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ORIGINAL PAPER

Iron deposits and dietary patterns in familial combined hyperlipidemia and familial hypertriglyceridemia

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Abstract Iron deposits are associated with lipid phenotype in familial hypertriglyceridemias, mainly familial combined hyperlipidemia (FCH) and familial hypertriglyceridemia (FHTG). In turn, diet plays an important role in hypertriglyceridemias although it is not known if dietary patterns are associated with iron concentration in these disorders. The objective was to determine the relationship between diet and iron deposits, measured through serum ferritin concentration, in patients with FCH and FHTG. The study was composed of 140 patients, 107 with FCH and 33 with FHTG. Subjects completed a validated 137-item food frequency questionnaire. Dividing subjects by ferritin tertiles adjusted by sex, there were no significant differences in dietary patterns except in dairy products consumption which was lower in the highest ferritin tertile. Subjects were also divided by triglycerides tertiles adjusted by sex. Those subjects in the highest tertile had lower HDL cholesterol and higher ferritin concentrations. Regarding to dietary parameters, there were significant differences in marine omega three fatty acids and vegetables presenting higher and lower consumption, respectively, those patients in the highest tertile of triglycerides. Moreover, there was not a significant correlation between dietary iron intake and any parameter, both biochemical and dietary, including ferritin concentrations. In conclusion, in patients with primary hypertriglyceridemia, triglycerides are associated with ferritin concentrations but dietary patterns are not related to iron deposits. Our results highly support the concept that the genetic mechanisms driven to hypertriglyceridemia also favor iron overload.

Keywords Primary hypertriglyceridemias · Familial combined hyperlipidemia · Familial hypertriglyceridemia · Dietary patterns · Iron deposits · Ferritin concentration

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Introduction

Hereditary hyperlipidemias (HH) are a heterogeneous group of lipid metabolism disorders that are usually associated with a high cardiovascular risk [9, 12, 21]. Within the HH disorders those which associate hypertriglyceridemia: familial combined hyperlipidemia (FCH) and familial hypertriglyceridemia (FHTG) are the most common. FCH and FHTG are complex genetic diseases with an important interac-

tion with environmental factors especially with overweight, obesity and peripheral insulin resistance [5, 27, 28]. In fact, a lipid-lowering diet is the first line treatment option in FCH and FHTG especially if overweight coexists since the lipid profile of these subjects could be markedly improved with weight loss or/and a low fat diet [17, 20].

Iron metabolism has been involved in peripheral insulin resistance pathogenesis [7, 30]. In presence of overweight and obesity, excess adipose tissue induces peripheral insulin resistance and increases hepatic iron deposits that are associated with an increased production of inflammatory adipokines [1, 15, 29]. In agreement, increased iron deposits, usually measured through serum ferritin concentration, correlate with different metabolic syndrome criteria [4, 13, 25]. Furthermore, phlebotomy in these subjects, which associates a reduction in iron deposits, improves peripheral insulin resistance in metabolic syndrome [26].

We have recently described that iron metabolism is associated with the lipid phenotype in primary hypertriglyceridemia (HTG) with an independently correlation between ferritin and triglycerides concentration [18, 24]. Moreover, a high percentage of subjects with primary HTG showed genetic predisposition to develop hemochromatosis, indicating that genetic factors play also an important role in iron overload in these subjects. However, the exact contribution of genetic and environmental factors to increased iron deposits is not known.

Due to the strong association between diet and lipid phenotype in primary HTG and the concomitant association of primary HTG with iron deposits, our objective was to prove if dietary patterns could influence not only lipid profile but also iron deposits in these subjects with primary HTG.

Subjects and methods

Study subjects

From January 2006 to December 2009, consecutive unrelated patients aged ≥18 years with hypertriglyceridemia attending to our lipid unit were recruited into a protocol for the genetic and metabolic study of hyperlipidemia. This protocol has been approved by the local review boards and all subjects provided written informed consent. The protocol has been already published [18]. In summary, the diagnosis of FCH was based on the presence of primary combined hyperlipidemia with off treatment serum cholesterol and triglycerides over the sex- and age-adjusted 90th percentile for the Spanish population, serum total apolipoprotein B levels ≥120 mg/dL and, at least, one first-degree relative with hyperlipidemia (total cholesterol and/or triglycerides >90th percentile). The diagnosis of FHTG was based on the presence of primary hypertriglyceridemia, with off treatment triglycerides over the sex- and age-adjusted 90th percentile, at least one first-degree relative with triglycerides >90th percentile and serum total apolipoprotein B levels <120 mg/dL. Secondary hyperlipidemia including alcohol consumption >30g/day were ruled out in all subjects. Subjects with C-reactive protein (CRP) concentration >10 mg/L, indicating active inflammatory disease, were excluded.

