

## EXCESSIVE TEA CONSUMPTION CAN INHIBIT THE EFFICACY OF ORAL IRON TREATMENT IN IRON-DEFICIENCY ANEMIA

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### ABSTRACT

Intestinal absorption of non-heme food iron may be inhibited by tea, which, on the contrary, does not exert any appreciable effect on heme iron assimilation. Therefore, while an iron-deficiency anemia cannot develop in non-vegetarian subjects as a consequence of tea consumption only, it is possible that tea could inhibit the therapeutic effect of oral iron drugs, which are usually non-hemic ferrous salts, in iron-deficient subjects. This view is supported by the case we describe here, a young woman affected by hypermenorrhea and iron-deficiency anemia, who did not respond to oral iron treatment until she stopped her long-established habit of consuming large quantities of tea. We also believe that oral iron drugs should never be taken together with a cup of tea; therefore we think it useful to advise our iron-deficient patients clearly not to combine tea with the oral assumption of non-hemic ferrous salts.

*Key words: iron-deficiency anemia, tea, oral iron treatment*

It is well known that intestinal iron absorption may be affected by the composition of a meal, since some substances have a facilitating role and others an inhibiting one.

The effect of tea on iron absorption has been investigated in various studies on human volunteers, and the results have shown that tea inhibits the uptake of non-heme iron, with no appreciable effect on heme iron.<sup>1-3</sup>

If tea consumption affects the absorption of only one of the two pools of alimentary iron, it is not surprising that an iron-deficiency anemia due to tea is an unlikely event, at least in non strictly vegetarian subjects. As a matter of fact, a literature review we recently performed with the Medline Data Bank yielded only one case report of iron-deficiency anemia attributed to excessive tea consumption during the last twenty years.<sup>4</sup>

Therefore we describe herein a similar case recently observed of a young woman whose long-established iron-deficiency anemia was corrected only after her tea consumption had stopped.

### Case report

In August 1989, a 25-year-old woman had her first laboratory analyses as a result of an acute gastroenteritis which had spontaneously resolved within a few days. Those tests unexpectedly documented a microcytic anemia for the first time: hemoglobin (Hb) 9.0 g/dL, red blood cells (RBC)  $4.85 \times 10^{12}/L$ , hematocrit (Ht) 30.3%, mean corpuscular volume (MCV) 62.4 fL; leukocyte and platelet counts were in the normal range.

Three weeks later the erythrocytic parameters were unchanged, serum iron and ferritin levels were low (10  $\mu\text{g}/\text{dL}$  and 3.8 ng/mL, respectively) and total iron binding capacity was higher than normal (432  $\mu\text{g}/\text{dL}$ ); serum bilirubin, reticulocyte count, erythrocyte osmotic fragility and Hb A2 fraction were all normal. An iron-deficiency anemia was therefore diagnosed.

The patient only complained of a mild effort intolerance, which did not limit her active life. Nothing significant was found on physical examination and constitutional parameters

were within the normal range (height 160 cm; weight 59 kg; body mass index 23). Her past medical history was negative; she had never been pregnant and her menstrual flow had always been abundant. Her diet seemed to be well balanced. Ultrasonography of the abdomen and endoscopy of the upper and lower intestinal tract were all normal.

In September 1989, the patient started oral iron treatment (Ferro Grad, one tablet/day at breakfast; ferrous sulfate 525 mg/day) and a gradual improvement of the anemia was observed, until complete normalization of the erythrocytic values (Hb 12.8 g/dL; Ht 38.4%; RBC  $4.04 \times 10^{12}/L$ ; MCV 94.8 fL) was achieved in August 1990 (Figure 1). Iron therapy was therefore stopped but hemoglobin levels dropped again within the following three months (Hb 10.8 g/dL in December 1990), so that iron intake was resumed and maintained until a hemoglobin level of 13 g/dL was reached in August 1991.

In December 1991, hemoglobin concentration was again low and oral iron treatment was then resumed. However, the anemia worsened despite continuous iron intake, decreasing to a hemoglobin level of 9.9 g/dL in July 1993. At this time cyclic estroprogestinic treatment

(Triminulet) was added to reduce menstrual blood losses, but no positive effect was observed on the anemia and the Hb level fell further to 8.5 g/dL in the following months.

In July 1994, the patient presented a hemoglobin concentration of 9.1 g/dL, and was seen for the first time in our Institute, where her medical history was integrated with a careful review of dietary habits. An abundant consumption of tea was then identified, mostly in the last three years, when her daily intake of tea had risen to 1.5 liters and had replaced all other beverages such as mineral water, coffee, milk, wine and beer. We also verified that as a rule the patient took her oral iron supplement with a large cup of tea at breakfast. The patient was strongly advised to abstain completely from drinking tea. This was the only change in her prescription in order to verify the possible obstacle to adequate intestinal iron absorption; iron and estroprogestinic treatments were maintained unchanged.

