A 53-year-old woman presented in September 2004 with a stage IV adenocarcinoma in the upper lobe of the left lung with pleuropulmonary metastasis. She was treated with cisplatin (70 mg/m² on day 1 and gemcitabine 1250 mg/m² on days 1 and 8) by intravenous administration every 3 weeks. After five treatment cycles, the administration of cisplatin was stopped because of a low clearance creatinin, 60 ml/min. The cumulative dose of cisplatin at that point was 350 mg/m². The sixth course included carboplatin area under the curve 4 mg/ml min (administered dose 260 mg) in combination with the same doses of gemcitabine. At day 7, the woman experienced headache, abdominal pain, fever, sudden cortical blindness and generalized seizure requiring intensive care. Biological investigations showed grade 4 neutropenia (0.424 $\times$ 10⁹/l), moderate thrombocytopenia (106 $\times$ 10⁹/l), borderline hypomagnesemia (0.7 mmol/l) and hypophosphataemia (0.75 mmol/l). A computed tomographic brain scan revealed bilaterally occipital white matter edema, not really consistent with metastasis. A lumbar puncture gave normal results and no organism cultured. T2 (Figure 1) and fluid attenuation inversion recovery (FLAIR) (Figure 2) magnetic resonance sequences showed cortical and subcortical hyperintense signals in occipital and frontoparietal lobes which were of low-intensity areas in T1 sequences. All were consistent with a reversible posterior leukoencephalopathy syndrome (RPLS). Over a few days, her condition gradually improved with supportive therapy alone, corticosteroids and antiepileptic treatment. She returned to normal with complete neurological recovery. Repeat Magnetic Resonance scan 4 weeks later showed complete resolution of the previous abnormalities.

Central nervous system (CNS) toxicity has emerged as a potential complication of cisplatin therapy, and the first report about RPLS was published in 1980 [1]. RPLS is a clinicoradiological entity, characterized by subacute onset of headache, altered conscious state and visual disturbance ranging from blurred vision to total cortical blindness; seizures
are common [2, 3]. Characteristic radiological changes consist of bilateral cortical and subcortical edema, predominantly with a posterior distribution resulting in hyperintense signal on T2 MR scanning; better visualized on FLAIR sequences. This toxicity usually occurs immediately at the end or shortly after the end of the cisplatin administration, often when the total dose is ≥200 mg/m² [4, 5]. Fever, neutropenia, thrombocytopenia, hypomagnesemia, renal dysfunction, hypokalemia and hyponatremia are considered by some authors to contribute to the CNS toxicity of cisplatin [6, 7]. With appropriate management, RPLS is reversible in the majority of the cases. This management is on the basis of the supportive measures, antiepileptic drugs, correction of possible fluid and electrolytes imbalances [6]. The administration of steroids might speed up recovery [1, 5, 8]. This toxicity may not preclude further treatment with cisplatin.

Our observation is of particular interest because this encephalopathy occurs after carboplatin infusion. Yet, this agent has not been related to either focal or diffuse encephalopathy in the literature. In conclusion, RPLS is a rare complication of intravenous cisplatin therapy. The question whether RPLS was specifically related to carboplatin or to the cumulative dose of platinum cannot be solved.

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references

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