 10 days after treatment with intrathecal methotrexate. MRI demonstrated a rounded focus of restricted diffusion (A, DWI and B, ADC map) in the left precentral gyrus with no obvious T2 abnormality (C, FLAIR). The follow up MRI a few days later (not shown) demonstrated complete resolution of restricted diffusion, most likely on the basis of subacute methotrexate toxicity.*

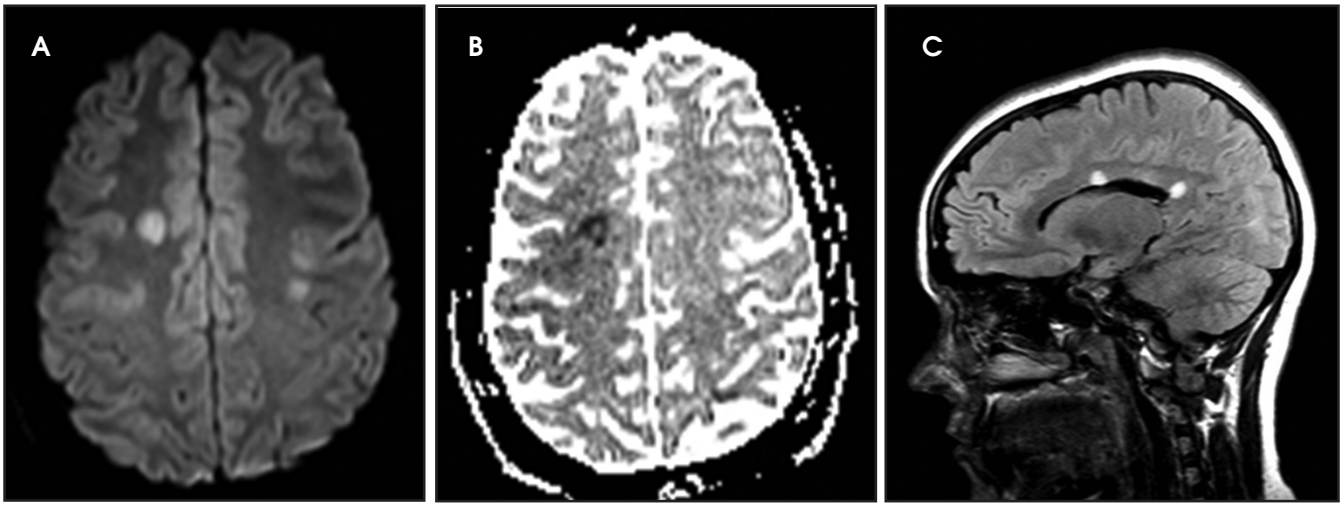

FIGURE 20. *A 21-year-old female with relapsing remitting MS. Initial MRI at age 17 for evaluation of mild concussion syndrome suggested incidental multiple sclerosis with multiple supratentorial T2 hyperintense foci in a characteristic pattern (C, FLAIR) and a lesion with restricted diffusion (A, DWI and B, ADC) in the right frontal white matter.*

