1. Introduction

Within the last decade, primary aldosteronism has been recognised as a frequent cause of arterial hypertension and is estimated to account for up to 12% of the hypertensive subjects. Patients with primary aldosteronism have an increased risk of developing the metabolic syndrome, with alterations in glucose homeostasis being the best established component, in addition to blood pressure elevation.

2. Methods

Review of the literature and evaluation of metabolic comorbidities in primary aldosteronism treated between 1990 and 2006 in five different German centres (German Conn’s Registry). The analysis was done in 555 patients (327 males and 228 females, aged 55 ± 13 years) with primary aldosteronism retrospectively and in 100 patients prospectively during follow-up.

3. Results

J. Conn was the first to report an increased incidence of impaired glucose tolerance in patients with primary aldosteronism. Subsequent studies generated conflicting data on its prevalence and its potential mechanisms. Most of the discrepancies might be related to limited sample sizes. Carranza et al. (1991) showed decreased insulin receptor levels and affinity in subcutaneous adipose tissue of a patient with primary aldosteronism. Another finding is that of Shimamoto et al. (1994) who described impaired pancreatic insulin release. In addition to the effects of aldosterone per se, hypokalemia in primary aldosteronism may play a role. For example, Giacchetti et al. showed a positive correlation between [K+] and metabolic alteration (2007). The recent study by Catena et al. (2007) showed that patients with primary aldosteronism have a greater insulin response to an oral glucose load and lower insulin sensitivity than normotensive controls. Treatment of primary aldosteronism restored parameters of insulin sensitivity to normal.

4. Conclusions

It appears that primary aldosteronism is associated with a reversible type of insulin resistance.