



Posterior Reversible Encephalopathy Syndrome (PRES) due to reversible cerebral vasoconstriction syndrome after Rituximab treatment: a case report

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BACKGROUND - We present a case of PRES in a patient treated with Rituximab because of a necrotizing extracapillary glomerulonephritis. PRES is a rare adverse event in patients treated with RTX, and it is more frequent if RTX is associated to other risk factors such as hypertension and dialysis.

PRES is characterized by areas of vasogenic oedema caused by endothelial dysfunction which especially involves the posterior cerebrum because of the poor sympathetic innervation of the vasculature, making it more susceptible to systemic blood pressure oscillations. Mechanisms involved in this syndrome are still not well known.

THE CASE - A 51-year-old woman, family history of arterial hypertension and diabetes, in good health until December 2014, suddenly presented vomit, fever, gross haematuria, renal failure. In our Nephrology department she underwent haemodialysis and steroid therapy. Renal biopsy showed a necrotizing extracapillary glomerulonephritis. So in February 2015 she started therapy with Rituximab (500mgs fortnightly for 4 times). Few days after the first infusion, her arterial blood pressure became constantly high. After a dialysis treatment she became confused, manifested expressive aphasia and left hemiparesis.

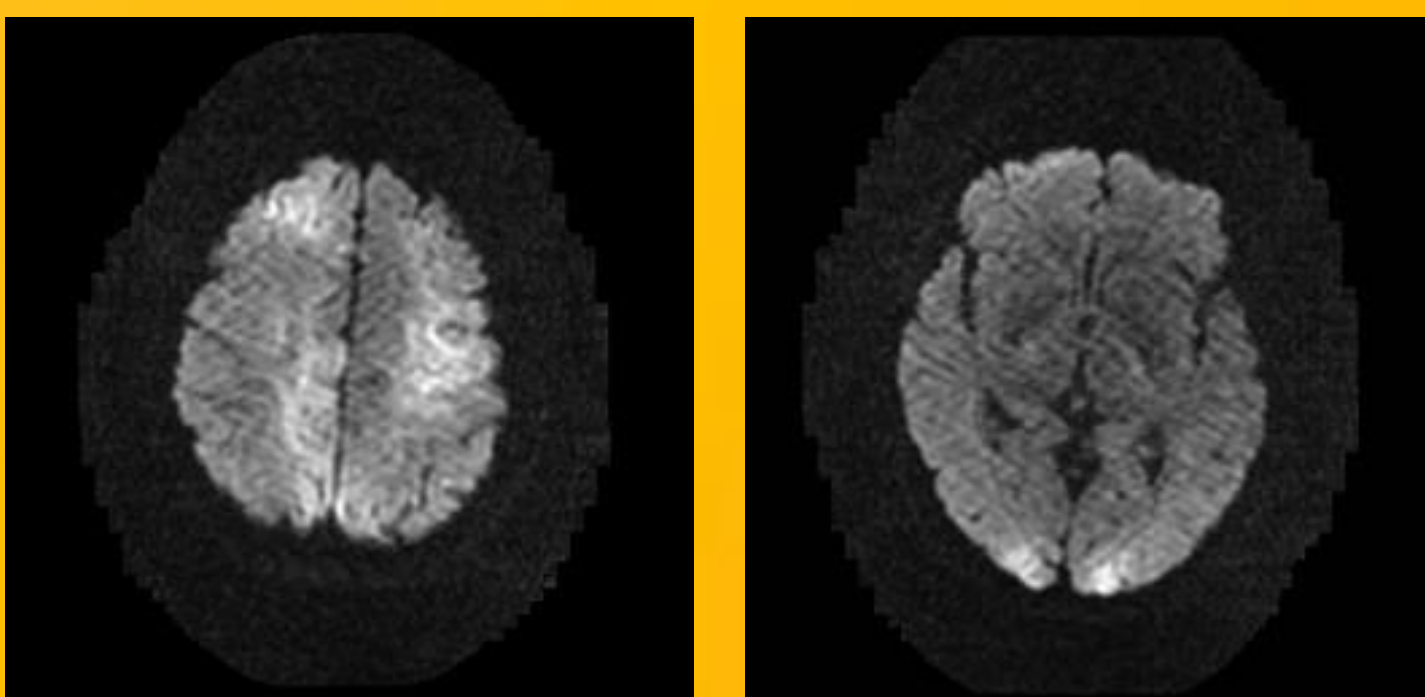
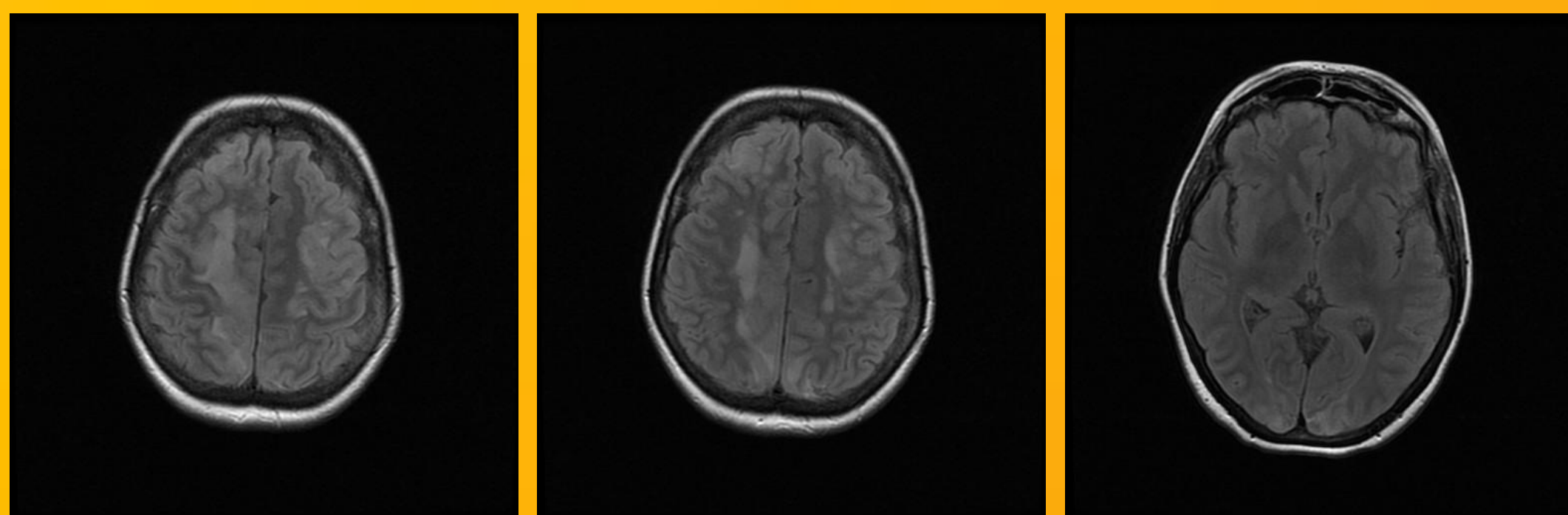