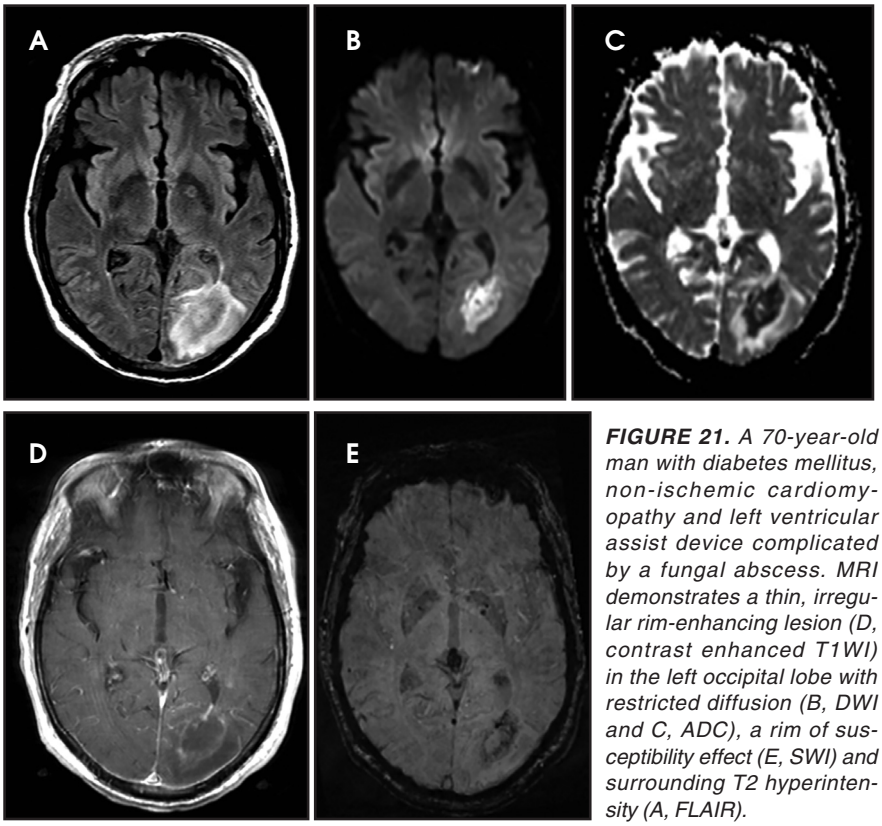

FIGURE 21. *A 70-year-old man with diabetes mellitus, non-ischemic cardiomyopathy and left ventricular assist device complicated by a fungal abscess. MRI demonstrates a thin, irregular rim-enhancing lesion (D, contrast enhanced T1WI) in the left occipital lobe with restricted diffusion (B, DWI and C, ADC), a rim of susceptibility effect (E, SWI) and surrounding T2 hyperintensity (A, FLAIR).*

matter and posterior limbs of internal capsules. There is a predilection for parieto-occipital white matter. The subcortical U fibers, dentate nuclei and anterior limbs of internal capsules are preserved. Restricted diffusion in the acute phase correlates with initial development of small vacuoles within the myelin lamellae.[16, 149-151] In sub-acute and chronic stages, these vacuoles become larger and coalesce, resulting in increased diffusibility of water molecules.[16, 150]

Multiple sclerosis

Multiple sclerosis may present with sudden-onset focal neurologic deficits such as aphasia or hemiplegia.[152, 153] The typical MRI findings are T2 hyperintense lesions in the periventricular, deep and juxtacortical white matter. Acute lesions are usually associated with increased diffusion, but they may occasionally show transient (2–7 days) restricted diffusion (Figure 20). The restricted diffusion is characteristically seen in the periphery of the lesions, but may be central. These lesions are most often associated with contrast enhancement similar to subacute stroke, indicating active inflammation.[154, 155] Cytotoxic edema due to excitotoxic injury to oligodendrocytes, myelin sheaths and axons has been proposed as the cause of restricted diffusion in multiple sclerosis.[156-158]

Infectious cerebritis and abscess

Clinically cerebritis or brain abscess may be mistaken as stroke. Early cerebritis may present with an ill-defined T2-hyperintense lesion with restricted diffusion and minimal or absent contrast enhancement (Figure 21).[159, 160] Bacterial abscesses usually have a well-defined enhancing rim with central restricted diffusion and surrounding edema, an appearance that can be seen but is unusual for late subacute stroke. However, fungal abscesses may have a less well defined rim and are more likely to be confused with stroke. The cause for restricted diffusion in pyogenic brain abscess is attributed to restricted water diffusion in purulent

