



## Low dose cytarabine-induced posterior reversible encephalopathy syndrome with atypical features

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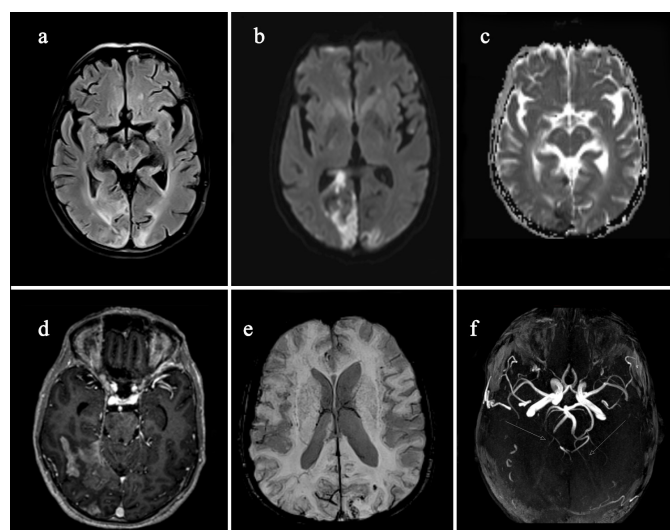


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A 70-year-old man presented with fatigue and weight loss and was diagnosed with AML-M5b after pancytopenia was detected. Low-dose cytarabine treatment (20 mg twice daily, subcutaneously) was initiated. Five days after completing therapy, he developed blurred vision. Apart from bilateral visual impairment on ophthalmologic evaluation, his vital signs, physical examination, serum metabolic parameters, ECG, and echocardiography were normal.

Contrast-enhanced brain MRI revealed bilateral, symmetrical T2-FLAIR hyperintensities in the parieto-occipital watershed regions with gyral and leptomeningeal enhancement. These areas showed both restricted diffusion and T2 shine through on DWI/ADC, indicating mixed cytotoxic and vasogenic edema, along with small hemorrhagic foci (Figure 1). Non-contrast MRA demonstrated non-visualization of the distal P2 segments of both posterior cerebral arteries, likely secondary to vasospasm. After excluding other etiologies, cytarabine-associated posterior reversible encephalopathy syndrome (PRES) was diagnosed.

PRES results from impaired cerebrovascular autoregulation and is commonly triggered by acute hypertension, cytotoxic agents, or renal dysfunction, typically presenting with symmetric vasogenic edema in the parieto-occipital regions of brain [1]. In this case, the mixed diffusion pattern, presence of hemorrhagic products, and contrast enhancement were atypical for classical PRES and are also not expected in the hyperacute phase of arterial ischemia [2]. While PRES associated with intermediate- and high-dose cytarabine ( $2 \times 3 \text{ g/m}^2/\text{day}$ ) has been reported, no cases linked to low-dose cytarabine have been described to date. Even low-dose cytarabine may lead to



**Figure 1.** (a) Bilateral parieto-occipital cortical and subcortical hyperintensities on FLAIR; (b, c) DWI and corresponding ADC map show restricted diffusion together with areas of T2 shine-through in these regions; (d) Contrast-enhanced T1-weighted image demonstrates a gyral enhancement pattern in the right temporo-occipital region; (e) SWI reveals a few punctate blooming artifacts representing small hemorrhagic foci in the left parietal cortex; (f) Non-contrast MRA demonstrates absence of the distal P2 segments of both posterior cerebral arteries.

PRES; therefore, both classical and atypical imaging features should be recognized.

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