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Does hair loss hang over iron deficiency?

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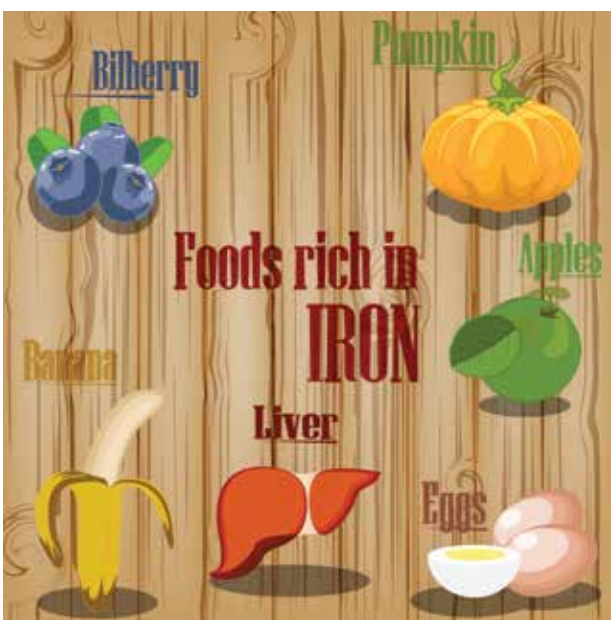
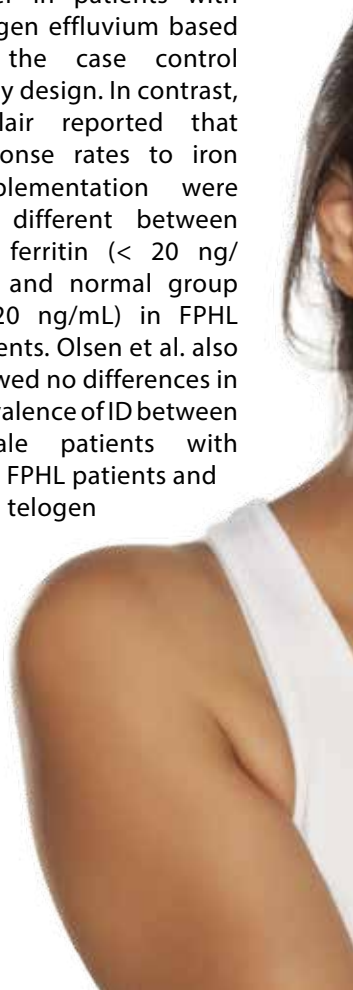
Loss of scalp hair is not a serious life-threatening disorder, but it can cause psychological distress and adversely affect quality of life. Hairs are rapidly proliferating organ with much requirement of blood supply. Therefore, the relationship between micronutrients and hair loss has been evaluated in several studies since the 1960s. The most widely cited nutritional causes of hair loss include iron, one of the key micronutrients in metabolism of our body. It is known that iron deficiency (ID) is associated with many pathological conditions. However, its role in hair loss is not well established yet. While studies show relationship between ID and hair loss, including female pattern hair loss (FPHL), telogen effluvium, alopecia areata, alopecia universalis or totalis, some advocated the association and others opposed it. The controversy might be caused by study designs, methodology and clinical condition defining hair loss. For detection of ID, serum ferritin level can be used as a very early marker. It is a main iron-binding protein in nonerythroid cells reflecting total body iron stores. It decreases from very early stage of ID as iron reserves go down. Because only ID can cause very low serum ferritin concentration (FC), a FC is very specific for ID.