Clinical and laboratory determinations

Fasting blood for baseline biochemical profiles was drawn after at least 4 weeks without hypolipidemic drug treatment. In patients with prior coronary heart disease, baseline lipid values were obtained from clinical records. Cholesterol and triglycerides were determined by standard enzymatic methods. HDL cholesterol was measured by a precipitation technique. Non-HDL cholesterol was calculated as total cholesterol minus HDL cholesterol. Apolipoprotein (apo) B was determined by using immunoturbidimetry (Unimate 3, Roche, Basel, Switzerland). Serum iron and unsaturated iron-binding capacity was measured by spectrophotometry, ferritin by turbimetric immunoassay (Hitachi 911, Roche) and transferring saturation was calculated.

Dietary assessment

Dietary intake was determined in all subjects by interview with one single registered dietician (R. M-G.). In this interview, a Spanish validated 137-item food frequency questionnaire (FFQ) was used [6]. The questionnaire included the consumption frequency of each of the 137 food by choosing between nine possibilities of frequency (from never or less than once per month to six or more times per day); and the portion size. The total energy and nutrients intakes

were calculated based on previously validated Spanish food composition tables [16, 19].

Body composition assessment

Fat mass and lean mass percentages were assessed by bioelectrical impedance using OMRON BF-500 (Omron Corporation, Kyoto, Japan).

Statistical analyses

Continuous variables were expressed as means (SD; if normal distribution) or as medians [interquartile range] otherwise, whereas categorical variables were reported as percentages. Differences in mean values were assessed using *t* tests or the Mann–Whitney *U* test, as appropriate. Categorical variables were compared using chi-square tests. Spearman correlations between ferritin and triglycerides, and the other variables were performed. The Kruskal–Wallis one-way analysis of variance by ranks was used for testing equality of clinical and biochemical characteristics. All statistical analyses were performed with SPSS software (version 15.0), with significance set at $p<0.05$.

Results

Clinical characteristics

The group studied was composed of 140 patients, 107 diagnosed with FCH and 33 with FHTG. Regarding to sex distribution, males ($n=99$, 70.7%) predominated over females ($n=41$, 29.3%). Table 1 shows the main characteristics of patients divided by clinical diagnosis. Among subjects with FCH and FHTG, there were significant differences in gender, prevalence of coronary heart disease and percentage of fat mass. FCH showed higher concentrations of total cholesterol, non-HDL cholesterol, HDL cholesterol and apolipoprotein A1 and B than FHTG. However, FHTG had higher concentrations of triglycerides.

Dietary patterns

The total energy intake, the distribution of macronutrients, the percentage of different fatty acids in diet, dietary iron and alcohol are also reflected, divided by clinical diagnosis, in Table 1. Males had a higher energy intake, lower protein consumption and a higher non-marine omega three fatty acids intake. Males showed upper alcohol consumption than females. There were no statistically differences in any dietary parameter between FCH and FHTG patients except for a slightly higher percentage of protein intake in FCH subjects.

Association between dietary patterns and iron metabolism

To study the relationship between iron deposits and dietary patterns, we divided subjects by ferritin tertiles adjusted by sex. The different clinical, biochemical and dietary parameters of subjects, divided by ferritin tertiles, are shown in Table 2. There were no significant differences in dietary patterns, total energy intake, distribution of macronutrients and the percentage of different fatty acids in the diet in relation to iron deposits. Only the dairy products consumption was significantly lower in the upper ferritin tertile and remained significant after adjustment for total caloric intake. Alcohol intake did not show differences among three subgroups either.

Association between dietary patterns and triglycerides

As for iron deposits, subjects were divided according to triglycerides tertiles adjusted by gender. These results are shown in Table 3. Those subjects in the highest tertile of triglycerides had, as expected, lower HDL cholesterol and higher ferritin concentrations. Dietary patterns did not show important differences among tertiles except for marine omega three fatty acids which intake was higher in subjects with upper concentrations of triglycerides. There were also significant differences in vegetables consumption with a higher intake in the lowest tertile. Both differences remained significant after adjustment for total caloric intake.

