

Pancytopenia secondary to hypopituitarism may just be due to hypothyroidism alone

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Dear Editor,

I read with interest the case series and review of Sheehan's syndrome and pancytopenia by Laway et al. [1]. The authors suggest that thyroid and cortisol hormones are ultimately responsible for the marrow hypoplasia. As multiple hormone deficiencies in Sheehan's syndrome are often replaced simultaneously, it is difficult to discern if the hematological abnormalities are related to any one or all of the deficient hormones. My experience of an 11-year-old girl who developed post-ablative hypothyroidism and prolonged pancytopenia after radiotherapy for suprasellar germinoma while receiving cortisol replacement suggests that thyroid hormone alone may be responsible for the marrow hypoplasia.

An 11-year-old girl was receiving chemotherapy for a suprasellar germinoma which was complicated by growth hormone deficiency, hypoadrenalism, and central diabetes insipidus. Hydrocortisone and desmopressin were replaced while she was receiving carboplatin at 500 mg/m² on day 1 and etoposide at 120 mg/m² on days 1 to 3 at 3-week cycles. Pegfilgrastim (0.1 mg/kg, subcutaneously) was used following each cycle of chemotherapy. The first two cycles of chemotherapy were completed without much adverse effects. The lowest hemoglobin, absolute neutrophil count, and platelet recorded were 8.2 g/dL, 0.63 × 10⁹/L, and 129 × 10⁹/L, respectively. Transfusion with packed cells or platelet was not required. Thyroxine treatment was not needed as the serum levels of thyrotropin and free T4 were normal.

She then underwent cranial radiotherapy during which chemotherapy was stopped for 2 months. The third cycle of chemotherapy was then continued. Despite the use of pegfilgrastim and additional doses of filgrastim, she lapsed into prolonged pancytopenia that lasted for 4 weeks. The trough Hb, absolute neutrophil count, and platelet were 6.9 g/dL, 0 × 10⁹/L, and 8 × 10⁹/L, respectively. Two transfusions with packed cells and two transfusions with platelet were required. Serum thyrotropin was at 1.7 mIU/L (normal, 0.35–5.00) and free T4 was at 9.32 pmol/L (normal, 11.35–21.03). Thyroxine replacement was therefore added to the combination of hormonal replacement therapy.

The pancytopenia resolved promptly within a week and she received the fourth cycle of chemotherapy without much adverse effects similar to the first two cycles of treatment. The hematology remained stable and normal during the next 12 months of follow-up before growth hormone treatment was commenced.

The case reported is therefore suggestive of hypothyroidism as the major contributing factor in the pathogenesis of pancytopenia in patients with panhypopituitarism. Pancytopenia and marrow hypoplasia has also been reported previously in a 68-year-old woman who presented with myxoedema coma [2]. Again, pancytopenia resolved after thyroid hormone replacement. Thus, hypoplastic anemia complicating hypopituitarism may just be due to hypothyroidism alone.

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