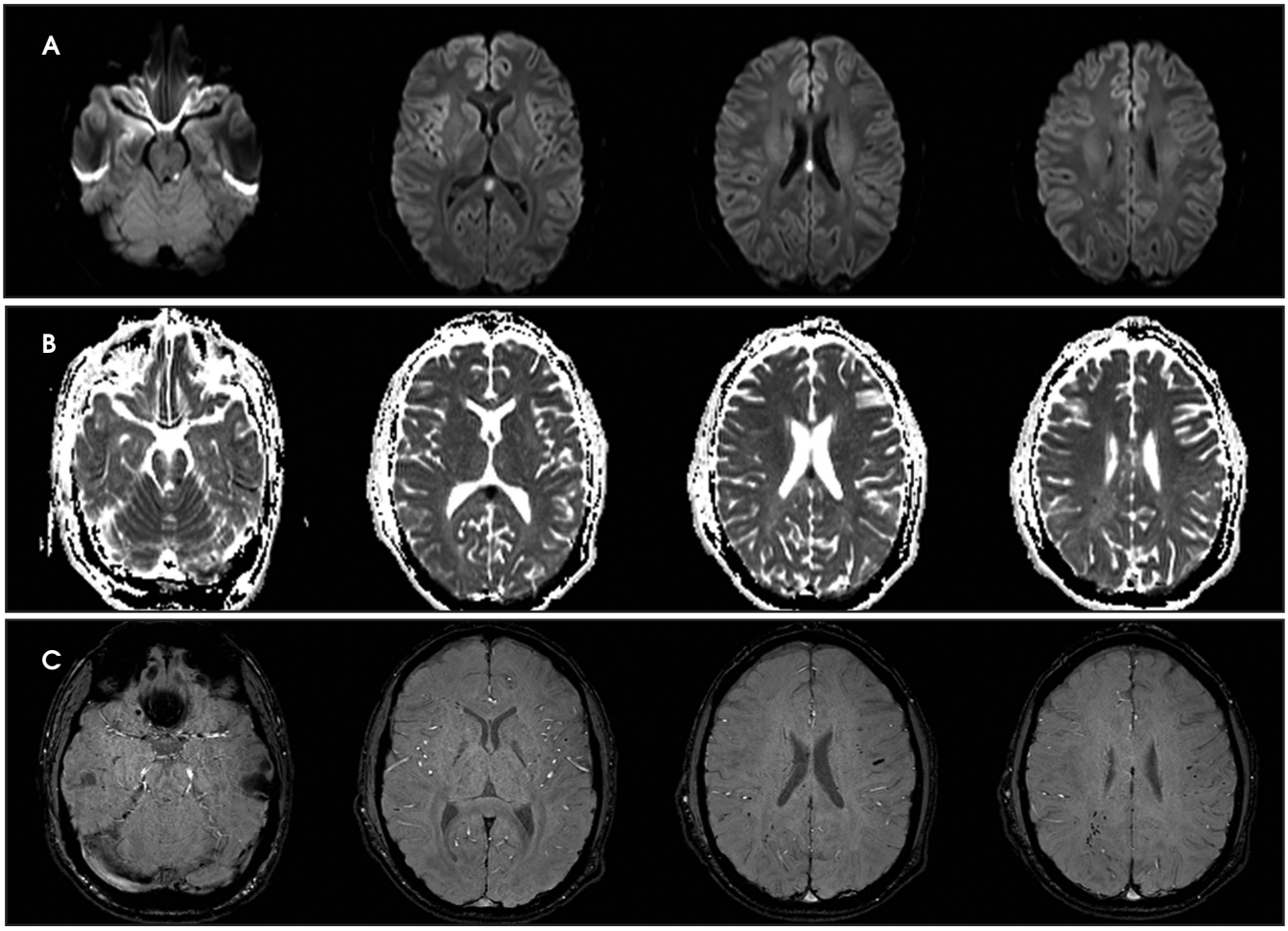

FIGURE 22. *A 19-year-old man unrestrained driver in a high speed motor vehicle collision. MRI demonstrated foci of restricted diffusion (A, DWI and B, ADC) in the corpus callosum, in the left superior cerebellar peduncle and posterior corona radiata. There are multiple punctate foci of susceptibility effect (C, SWI).*