Association between dietary iron consumption and triglycerides concentration and iron deposits

Dietary iron intake, valuated through a FFQ, did not show a significant correlation with any of the biochemical parameters studied, both lipid and not lipid, including iron deposits. The correlation between iron intake and ferritin concentration was −0.037, $p=0.782$ and $r=0.278$, $p=0.223$ for men and women, respectively.

Table 1 Clinical and dietary characteristics of the patients according to clinical diagnosis

	FCH (*N*=107)	FHTG (*N*=33)	*p*
Age (years)	48.5±12.0	46.7±10.9	0.427
Sex (male/female)	67/40	32/1	0.000
Coronary heart disease (*n* (%))	15 (14.2)	0 (0)	0.024
Diabetes (*n* (%))	13 (12.1)	3 (9.1)	0.629
Hypertension (*n* (%))	33 (30.8)	10 (30.3)	0.953
Tobacco consumption			0.681
Non smoker (*n* (%))	39 (37.1)	10 (31.3)	
Current Smoker (*n* (%))	34 (32.4)	13 (40.6)	
Former smoker (*n* (%))	32 (30.5)	10 (31.3)	
Waist circumference (cm)	97.6±10.0	98.1±7.99	0.812
Body mass index (kg/m^2)	28.5±3.35	28.0±3.38	0.481
Fat mass (%)	31.8±7.77	27.3±5.92	0.007
Systolic blood pressure (mm Hg)	136±17.4	141±14.1	0.115
Diastolic blood pressure (mm Hg)	83.3±10.5	85.3±9.37	0.463
Total cholesterol (mg/dL)	394±44.9	233±71.2	0.000
Triglycerides (mg/dL)	254 (204–394)	365 (261–700)	0.000
HDL cholesterol (mg/dL)	41.7±11.4	33.0±9.37	0.000
Non-HDL cholesterol (mg/dL)	253±42.7	204±72.0	0.000
Apolipoprotein A1 (mg/dL)	138±24.2	126±30.3	0.017
Apolipoprotein B (mg/dL)	163±33.7	116±25.1	0.000
Lipoprotein(a) (mg/dL)	17.7 (7.06–50.4)	15.0 (2.69–28.8)	0.079
Glucose (mg/dL)	98.0 (90.0–110)	102 (91.0–111)	0.406
HbA_1c (%)	5.40 (5.10–5.70)	5.40 (5.00–5.50)	0.378
ALT (IU/L)	27.0 (21.0–38.0)	27.0 (23.0–35.0)	0.783
GGT (IU/L)	30.0 (20.0–51.0)	34.0 (24.5–50.0)	0.569
CRP (mg/L)	2.50 (1.10–4.80)	2.10 (1.30–4.10)	0.833
Iron (μg/dL)	94.4±32.4	92.2±32.4	0.738
Ferritin (μg/L)	111 (49.8–172)	143 (94.4–203)	0.057
Transferrin saturation (%)	25.9±10.3	25.1±10.1	0.675
Dietary intake per day			
Energy (kcal)	2,443±762	2,839±807	0.011
Carbohydrates (%)	44.2±7.86	46.9±8.05	0.083
Protein (%)	16.3±2.68	15.2±2.50	0.037
Fat (%)	35.9±7.15	33.6±6.28	0.098
Monoinsaturated fat (%)	17.5±4.49	16.5±3.71	0.238
Polinsaturated fat (%)	5.16±1.99	4.95±2.01	0.585
Saturated fat (%)	9.58±2.12	8.83±1.97	0.075
Trans fat (%)	0.24±0.16	0.22±0.15	0.608
Non-marine *n*-3 fatty acids (g/day)	1.04 (0.88–1.39)	1.05 (0.90–1.59)	0.503
Marine *n*-3 fatty acids (g/day)	0.72 (0.48–1.33)	0.60 (0.44–0.90)	0.229
Dietary iron (mg/day)	14.7±4.09	16.7±4.40	0.100
Alcohol (g/day)	6.97 (1.46–20.6)	10.2 (1.81–24.4)	0.430

Values are mean ± standard deviation or median (interquartile range)

Coronary heart disease was considered in presence of documented history of myocardial infarction, coronary artery bypass graft surgery, percutaneous transluminal coronary angioplasty or angina pectoris with angiographically coronary atherosclerosis (>50% stenosis)

