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Letter to the Editor

Thymopentin is More Likely to Prevent Myasthenia Gravis than to Cause it

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Letter to the Editor

We read with interest the article by Zhao *et al*. about a 75-year-old woman with triple-negative breast cancer who was treated with mastectomy, doxorubicin, cyclophosphamide, and paclitaxel and developed myasthenia gravis (MG) four years after completing chemotherapy and four years after receiving thymopentin [1]. The diagnosis of MG was made based on a positive neostigmine test, abnormal low-frequency repetitive nerve stimulation, elevated antibodies against the acetylcholine receptor, and improvement of MG symptoms after intravenous administration of immunoglobulins, prednisone, and pyridostigmine [1]. The study is interesting, but some uncertainties still need to be clarified.

The first point is that the causal relationship between thymopentin and MG is questionable [1]. A temporal relationship does not necessarily imply a causal relationship. Arguments against thymopentin as a trigger of MG are that breast cancer itself can be complicated by MG as a paraneoplastic phenomenon [2], that chemotherapy for breast cancer may have triggered MG [3], and that MG may have developed independently of the malignancy and its treatment, possibly in connection with the patient's psychopathological condition.

The second point is that thymopentin has been shown to be beneficial for patients with MG after thymectomy and does not trigger MG [4]. In a study of 72 pediatric and adult patients with MG who were treated with thymectomy, prednisone, pyridostigmine, and thymopentin for three months, remission rates were higher at all time points than in the control group that received only prednisone and pyridostigmine [4]. In pediatric patients, the withdrawal rate was higher and relapse rates were significantly lower compared to the control group [4]. The authors concluded that thymopentin effectively reduces MG relapses and has a higher drug withdrawal rate, especially in children [4].

The third point is that it is not clear why thymopentin was only discontinued one year after the onset of MG, even though it was suspected to be the cause [1]. What was the reason for the patient continuing to take thymopentin even though it was considered to be responsible for the MG?

The fourth point is that the outcome of the breast cancer treatment was not reported [1]. Since triple-negative breast cancer is particularly aggressive, it is conceivable that the mastectomy and adjuvant chemotherapy were not particularly effective or even ineffective. Did the breast cancer recur or metastasize during the eight-year follow-up period?

The fifth point is that there was no mention of whether the patient had a thymoma and whether she underwent a thymectomy in addition to immunosuppressive treatment for MG.

Overall, the causality between the administration of thymopentin for breast cancer and MG remains only a temporal association, and several arguments speak against a causal relationship. More extensive research is needed before a final assessment can be made as to whether thymopentin actually triggers MG.

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