Plasminogen activator inhibitor-1(PAI-1) as a marker of insulin resistance in obese adolescents

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Background

Obesity is associated with a chronic inflammatory response, characterized by abnormal adipokine production, and the activation of some pro-inflammatory signalling pathways, resulting in the induction of several biological markers of inflammation. Pro-inflammatory molecules such as serum tumor necrosis factor- α (TNF- α) and plasminogen activator inhibitor-1 (PAI-1) levels are increased in obese children. On the contrary plasma adiponectin levels which is considered an anti-inflammatory molecule, are decreased in obese and/or type 2 diabetic children.

Objective

We aimed to evaluate the inflammation markers and the correlation with insulin resistance in obese adolescents.

Research design and methods

Seventy-eight obese children (38 male; age 14,3 \pm 1,8 years) and 60 healthy adolescents (21 male; age 14,9 \pm 2,2 years) were included in the study. BMI SDS, waist/hip ratio, systolic and diastolic blood pressure values of all participants were recorded. Serum fasting lipid profile, glucose, insulin, plasminogen activator inhibitor-1 (PAI-1), TNF- α , adiponectin levels of obese adolescents were compared with healthy controls.

Results

Obese adolescents had significantly higher BMI-SDS, waist-hip ratio, systolic and diastolic BP measurements (p<0,001) (Table 1). As anticipated, obese adolescents had higher insulin and HOMA-IR values, indicative of insulin resistance, and also exhibited higher triglyceride, LDL and lower HDL concentrations compared to control subjects. PAI-1 levels were significantly elevated and TNF- α levels were lower in the obese group (p<0,001). Adiponectin levels did not differ between two groups (Table 2). PAI-1 was positively correlated with HOMA-IR (r=0,445, p<0,05) and TNF- α was negatively correlated with BMI and HOMA-IR in obese adolescents (r=-0,463 and r=-0,319 respectively; p<0,05). No significant correlation was found between adiponectin and clinical and metabolic parameters. The insulin resistant obese adolescents had higher BMI, insulin, PAI-1 and lower TNF- α levels (p<0,05). There was no significant difference in lipid profiles between the insulin resistant and noninsulin resistant obese adolescents (Table 3).

Table 1. Clinical features of obese and non-obese adolescents

	Obese group	Control group	р
	(n=78)	(n=60)	
Age (year)	14,3±1,8	14,9±2,2	0,063
BMI	31,8±3,9	20,2±3,3	<0,001
BMI SDS	2,79±0,39	0,05±1,17	<0,001
Waist/hip ratio	0,95±0,05	0,88±0,04	<0,001
Systolic BP (mmHg)	120 (90-160)	110 (70-120)	<0,001
Diastolic BP (mmHg)	75 (50-100)	70 (40-80)	<0,001

Table 2. Comparisons of metabolic and inflammatory markers between obese and non-obese adolescents

	Obese group	Control group	р
	(n=78)	(n=60)	
Glucose (mg/dl)	89±8	87±8	0,129
Total chol (mg/dl)	162±28	154±31	0,100
Triglyceride (mg/dl)	141±73	96±39	<0,001
LDL chol (mg/dl)	90±22	81±25	0,032
HDL chol (mg/dl)	44±9	53±13	<0,001
Insulin (uIU/mL)	22,3±14,5	10,9±5,3	<0,001
HOMA- IR	4,97±3,5	2,36±1,2	<0,001
Adiponectin (ng/ml)	15,2±8,1	17,4±7,3	0,100
TNF-α (pg/ml)	0,13 (0,1-2,8)	0,28 (0,1-0,4)	<0,001
PAI-1 (pg/ml)	79,2(12,4-500)	40,8 (10,2-291,5)	<0,001

Table 3. Comparisons of clinical and metabolic parameters between insulin resistant and noninsulin resistant obese adolescents

	Insulin resistant obese	Non insulin resistant	р
	(n= 51)	obese	
		(n=27)	
Age (year)	14,4±1,6	14,0±2,2	0,470
BMI	32,7±3,9	30,1±3,4	<0,001
Glucose(mg/dl)	90±8	88±8	0,136
Total chol (mg/dl)	161±29	163±27	0,846
Triglyceride (mg/dl)	152±80	122±53	0,08
LDL chol(mg/dl)	88±23	93±22	0,394
HDL chol (mg/dl)	44±9	44±10	0,877
Insulin (uIU/mL)	28,3±14,6	11±2,7	<0,001
Adiponectin (ng/ml)	14,4±7,8	16,8±8,7	0,223
TNF-α (pg/ml)	0,15±0,06	0,30±0,51	0,015
PAI-1 (pg/ml)	147,8±123,2	83,5±78,8	0,001

Conclusion

A positive correlation between PAI-1 and indices of insulin resistance was demonstrated in this study. Our results suggest PAI-1 can be used as a marker of insulin resistance in obese adolescents.



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