ALT alanine aminotransferase, *GGT* gamma-glutamyl transpeptidase, *CRP* C-reactive protein

Table 2 Clinical and dietary characteristics of the studied subjects according to serum ferritin tertiles adjusted by gender

	Tertile 1 (*N*=47)	Tertile 2 (*N*=46)	Tertile 3 (*N*=47)	p
Age (years)	47.0 (39.3–57.0)	48.0 (39.0–55.3)	53.0 (37.0–58.0)	0.856
Sex (men/women)	33/14	33/13	33/14	0.983
Dyslipidemia (FCH/FHTG)	36/11	36/10	35/12	0.911
Body mass index (kg/m^2)	27.8 (26.4–30.2)	27.8 (25.5–31.0)	28.7 (26.1–29.7)	0.855
Total cholesterol (mg/dL)	274 (245–310)	289 (247–312)	277 (238–325)	0.696
Triglycerides (mg/dL)	266 (209–394)	254 (202–427)	305 (229–500)	0.199
HDL cholesterol (mg/dL)	34.0 (30.0–42.0)	39.0 (33.8–48.5)	40.0 (34.0–47.0)	0.076
Non-HDL cholesterol (mg/dL)	243 (210–280)	245 (211–270)	245 (204–271)	0.933
Glucose (mg/dL)	99.0 (89.0–108)	99.5 (90.8–110)	98.0 (93.0–112)	0.503
GGT (IU/L)	29.0 (18.0–45.0)	28.0 (19.0–38.3)*	41.0 (28.0–69.0)	0.002
CRP (mg/L)	3.50 (1.50–5.95)	1.70 (0.90–3.45)	2.70 (1.15–4.35)	0.049
Iron (μg/dL)	83.0 (71.0–99.5)	86.0 (72.8–102)**	103 (78.0–129)**	0.011
Ferritin (μg/L)	45.9 (23.1–83.8)**	119 (68.1–146)**	235 (180–320)**	0.000
Transferrin saturation (%)	22.1 (17.5–26.9)	23.1 (17.7–29.2)**	28.3 (21.8–38.5) **	0.000
Dietary intake per day				
Energy (kcal)	2,378 (1,834–3,177)	2,269 (1,966–2,882)	2,425 (1,914–3,015)	0.853
Carbohydrates (%)	44.0 (39.7–49.2)	45.4 (38.9–49.3)	43.7 (39.8–49.4)	0.977
Total fiber (g/day)	23.3 (19.3–31.0)	21.7 (16.8–28.1)	23.8 (18.3–27.2)	0.320
Protein (%)	15.7 (14.6–17.5)	15.8 (14.1–18.4)	15.3 (13.9–17.3)	0.628
Fat (%)	35.8 (30.2–38.5)	35.4 (30.2–41.0)	35.0 (32.5–38.7)	0.981
Monoinsaturated fat (%)	16.6 (13.9–19.9)	17.0 (14.8–20.4)	17.5 (15.0–20.1)	0.642
Polinsaturated fat (%)	4.53 (3.51–5.25)	4.54 (3.81–6.14)	4.79 (3.97–6.43)	0.225
Saturated fat (%)	9.61 (8.11–11.3)	9.36 (7.59–11.0)	8.77 (7.97–3.4)	0.161
Trans fat (%)	0.23 (0.15–0.33)	0.20 (0.13–0.35)	0.18 (0.11–0.29)	0.341
Cholesterol (mg/day)	387 (311–448)	356 (267–481)	357 (269–454)	0.382
Non-marine *n*-3 fatty acids (g/day)	1.01 (0.88–1.38)	1.05 (0.88–1.50)	1.11 (0.88–1.54)	0.691
Marine *n*-3 fatty acids (g/day)	0.69 (0.52–1.27)	0.62 (0.47–1.17)	0.89 (0.44–1.43)	0.472
Dietary iron (mg/day)	14.6 (11.9–16.8)	13.8 (11.3–17.2)	15.2 (12.5–17.9)	0.723
Alcohol (g/day)	5.56 (1.46–25.6)	10.1 (1.71–16.3)	6.97 (1.38–26.0)	0.806
Vegetables (g/day)	221 (121–307)	221 (119–307)	179 (150–279)	0.924
Fruits (g/day)	368 (154–368)	368 (89.3–368)	171 (45.7–368)	0.179
Legumes (g/day)	8.57 (8.57–21.14)	8.57 (8.57–18.0)	8.57 (4.00–16.6)	0.361
Cereals (g/day)	262 (163–357)	244 (156–355)	286 (196–394)	0.266
Dairy products (g/day)	319 (235–518)	328 (221–571)*	211 (114–503)	0.034
Meat products (g/day)	156 (117–198)	156 (95.5–193)	153 (128–182)	0.863
Olive oil (g/day)	25.0 (25.0–50.0)	25.0 (25.0–50.0)	50.0 (25.0–50.0)	0.587
Fish (g/day)	103 (69.4–119)	87.9 (51.3–122)	97.6 (64.9–138)	0.352

