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Comment on “Posterior reversible encephalopathy syndrome in a patient with metastatic breast cancer: A case report”

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Abstract

Posterior reversible encephalopathy syndrome (PRES) is a neurotoxic encephalopathic state, manifesting clinical symptoms of headache, altered consciousness, visual disturbances, and seizures. Although several diseases have been identified as causative of PRES, the underlying mechanism remains unclear. Song *et al* recently published “Posterior reversible encephalopathy syndrome (PRES) in a patient with metastatic breast cancer: A case report” in the *World Journal of Clinical Cases*, highlighting and discussing the role of hypercalcemia in PRES as related to uncontrolled hypertension. To build upon this case description, we provide further insight into the possible underlying mechanisms of PRES through this commentary.

Key Words: Case report; Hypercalcemia; Paraneoplastic syndrome; Posterior reversible encephalopathy syndrome

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Core Tip: We speculate that posterior reversible encephalopathy syndrome may be caused by paraneoplastic antibodies, tumors or even antitumor therapy, per se, in addition to the hypercalcemia postulated by Song *et al* in “Posterior reversible encephalopathy syndrome (PRES) in a patient with metastatic breast cancer: A case report” (*World Journal of Clinical Cases*, March 2022).

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TO THE EDITOR

We read the article “Posterior reversible encephalopathy syndrome (PRES) in a patient with metastatic breast cancer: A case report”, written by colleagues Song *et al*[1] and published in the March online issue of *World Journal of Clinical Cases*, with great interest. We now welcome the opportunity to make a short comment, as this very interesting article assumes the role of hypercalcemia in PRES by causing uncontrolled hypertension.

With regard to the 51-year-old woman with PRES-related hypercalcemia described in the Case Report, we agree with authors’ insight that there are various possible causes of the neurological symptom of high-level serum calcium. In a similar Case Report by Mirian *et al*[2], a 74-year-old woman with PRES is described with elevations in serum calcium associated with this syndrome; her imaging abnormalities completely resolved after the serum calcium returned to normal. There is also the case of a 38-year-old woman with breast tumor reported by Camara-Lemarroy *et al*[3]; for this patient, the treating physicians considered the physiopathological mechanisms of malignant hypercalcemia (14.5 mg/dL) that can lead to neurological symptoms corresponding to PRES. Ultimately, all 3 cases support the role of hypercalcemia in PRES.

However, Barber *et al*[4] have clinical experience with PRES presenting in a 58-year-old woman with ovarian cancer in the presence of paraneoplastic antibodies (namely, antibodies to collapsin response-mediator protein-5), detected in cerebrospinal fluid. This association may point to another cause for this condition[4]. There is also a patient case of delayed gemcitabine-induced PRES described in the literature by Schaub and Tang[5].

It is a well-known fact that malignant tumors can cause activation of endothelial cells, proliferation and neovascularization, all which lead to vascular cerebral dysregulation. We speculate, thusly, that PRES may be caused by paraneoplastic antibodies, tumors or even antitumor therapy, *per se*, in addition to the hypercalcemia postulated by Song *et al*[1]. In this regard, we suggest that clinicians addressing similar cases in the future should include screening for paraneoplastic syndrome and parathyroid hormone changes in their clinical investigations. Since treatment success and overall prognosis are related to the underlying etiology, solidifying our knowledge of such on a case-by-case basis will benefit this patient population overall.

FOOTNOTES

Author contributions: Kunić S and Ibrahimagić OĆ designed the commentary; Kunić S, Kojić B and Džananović Dž performed the data analysis and wrote the letter; and Ibrahimagić OĆ revised the letter for important intellectual content

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