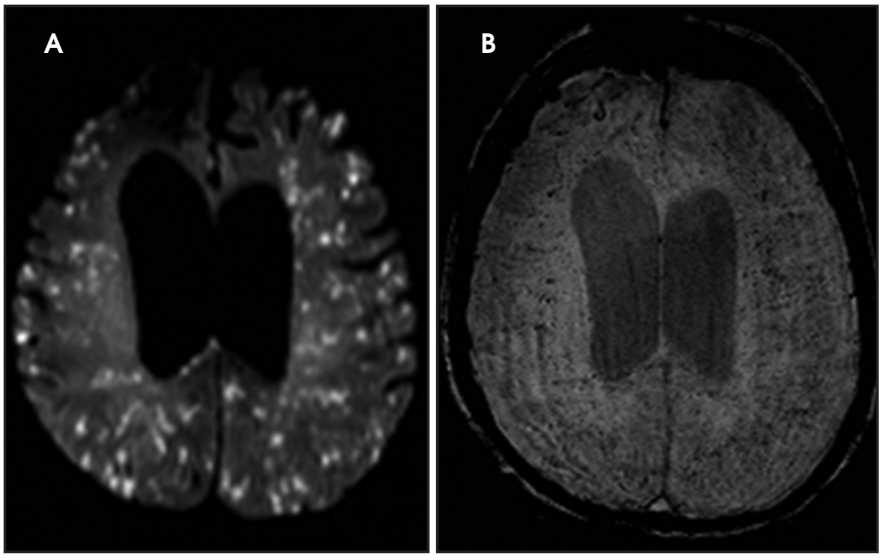

FIGURE 23. *A 71-year-old status postremote resection of right frontal glioblastoma multiforme was found unresponsive one day after left hip hemiarthroplasty. MRI shows innumerable foci or restricted diffusion (A, DWI) throughout the brain with associated susceptibility effect (B, SWI), most likely related to fat embolism.*

fluid.[161] Decreased diffusion in case of cerebritis may be related to cytotoxic edema due to necrotizing angiitis or venous thrombosis.[159, 160]

Scattered punctate foci

Strokes

Cardioembolic sources are the most common cause, but other etiologies such as vasculitides can produce a similar pattern.[162]

Diffuse axonal injury

Diffuse axonal injury (DAI) is a widespread disruption of the axons caused by abrupt acceleration, deceleration or rotational injury in severe head trauma.[163] FLAIR shows multiple small lesions in the gray-white matter junction, corpus callosum, fornix, along the cerebral white matter tracts, and the dorsolateral

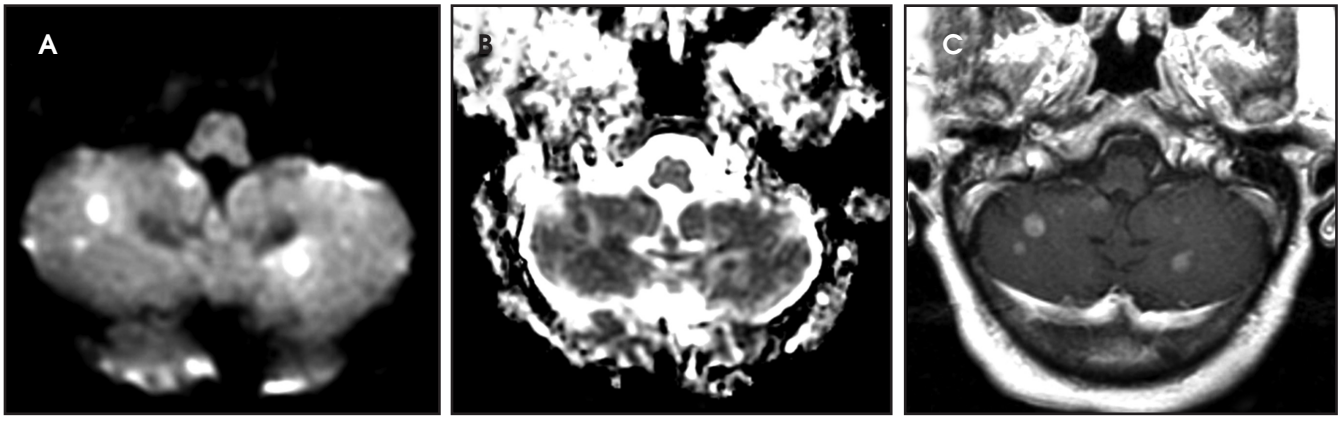

FIGURE 24. *A 51-year-old female with small cell lung carcinoma presented with sudden onset of nausea and vomiting every morning. There are multiple foci of restricted diffusion in the cerebellum (A, DWI and B, ADC map) which demonstrate contrast enhancement (C, contrast enhanced T1WI).*