As the Figure shows, the hemoglobin level completely normalized after the first five months and has remained in the normal range up to the most recent test, a year later (Hb 12.9 g/dL in June 1995). Also serum iron and ferritin rose to normal values (106  $\mu\text{g}/\text{dL}$  and 52 ng/mL, respectively) already in January 1995.

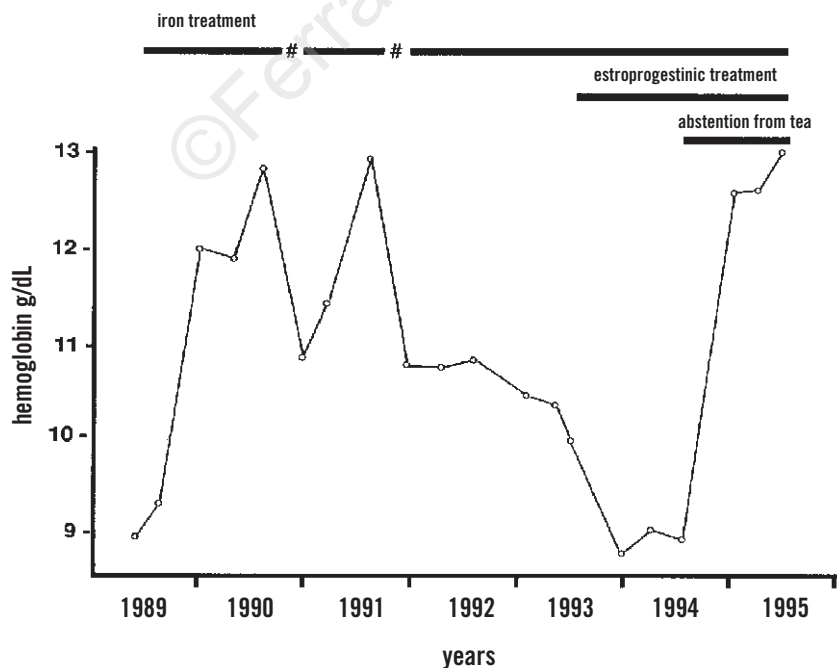


Figure 1. Changes in the patient's hemoglobin level over time (from August 1989 to June 1995) in relation to iron treatment, estroprogestinic treatment and abstinence from tea.

### Discussion

The anemia fortuitously found in our patient in 1989 was an iron-deficiency anemia probably related to chronic hypermenorrhea, since no other common cause of iron depletion in women was detected.

Treatment with an oral iron compound promptly corrected the anemia for the first two years (from August 1989 to August 1991); however, the anemia relapsed soon after the drug was discontinued (Figure 1).

In December 1991 oral iron treatment was resumed and continued indefinitely, but it was no longer effective in correcting the anemia. Estroprogestinic treatment was then added in July 1993, in order to reduce menstrual blood losses, but nevertheless the anemia clearly worsened during the following year (Figure 1).

In July 1994, we first hypothesized a possible etiologic role for tea, the beverage which the patient had begun drinking almost exclusively, replacing mineral water, milk, coffee, wine and beer. Moreover, she had increased her daily tea intake to 1.5 liters over the last three years, in parallel with an apparent decrease in the efficacy of the iron therapy to control the anemia.

The Figure shows clearly that complete abstention from drinking tea, which started at the end of July 1994, was followed by a rapid improvement of the anemia, that continued to normalization of the hemoglobin level, in the absence of any other change in diet or drug treatment.

It is therefore evident that iron absorption was inhibited by tea in our patient, as well as in a patient of Simon *et al.*,<sup>4</sup> who also described the complete recovery from an iron-deficiency anemia in a woman who habitually drank one to two liters of tea daily a few months after tea had been removed from her diet.

Drinking tea inhibits the absorption of non-heme iron only (i.e. iron from non-animal sources), probably through the formation of insoluble iron-tannate complexes;<sup>1</sup> however, heme iron intake (from animal food) is not affected by tea.<sup>2</sup> For these reasons the consumption of tea could not by itself induce an iron deficiency, at least in non vegetarian subjects, like our patient. It is possible, however, that large amounts of tea could inhibit the therapeutic effect of oral iron compounds, which often consist of non-heme ferrous salts, as in our case. This inhibiting effect on oral iron therapy is greater if tea and iron are taken at the same time, as our patient did during the last several years. We believe therefore that oral iron drugs and tea should not be ingested at the same time, since the therapeutic effect of the iron would be greatly inhibited by such an association.

In conclusion, it must be stressed that tea intake should be considered when examining cases of iron-deficiency anemia resistant to iron therapy.<sup>5</sup> Abstention from tea must be prescribed in these patients before the conclusion can be drawn that their intestinal absorption of iron is insufficient to correct the iron-deficiency anemia, making parenteral iron necessary.

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### References

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