obesity [10] and type 2 diabetes [11], which are states characterized by insulin resistance (IR) and typically also observed in gestational diabetes mellitus (GDM).

Acute administration of recombinant visfatin to mice leads to a reduction of plasma glucose independent of changes in plasma levels of insulin. Thus it works synergistically with insulin to lower blood glucose concentrations [9]. Chronic elevation of visfatin in mice reduces insulin plasma concentrations [9], and it was suggested that visfatin improves insulin sensitivity [12]. Visfatin affects the insulin signal transduction pathway by inducing tyrosine phosphorylation of the insulin receptor and IRS1 and 2 (insulin receptor substrate 1 and 2) in the liver. Furthermore, an autocrine/paracrine function on visceral adipose tissue as well as an endocrine role modulating insulin sensitivity in peripheral organs might be modes of action [12].

To evaluate the role of visfatin in GDM we determined this novel adipocytokine in women with GDM and healthy pregnant controls.

Methods

All subjects were carefully instructed about the aims of the study and written informed consent was given. Thirty women with GDM (mean age, 36±2 years) diagnosed during pregnancy weeks 24-28 and 30 healthy pregnant controls (mean age, 29±2 years) were included in the study. All subjects were non-smokers.

Women were diagnosed as GDM if two or more of the four glucose levels in the tolerance test exceeded the National Diabetes Data Group Criteria as follows:

Fasting more than ≥5.3 mmol/l

1-hour postload 75-g glucose value ≥10.0 mmol/l

2-hour postload glucose value ≥8.6 mmol/l

3-hour postload glucose value ≥7.5 mmol/l

The (OGTT) was performed between 24th and 28th weeks of gestation.

Blood samples were obtained directly from a cannulated vein for the purpose of a routine glucose challenge test at 24-28 weeks of gestation. The serum was separated by centrifugation, and stored at -20 ^{o}C until further analysis. Serum visfatin was analyzed using kit manufactured by (BIO VISION).

Inclusion criteria were signed informed consent, absence of a clinically relevant illness, normal findings in the medical history and physical examination except for GDM, and normal laboratory values. Subjects were excluded if any clinically relevant abnormality was found as part of the screening or in any of the laboratory tests including circulating anti-insulin antibodies and anti-islet cell antibodies. No subject was on a special diet or reported to have any medication, including "over-the-counter" drugs, at the time of blood sampling.

Results

Table 1 shows the clinical results of our subjects, both those with GDM and those with healthy control groups. Serum visfatin concentration was significantly lower in the GDM group (Mean= 0.27±0.1) than in the healthy control group (Mean= 1.37±0.25) (p-value= 0.0001) as shown in and Figure 1.

Table 1. Clinical Data

Parameters	Controls	GDM	P-Value
Visfatin Level (ng/ml)	1.37±0.25	0.27±0.1	0.0001

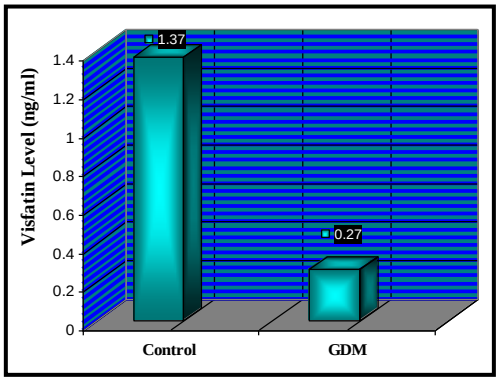

Figure 1. Serum Visfatin Level plotted for both GDM subjects and those who were in the healthy group