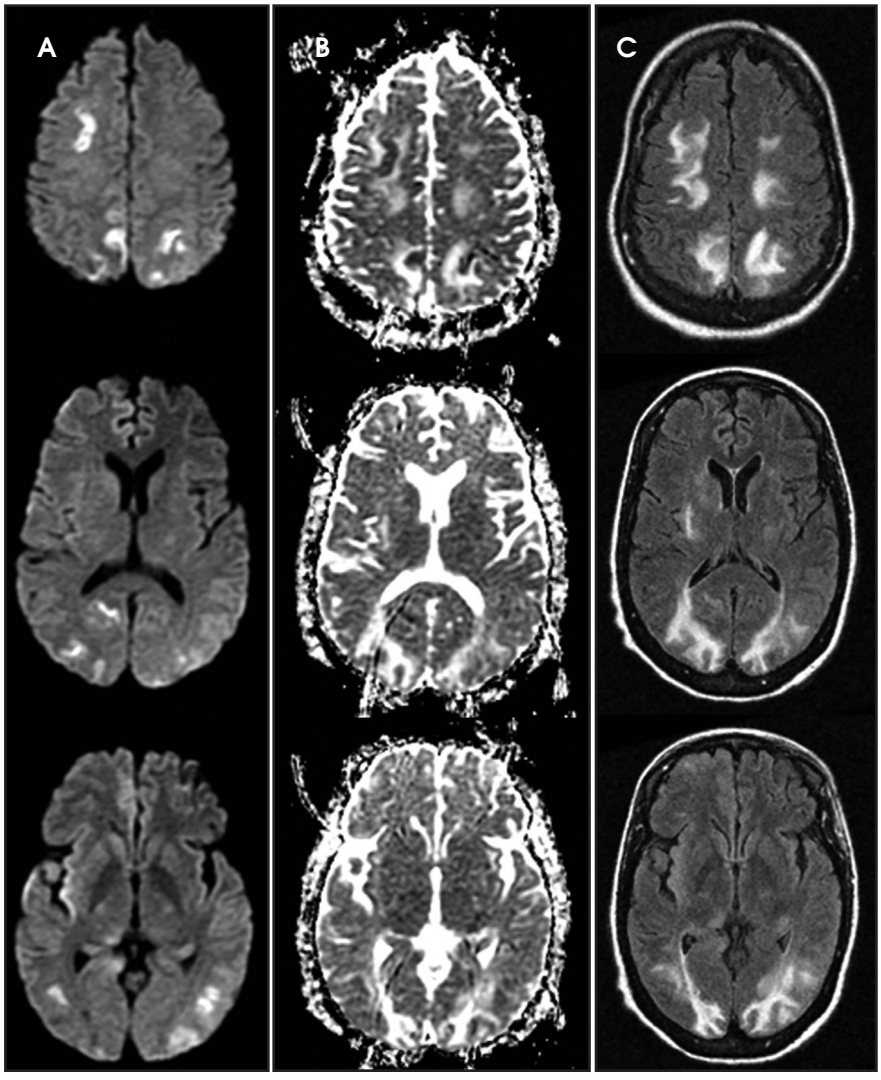

FIGURE 25. *A 40-year-old woman with systemic lupus erythematosis (SLE) and uncontrolled hypertension presented with seizures and visual impairment. MRI shows FLAIR signal abnormality (C) in the bilateral occipital, frontal and parietal lobes in predominantly a borderzone distribution with multiple areas of increased diffusion characterized by increased signal on the DWI, A and ADC, B maps.*

brainstem (Figure 22).[163,164] Many of these lesions also have restricted diffusion, with a variable time course that may persist for days or weeks. The restricted diffusion may be due to ischemia related to traumatic microvascular injury, swelling of the retracted axons or Wallerian degeneration of crossing interhemispheric fibers.[165] T2*-weighted GRE images demonstrate additional lesions that are hemorrhagic.[166]