Values are median (interquartile range) for continuous variables and number (percentage) for qualitative variables. P refers to the Kruskal-Wallis one-way analysis of variance by tertiles. The symbols (*) or (**) in Tertile 1 column refers to the difference between tertiles 1 and 2; in the Tertile 2 between tertiles 2 and 3 and in the Tertile 3 between tertiles 1 and 3

*$p \leq 0.05$; **$p < 0.01$, adjusted by Bonferroni correction for multiple testing

Table 3 Clinical and dietary characteristics of the studied subjects according to triglycerides tertiles

	Tertile (1*N*=47)	Tertile (2*N*=47)	Tertile (3*N*=46)	*p*
Age (years)	49.0 (41.0–58.0))	52.0 (37.0–57.0)	46.5 (36.8–56.0)	0.394
Sex (men/women)	30/17	32/15	37/9	0.189
Dyslipidemia (FCH/FHTG)	43/4	34/13	30/16	0.008
Body mass index (kg/m^2)	27.7 (25.3–30.2)	27.9 (25.9–29.7)	28.8 (26.6–32.2)	0.349
Total cholesterol (mg/dL)	290 (257–314)	265 (222–310)*	282 (254–322)	0.160
Triglycerides (mg/dL)	198 (179–212)	278 (251–308)**	516 (457–794)**	0.000
HDL cholesterol (mg/dL)	41.0 (36.0–52.0)*	38.0 (30.0–44.0)	34.0 (28.0–39.3)**	0.000
Non-HDL cholesterol (mg/dL)	245 (219–270)	236 (186–266)*	252 (222–290)	0.059
Glucose (mg/dL)	99.0 (91.0–109)	98.0 (89.0–108)	101 (91.5–112)	0.683
GGT (IU/L)	29.0 (18.0–43.0)	32.0 (20.0–52.0)	35.0 (24.8–53.8)	0.234
CRP (mg/L)	2.60 (1.10–4.90)	1.90 (1.13–4.55)	2.30 (1.23–4.35)	0.803
Iron (μg/dL)	86.0 (31.0–103)	84.5 (73.0–112)	94.0 (74.5–117)	0.297
Ferritin (μg/L)	91.5 (41.5–137)	112 (58.3–205)*	154 (89.0–250)	0.011
Transferrin saturation (%)	23.9 (18.2–29.0)	24.5 (17.8–31.2)	25.6 (19.7–30.4)	0.729
Dietary intake per day				
Energy (kcal)	2,216 (1,942–3,036)	2,354 (1,914–2,853)	2,506 (1,922–3,065)	0.808
Carbohydrates (%)	44.7 (39.6–49.1)	43.7 (39.6–49.6)	44.5 (40.5–48.8)	0.917
Total fiber (g/day)	21.9 (17.8–29.7)	22.2 (18.3–28.7)	23.7 (19.0–29.5)	0.580
Protein (%)	16.0 (14.6–17.8)	15.1 (13.9–18.7)	15.7 (14.0–17.3)	0.349
Fat (%)	35.9 (30.5–40.3)	35.9 (30.9–39.9)	34.9 (30.9–38.9)	0.823
Monoinsaturated fat (%)	16.6 (14.8–19.2)	17.5 (15.1–20.7)	17.1 (14.0–19.4)	0.442
Polinsaturated fat (%)	4.70 (3.91–6.16)	4.40 (3.70–5.47)	4.60 (3.76–5.96)	0.735
Saturated fat (%)	8.89 (7.61–10.9)	9.56 (8.46–10.8)	9.01 (7.81–10.9)	0.611
Trans fat (%)	0.19 (0.14–0.28)	0.22 (0.11–0.38)	0.19 (0.13–0.30)	0.577
Cholesterol (mg/day)	369 (297–433)	363 (284–453)	365 (251–480)	0.940
Non-marine *n*-3 fatty acids (g/day)	1.02 (0.88–1.49)	1.05 (0.92–1.32)	1.04 (0.80–1.42)	0.670
Marine *n*-3 fatty acids (g/day)	0.71 (0.53–1.40)	0.53 (0.40–1.14)	0.89 (0.52–1.34)	0.030
Dietary iron (mg/day)	14.0 (12.6–16.2)	14.8 (10.5–16.7)	16.1 (12.4–19.9)	0.245
Alcohol (g/day)	10.2 (3.27–23.0)	5.48 (0.68–14.1)	10.1 (1.44–26.1)	0.278
Vegetables (g/day)	275 (179–311)	179 (121–279)	196 (126–264)	0.036
Fruit (g/day)	368 (129–368)	368 (45.7–378)	314 (54.3–371)	0.249
Legumes (g/day)	8.57 (8.57–16.6)	8.57 (4.00–12.0)	8.57 (8.57–2.1)	0.262
Cereals (g/day)	248 (169–354)	281 (163–378)	286 (172–373)	0.828
Dairy products (g/day)	296 (179–525)	313 (207–525)	287 (135–516)	0.655
Meat products (g/day)	160 (128–185)	155 (108–186)	141 (97.9–195)	0.706
Olive oil (g/day)	25.0 (25.0–50.0)	50.0 (25.0–50.0)	25.0 (25.0–50.0)	0.556
Fish (g/day)	104 (72.6–137)*	77.1 (50.6–105)	106 (69.9–124)	0.032

