CT and MR findings of systemic lupus erythematosus involving the brain: Differential diagnosis based on lesion distribution

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Authors: K. Tsuchiya, M. Imai, K. Honya, T. Nitatori, A. Fujikawa; Tokyo/JP
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Learning objectives

To learn the spectrum of CT and MR findings of systemic lupus erythematosus involving the brain (CNS lupus).

To know how to make the differential diagnosis of lesions of CNS lupus based on lesion distribution as an initial step.

Background

Systemic lupus erythematosus (SLE) is one of most frequent chronic autoimmune connective tissue diseases involving multiple organs. As one of target organs, it often affects the brain resulting in CNS lupus. Clinically CNS lupus presents psychological symptoms, convulsive attacks, and cerebrovascular events. It also reveals meningitis and cranial nerve symptoms. As CNS lupus is the most severe disease form of SLE, it greatly affects patients’ prognosis.

Main pathologic feature of CNS lupus is vasculitis. Due to immunocompromised status caused by itself as well as by therapy using immunosuppressive agents, patients with CNS lupus also present with secondary changes. As CNS lupus presents with a wide variety of CT and MR findings, radiologists should be familiar with them and be able to guide physicians to provide proper treatment.

First, this exhibit reviews lesions of CNS lupus that include, lesions related to vasculitis, opportunistic infections, and other lesions including those related to treatment. Then, their differential diagnosis based on lesion distribution is discussed. Imaging techniques that are helpful in the differential diagnosis are also reviewed.

Imaging findings OR Procedure details

As stated above, lesions of CNS lupus are categorized as follows.

(1) Lesions related to vasculitis

They usually involve perforating arteries and capillaries resulting in cerebral ischemia or hemorrhage. They are often associated with inflammatory changes and secondary reversible edema.

(2) Opportunistic infections
Intracranial tuberculosis, septic meningitis, and brain abscess are common in CNS lupus.

(3) Other lesions including those related to treatment

Brain atrophy, intracerebral calcification, and thickened meninges are often noted.

Clinically, differential diagnosis grouped by lesion distribution with additional imaging findings as specifically stated below seems to be of practical help.

**Diffusely distributed lesions:**

1) Ischemic lesions due to vasculitis

In this most frequent lesion, small hyperintense lesions on T2WI or FLAIR are found in the white matter like non-specific lesions seen in the aged population (Fig. 1 on page 4) (1). Fresh lesions can be hyperintense on DWI. Such lesions are more frequently noted when a patient is complicated by antiphospholipid antibody syndrome.

2) Meningitis

Aseptic meningitis as a process of inflammation and septic (pyogenic)/tuberculous meningitis as an opportunistic infection often occur. Abnormal leptomeningeal enhancement on postcontrast T1WI or FLAIR is a key to the diagnosis (Fig. 2 on page 5). As well known, tuberculous meningitis frequently involves the basal cisterns.

3) Meningeal enhancement

Widespread thick dural enhancement can be revealed on postcontrast T1WI (Fig. 3 on page 6). This reflects granulomatous changes of the meninges (1, 2).

4) Brain atrophy

Diffuse cerebral atrophy secondary to steroid administration is a frequent finding (Fig. 4 on page 7) As this is a reversible finding in most cases, it is also called "brain shrinkage".

**Focal lesions:**

1) Arterial infarction

Infarction due to main arterial trunk involvement may be seen (Fig. 5 on page 8) Corresponding arterial lesions can be revealed on 3D time-of-flight MRA (Fig. 6 on page 9).

2) Focal inflammatory lesion
Inflammation due to vasculitis shows similar findings to those of purely ischemic lesions. They may be reversible and show transient contrast enhancement (Fig. 7 on page 10). Additionally, they can at least partially be hyperintense on DWI due to restricted diffusion.

3) Lesions of posterior reversible encephalopathy syndrome

It has been recognized that posterior reversible encephalopathy syndrome not infrequently occurs in CNS lupus (Fig. 8 on page 12)(3). Radiological findings do not differ from those seen in cases by other causes.

4) Venous infarct due to sinus thrombosis

Sinus thrombosis and resultant venous infarct often occur in CNS lupus, especially in patients with antiphospholipid antibody syndrome. MR or CT venography should be considered when this is suspected.

5) Intracerebral hemorrhage

Intracerebral hemorrhage, possibly secondary to vasculitis, is not rare in CNS lupus (Fig. 9 on page 11)(4). Such hemorrhage often develops in a region where hypertensive one frequently occurs.

6) Calcification

This frequently occurs in the basal ganglia, centrum semiovale, and cerebellum in a bilateral manner (Fig. 10 on page 13)(5). Although detected clearly on CT, mild calcification is depicted showing hyperintensity on T1WI.

7) Tuberculoma

Intracranial tuberculosis is a frequent finding in patients with SLE (6). Tuberculoma may be solitary or multiple. It can develop both in the brain parenchyma and in the subarachnoid space.

8) Brain abscess

Brain abscess also develops as an opportunistic infection in CNS lupus. Ringlike enhancement and apparent hyperintensity on DWI are hallmarks like in brain abscess of other causes.

Images for this section:
**Fig. 1:** Ischemic lesions in a 49-year-old woman. FLAIR image shows hyperintense lesions in the subcortical white matter.
Fig. 2: Aseptic meningitis in a 34-year-old woman. Postcontrast FLAIR shows intense enhancement within sulci. This finding resolved after treatment with corticosteroid. (Case courtesy of Dr. Masayuki Maeda at Mie University)
**Fig. 3:** Thick dural enhancement in a 43-year-old woman. Postcontrast T1WI shows diffusely thickened dura in the frontotemporal region bilaterally.
Fig. 4: Brain atrophy in an 18-year-old woman. Precontrast CT also shows calcification in the basal ganglia and insular cortex on the left side.
**Fig. 5:** Old MCA area infarct in a 40-year-old woman demonstrated on FLAIR image.
Fig. 6: MCA stenosis in a 22 year-old woman. 3D time-of-flight MRA shows severe left MCA stenosis.
Fig. 7: Reversible lesion in a 36-year-old man. FLAIR image (A) shows a hyperintense lesion in the inferior frontal lobe, which disappears in follow-up study performed two months later (B).
Fig. 8: Intracerebral hemorrhage in a 50-year-old woman. Precontrast CT shows a large hematoma extending from the deep temporal lobe to the basal ganglia.
Fig. 9: Posterior reversible encephalopathy syndrome in CNS lupus in a 38-year-old woman. FLAIR image shows large occipital lobe lesions predominantly on the right side.
Fig. 10: Calcification in the basal ganglia in a 19-year-old woman demonstrated on precontrast CT.
Conclusion

In order to make a correct diagnosis of lesions of CNS lupus, it is essential to know its wide spectrum of CT and MR findings. Lesion distribution is a valuable key to the differential diagnosis of CNS lupus lesions.

Personal Information

Details of authors:

(1) Kazuhiro Tsuchiya, M.D., Masamichi Imai, M.D., Maiko Yoshida, M.D., Keita Honya, M.D., and Toshiaki Nitatori, M.D.

(2) Akira Fujikawa, M.D.

(1) Department of Radiology, Kyorin University Faculty of Medicine, Tokyo, Japan

(2) Department of Radiology, Japan Self-Defense Forces Central Hospital, Tokyo, Japan

References