Fat emboli

Fat embolism typically occurs in the setting of a recent long bone fracture, orthopedic procedures or bone marrow infarction in sickle cell disease.[167] It usually presents with hypoxia, confusion, neurologic deficits, and petechial rash. MRI shows numerous punctate foci of restricted diffusion throughout the brain.[168] The release of fatty acids results in endothelial injury and microhemorrhages which manifest as numerous punctate foci of susceptibility, far more than would be expected for thrombo-embolic infarcts (Figure 23).[169,170]

Metastases

Highly cellular metastases, especially small-cell lung carcinoma can show restricted diffusion due to dense cell packing and be confused with acute or subacute embolic strokes (Figure 24). Some distinguishing features are presence of surrounding vasogenic edema, peripheral enhancement and

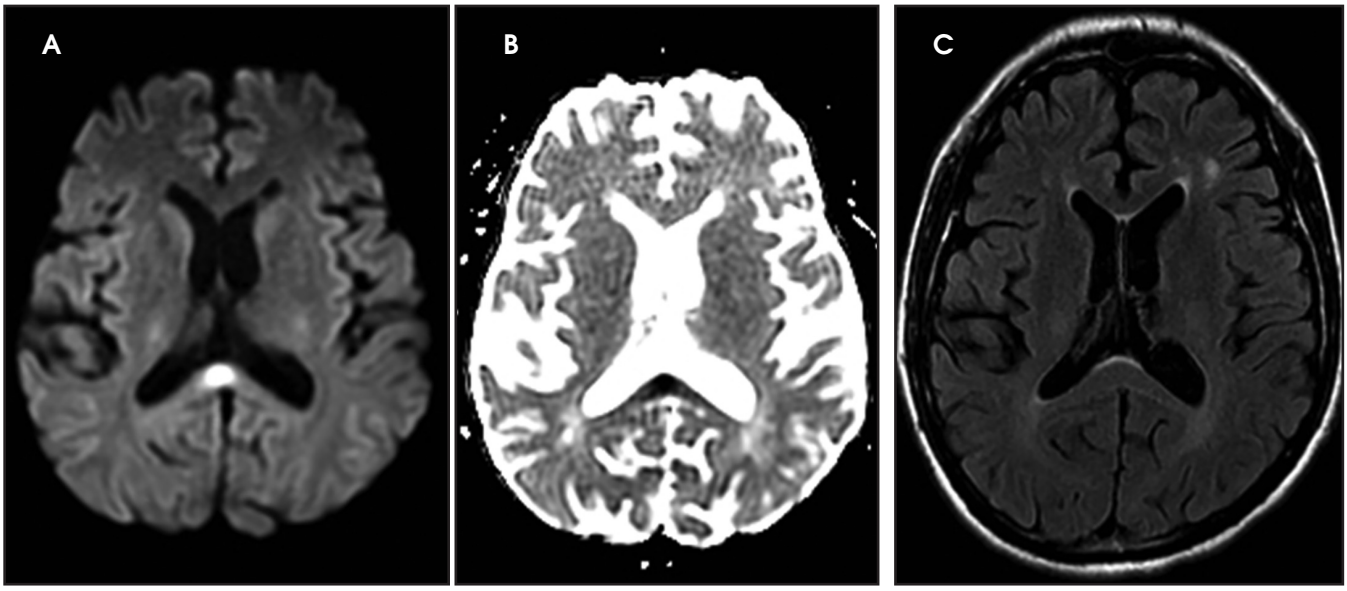

FIGURE 26. *A 53-year-old man with history of seizure disorder on lamotrigine and carbamazepine, admitted with confusion and tremors. MRI demonstrates focal area of markedly restricted diffusion (A, DWI, B, ADC) in the splenium of the corpus callosum with no associated FLAIR hyperintensity (C). The restricted diffusion completely resolved on follow-up MRI supporting toxic etiology.*

additional extraparenchymal disease (such as calvarial lesions).

Border zone pattern

Border zone infarction

Border zone infarctions are defined as ischemia between two major cerebral arterial territories and it occurs due to hypotension in patients with severe carotid stenosis, Moya-moya disease, and reversible cerebral vasoconstriction syndrome.[171, 172] Posterior reversible encephalopathy syndrome (PRES) and cerebral hyperperfusion syndrome are among mimics.