Values are median (interquartile range) for continuous variables and number (percentage) for qualitative variables. P refers to the Kruskal-Wallis one-way analysis of variance by tertiles. The symbols (*) or (**) in Tertile 1 column refers to the difference between tertiles 1 and 2; in the Tertile 2 between tertiles 2 and 3 and in the Tertile 3 between tertiles 1 and 3

*$p \leq 0.05$; **$p < 0.01$, adjusted by Bonferroni correction for multiple testing

Discussion

In the present study, we have analyzed dietary patterns in subjects with primary HTG. It is well known that environmental and dietary factors play an important role in the phenotype of this hyperlipemia [11, 17]. Hence, the results confirm the important association between obesity with these HTG [5, 27].

However, the composition of diet is quite similar to the reported for general Spanish population [2, 3]. It is typical in our environment a high intake of calories from fat specially monounsaturated fatty acids with figures upper 17%. This dietary pattern is characteristic of Mediterranean countries and it is due to a high consumption of olive oil [22, 23]. Interestingly, omega 3 fatty acids intake is below 2%, lower than previously published data in our environment, and it should be increased in the context of a hereditary HTG.

One of our main objectives was to prove if diet plays an important role in high ferritin concentrations observed in primary HTG. Fleming et al. found an association between dietary patterns and high iron stores in 614 elderly subjects. The dietary factors associated with a higher risk of high iron stores were: fruit and fruit juice, red meat and iron and vitamin C supplements; whereas they found an inverse association with whole grains and sweet baked goods [8]. However, in our study, neither alcohol intake nor different dietary parameters studied were associated significantly with iron concentrations. We have only found a significantly lower intake of dairy products on those subjects in the highest ferritin tertile with respect to the other two tertiles. It is conceivable that this inverse association may have been due to calcium, a known inhibitor of iron absorption [10, 14]. Nonetheless, dietary pattern was homogeneous among tertiles, showing that diet would not explain the differences found in phenotypic expression of this HTG. Our data support the previously suggested idea that the primary mechanisms associated with hypertriglyceridemia, rather than the amount of adipose tissue or diet are the main factors associated with iron deposits in primary HTG [18]. Furthermore, iron deposits in primary HTG did not correlate with the number of metabolic syndrome criteria in this study and in our previous study [18].

One limitation of our study is that the dietary questionnaire was done in a specialized unit where subjects are referred by a general practitioner. That's involves that the most of these subjects may have received a dietary intervention before the FFQ. However, we think this limitation do not invalidate our results since it is homogeneous in all patients which have very different levels of iron deposits.

In summary, the triglycerides concentration is associated with high concentration of ferritin. Patients with primary HTG have a higher prevalence of overweight and obesity but neither triglycerides concentration nor iron deposits are related to dietary patterns valuated through a validated FFQ.

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