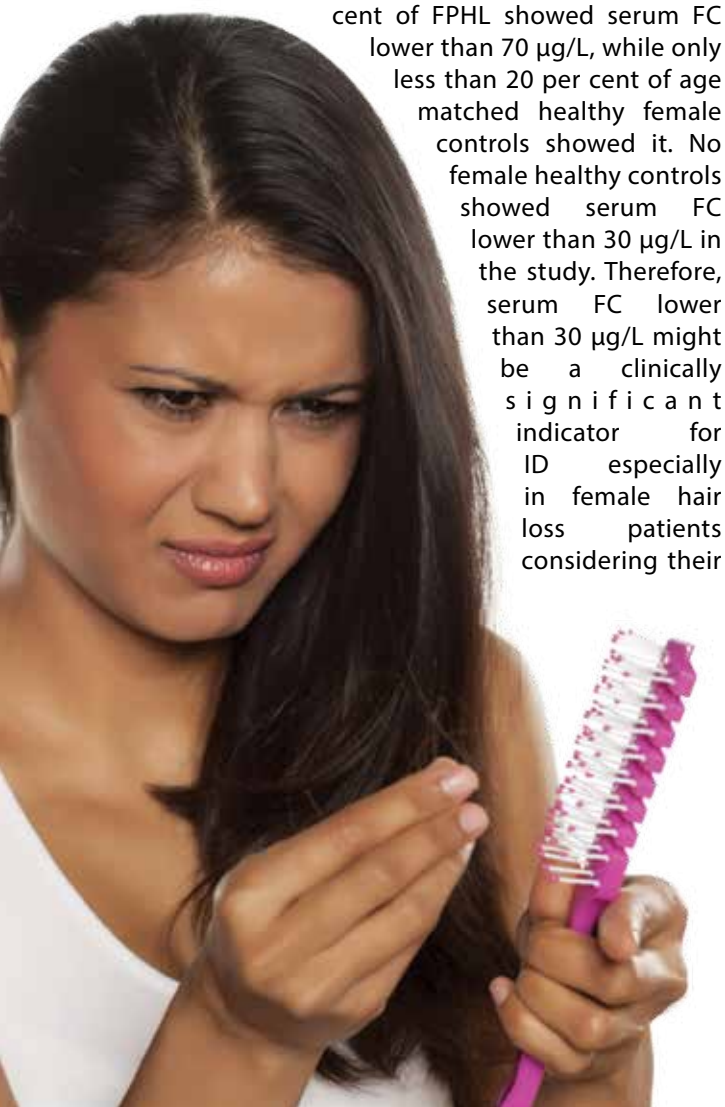
The association between ID and hair loss is debatable. Kantor et al. reported that alopecia areata, FPHL and telogen effluvium patients under 40 years old showed lower serum FC than controls without hair loss. Rushton et al. also demonstrated that there was significant decrease of hair loss and increase of FC in patients with telogen effluvium who received oral iron therapy. These results are supported by Moeinvaziri et al. who suggested that serum FC and transferrin saturation is lower in patients with telogen effluvium based on the case control study design. In contrast, Sinclair reported that response rates to iron supplementation were not different between low ferritin (< 20 ng/mL) and normal group (≥ 20 ng/mL) in FPHL patients. Olsen et al. also showed no differences in prevalence of ID between female patients with (285 FPHL patients and 96 telogen effluvium patients) or without hair loss (a total of 76 Caucasian women older than 18 year old). Rushton et al. criticised study of Olsen et al. in that it appears to have some confounding issues and contradictions such as no-standardised evaluation in



blood sampling and no quantitative hair evaluation in control group.

We conducted a study to find out the relationship between body iron status and various conditions with hair loss. We selected patients diagnosed with FPHL and MPHL. For better comparison, age-sex matched healthy controls without complaint of hair loss were selected. Even as body iron stores can be assessed by serum FC, there is no consensus which ferritin level is the right one to define ID in practice.

Although many laboratories use FC of 10 to 15 µg/L as the lower limits of normal based on reference sample groups, this only gives a sensitivity of 59 per cent and a specificity of 99 per cent for diagnosing ID. In women of childbearing age, using a cutoff of 10 to 15 µg/L yields a sensitivity of 75 per cent and specificity of 98 per cent in diagnosis of ID. A cutoff of 30 µg/L yields a sensitivity of 92 per cent and a specificity of 98 per cent, while a cutoff of 41 µg/L yields a sensitivity of 98 per cent and a specificity of 98 per cent. To avoid controversy regarding guideline to define ID, for the study, over 80 per cent of FPHL showed serum FC lower than 70 µg/L, while only less than 20 per cent of age matched healthy female controls showed it. No female healthy controls showed serum FC lower than 30 µg/L in the study. Therefore, serum FC lower than 30 µg/L might be a clinically significant indicator for ID especially in female hair loss patients considering their menstrual status.



In the study, patients with MPHL show relatively early onset age, which means they appear to visit the clinic earlier than those with FPHL. Patients with FPHL show definitely lower serum FC compared with age/sex-matched normal controls. Female patients with FPHL were divided into two groups based on their menstrual status. Premenopausal patients with FPHL demonstrate much lower serum FC than postmenopausal patients. When compared with normal age/sex matched controls, statistically significant low serum FC is observed in FPHL premenopausal patients, while it is not significantly different between FPHL patients and normal controls after menopause. This result implicates that ID plays a certain role in premenopausal female patients with FPHL. However, weak association of ID with FPHL in postmenopausal patients could be addressed from this study.

Patients with MPHL show considerably lower serum FC on the average than age-sex matched healthy controls. We failed to prove the correlation between onset age and serum FC in MPHL patient, either ($r = 0.12$). However, approximately 20 per cent of MPHL show serum FC lower than 70 µg/L and their age matched controls do not show that low serum FC. This result implicates that screening of iron status in even male patients with hair loss might provide clinical benefits. Clinical manifestations of F type of MPHL looks like those of FPHL in female patients. Therefore, we looked into patients with MPHL according to types (M type, F type and others based on BASP classification), which turns out there was no strong relationship between subtype of MPHL and ID. Patients showing low FC level < 70 µg/L had been on oral ferrous sulphate (130 mg of elemental iron/day) and serum level of FC doubled after six months of supplementation.

Clinical response to iron supplementation proves not to be much higher than expected. Especially patients with MPHL patients rated lower PGA and PS when they were on oral iron supplementation. Iron supplementation group did not receive any kind of oral 5 alpha reductase inhibitor, which might cause significantly lower PGA and PS.

This study owns its value because it demonstrates the first direct comparison between hair loss patients and same number of healthy controls matched by age and sex. It strongly supports the previous studies that ID can be a certain factor of developing or worsening FPHL. Its role in MPHL is hard to conclude from the study. NS