Moya-moya disease

Moya-moya disease is characterized by idiopathic, chronic and progressive stenosis of distal internal carotid and proximal anterior and middle cerebral arteries, with formation of vascular collateral networks similar to a "puff of smoke" (or "moya-moya" in Japanese). A number of conditions can cause a moya-moya-like appearance, including radiation, neurofibromatosis type 1, Down syndrome, sickle cell disease, trauma, and slowly progressive atherosclerosis. Infarctions typically occur in an anterior circulation border zone or basal ganglia distribution. Cerebral hemorrhage into the basal ganglia also occurs due to the fragility of lenticulostriate vessels.

Reversible cerebral vasoconstriction syndrome (RCVS)

RCVS is a poorly understood disease characterized by vasospasm not due to subarachnoid hemorrhage or trauma. Patients usually present with acute or recurrent severe "thunderclap" headaches, often with focal neurologic deficits. Resolution of the vasospasm over a period of weeks to three months confirms the diagnosis.[173, 174] About half of the patients develop ischemic stroke, usually in a border zone pattern.[173, 175] Intracranial hemorrhage (parenchymal or subarachnoid) and PRES-like edema can be seen in up to one third of the patients.[173, 176]

Posterior reversible encephalopathy syndrome (PRES)

Posterior reversible encephalopathy syndrome (PRES) is characterized by vasogenic cerebral edema due to loss of vascular autoregulation and capillary leakage[177] and mimics late subacute stroke. Patients typically present with headaches, cortical visual symptoms, seizures, and confusion.[177] It can be triggered by hypertension, eclampsia/pre-eclampsia, critical medical illness, and immunosuppressants.[177, 178] Imaging typically demonstrates T2-FLAIR hyperintensity with elevated diffusion in the bilateral occipital and parietal lobes (mimicking posterior circulation infarctions) and/or in a borderzone distribution between the anterior and middle cerebral arteries (mimicking borderzone infarctions).[179-181] Restricted diffusion, due to cytotoxic edema, is present in 10–25% patients and portends a worse outcome (Figure 25).[181-183] Intraparenchymal hemorrhage is seen in 15% of cases.

Hyperperfusion syndrome

Hyperperfusion syndrome occurs after carotid endarterectomy in 1-3% cases, likely due to impaired cerebral autoregulation. It presents with triad of headache, seizures and neurological deficits. It is typically characterized by vasogenic edema with elevated diffusion in the borderzone between the anterior and middle cerebral arteries and increased cerebral blood flow. The time course and seizures help differentiate the condition from late subacute infarction.[184]

Splenial lesions

Splenial lesions can be seen in many conditions (Figure 1), either as an isolated lesion or as part of a more widespread distribution pattern.

Antiepileptic drug treatment or withdrawal

A number of antiepileptic medications are associated with asymptomatic restricted diffusion in the splenium (Figure 26).[185] These lesions resolve even with continuation of treatment and are seen in patients on monotherapy with phenytoin, carbamazepine, oxcarbazepine, lamotrigine, vigabatrin, valproate and phenobarbital.[185]

Marchiafava–Bignami disease

Marchiafava-Bignami disease is a rare disease that occurs mostly in alcoholic men with malnutrition. Symptoms range from psychiatric disturbances, hemiparesis, aphasia and seizures to sudden onset of coma. The body of the corpus callosum, genu and then splenium or the entire corpus callosum are involved in a decreasing order.[186] The lesions are typically T2 hyperintense and may show restricted diffusion.[187]

Radiation therapy

Focal T2-FLAIR hyperintense signal in the splenium of the corpus callosum is a common finding after brain radiation therapy[188] and can be associated with restricted diffusion. It has been proposed that the splenium is more sensitive to radiation induced changes and the pathogenesis of these alterations is likely related to an indolent process, possibly demyelination.[188]

Conclusion

Stroke mimics are common in the emergency department and some of these patients may be treated with intravenous tPA. Despite many clinical and imaging overlaps, a pattern-based approach provides a reasonably accurate method to diagnose of many of these conditions and facilitate appropriate and timely management.

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