figure 1

She immediately underwent brain MRI showing a mild T2 hyperintensity in right centrum semiovale. The same day she developed incoming generalized tonic-clonic seizures and was transferred to ICU after orotracheal intubation. Antihypertensive therapy, dexamethasone, phenitoin, diazepam and levetiracetam were administered.

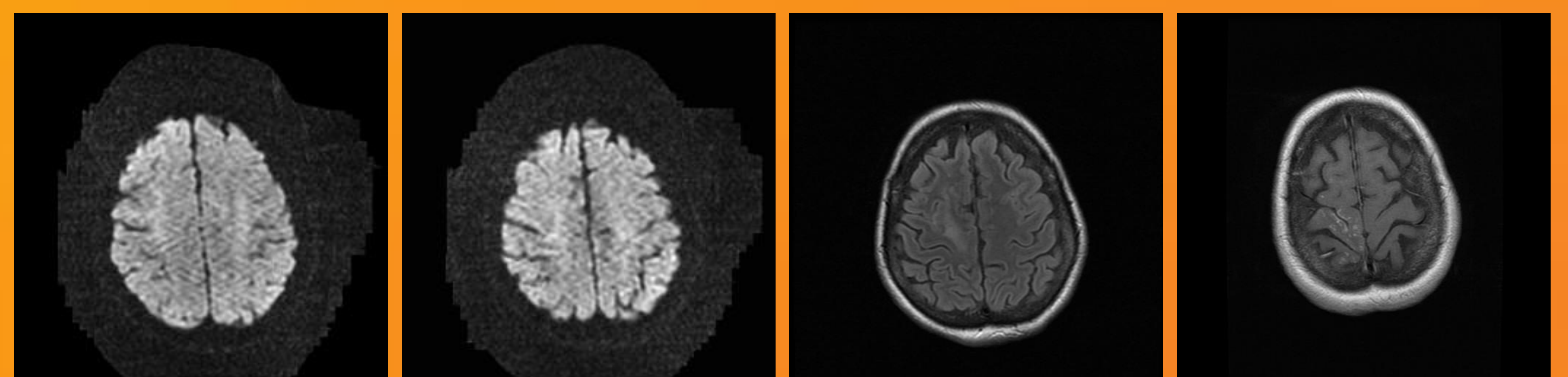


Two days after, a new MRI (*figure 1*) showed bilateral and partially symmetrical T2 and FLAIR hyperintense areas of brain oedema in frontal and occipital cortex and subcortical white matter.

After superficialization of the medically induced coma, neurological conditions appeared improved: the patient was able to execute orders and move the right limbs.

An ultrasound assessment of the intracranial arteries confirmed marked vasoconstriction. Therapy with Nimodipine (360 mgs/day) was begun.

A brain MRI, carried out a couple of weeks after, showed an almost complete recovery of the brain lesions, except for a small FLAIR hyperintense area in white matter of the right parietal lobe.



Ultrasound assessment also showed a clear improvement.

The patient was alert, cooperating, without language impairment and showed a mild paresis of the left leg (more evident in the distal sites).

She was still on dialysis; Nimodipine and anti-epileptic drugs were step-wise reduced in three months.

CONCLUSIONS - Reversible cerebral vasoconstriction syndrome must be taken into account as a possible mechanism of PRES after RTX treatment. Because it is a potentially treatable condition it must be recognized and searched by imaging and sonology. Vasodilatation therapy can improve prognosis and must be maintained since spasm resolve.

REFERENCES

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