P2- 362

Nonalcoholic fatty liver disease and intestinal inflammation in obese children

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Background The prevelance of obesity is increasing in childhood. Nonalcoholic fatty liver diasease (NAFLD) is frequently associated with obesity, insulin resistance (IR), diabetes and dyslipidemia.
Gut microbiota was suggested to play a role in both etiology of NAFLD and also progression to steatohepatitis.
Many fecal biomarkers have been identified but only a few have been studied in children. Faecal Calprotectin (FCP) is a noninvasive marker of intestinal inflammation that can be useful in children.

Objective To evaluate FCP and its association with IR and NAFLD in obese children.

Methods The study included 63 obese children (33F), with a mean age of 12.4±3.1 (range 6.0-18.0) years. Anthropometric measures including height and weight were taken by standard methods. Body mass index (BMI) was calculated as weight (kg)/height(m^2). Height, weight and BMI were expressed according to national standards as SDS and BMI >95% was defined as obesity.
After an overnight fasting, basal blood samples were obtained for glucose, insulin, lipid profile, transaminases and CRP. The oral glucose tolerance test (OGTT) was performed (1.75 g/kg glucose, maximum 75 g). Glucose and insulin levels were measured at 0, 30, 60, 90, 120 minutes.
IR positivity was defined as the sum of insulin values >300 µU/ml on OGTT
FCP was measured by an enzyme-linked immunosorbent assay (ELISA) test. Values greater than 50 µg/g indicate intestinal inflammation and this level was accepted as the cut-off for FCP.
NAFLD diagnosis was made by ultrasound. The patients were divided into 2 groups according to liver echogenicity as normal or NAFLD.

Results

- Age, BMI SDS, HOMA-IR and FCP levels of the patients are seen in Table 1.
- There was a positive correlation between BMI SDS and HOMA-IR ($r=0.274$, $p=0.045$).
- There was no correlation between FCP and BMI SDS or FCP and HOMA-IR.
- IR positivity was 9.1% versus 11.1% in NAFLD and normal echogenicity groups, respectively ($\chi^2=0.036$, $p=0.850$).
- In high FCP (>50 µg/g) patients NAFLD was seen in 92.6% while in patients with normal FCP, NAFLD was seen in 66.6% ($\chi^2=6.000$, $p=0.014$).

Table 1. Features of patients with/without NAFLD

	Normal Echogenicity (n=14)	NAFLD (n=49)	p
Age (years)	13.3±3.5	12.1±2.9	0.310
BMI SDS	2.6±0.5	2.6±0.7	0.908
HOMA-IR	4.5±2.4	4.4±2.0	0.790
Matsuda index	4.2±1.7	2.6±1.6	0.028
IGI	1.2±0.7	2.8±1.7	0.004
FCP (µg/g)	31.0±21.5	75.3±71.7	0.020
Total Cholesterol (mg/dl)	169.5±33.7	186.2±40.4	0.644
Triglyceride (mg/dl)	101.5±27.9	116.6±50.5	0.529
HDL-C (mg/dl)	52.4±10.3	47.6±9.6	0.06
LDL-C (mg/dl)	116±48.8	104.8±30	0.721
AIP	2.1±1.2	2.6±1.4	0.275
LDL-C/HDL-C	2.3±1.0	2.3±0.8	0.753
ALT (U/L)	23.3±13.1	32.4±25.6	0.203
AST (U/L)	22.5±7.7	29.5±20.2	0.508
C-reactive protein (mg/L)	2.9±2.7	3.0±1.6	0.715
Uric acid (mg/dl)	4.7±1.6	5.0±1.2	0.449

BMI: Body mass ındex, FCP:Faecal calprotectin, HOMA-IR: Homeostasis model assesment -insulin resistance, IGI: Insulinogenic index

Homeostasis model assessment-insulin resistance
$$(\text{HOMA-IR}) = \frac{\text{Insulin (µU/ml)} \times \text{glucose (mmol/l)}}{22.5.}$$

$$\text{Matsuda index} = \frac{10\,000}{\sqrt{(\text{glucose}_0 \times \text{Insulin}_0) \times (\text{glucose}_{mean} \times \text{insulin}_{mean})}}$$

$$\text{Insulinogenic index (IGI)} = \text{Insulin}_{30}\text{-Insulin}_0\ (\Delta\text{Insulin}_{30}) / \text{Glucose}_{30} - \text{Glucose}_0\ (\Delta\text{Glucose}_{30})$$

Atherogenic index of plasma (AIP) = triglyceride/HDL-C ratio

Conclusion

FCP could be a helpful method during the follow up of obese children with NAFLD.

References

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