

[PICTURES IN CLINICAL MEDICINE]

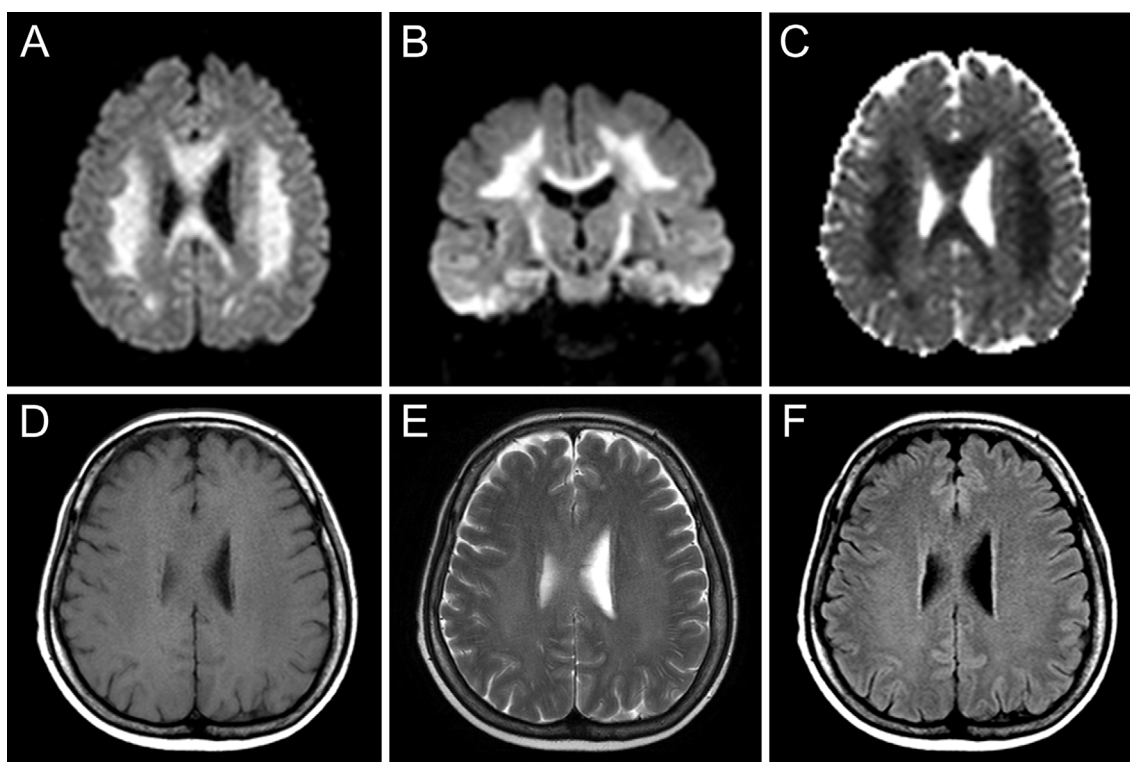
Capecitabine-induced Leukoencephalopathy

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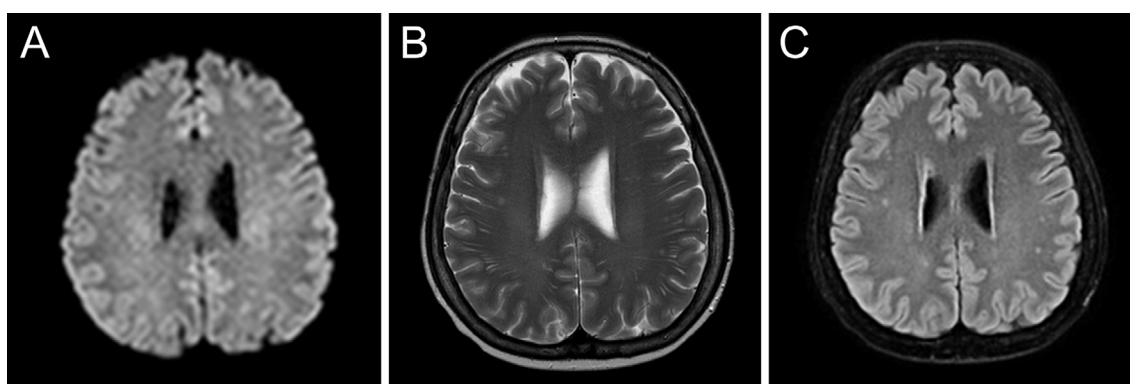
Key words: capecitabine, 5-fluorouracil, leukoencephalopathy, toxic leukoencephalopathy, capecitabine-induced leukoencephalopathy

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Picture 1.



Picture 2.

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A 51-year-old woman presented with acute-onset agitation and disturbance of consciousness. She had metastatic breast cancer and had been treated with capecitabine for one week prior to presentation. Brain magnetic resonance imaging (MRI) with diffusion-weighted imaging (DWI) revealed extensive leukoencephalopathy spreading along the bilateral pyramidal tracts and the corpus callosum [axial (Picture 1A) and coronal (Picture 1B)], which showed a low intensity on an apparent diffusion coefficient map (Picture 1C), although T1-weighted (Picture 1D), T2-weighted (Picture 1E) and fluid-attenuated inversion recovery (FLAIR) (Picture 1F) imaging showed only subtle changes. Her symptoms disappeared completely at one week after admission with the discontinuation of capecitabine, and brain MRI taken at that point showed no abnormalities on DWI (Picture 2A), T2-weighted imaging (Picture 2B) or FLAIR imaging (Picture 2C). Capecitabine is an oral prodrug of 5-fluorouracil and rarely causes leukoencephalopathy (1, 2). Broad symmetric restricted diffusion with relatively subtle T2/FLAIR changes is a characteristic finding (1), suggesting that cape-

citabine might induce cytotoxic edema by abnormal water shift from the extracellular to intracellular space. Since the discontinuation of capecitabine leads to a good clinical outcome by improving cytotoxic edema, clinicians should be aware of these imaging features to facilitate the early treatment of such patients.

The authors state that they have no Conflict of Interest (COI).

References

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