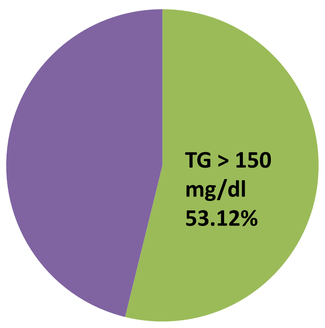
Cushing syndrome and dyslipidemia

AZZOUG S, RABEHI L, HANNACHI S, MEDJDOUBI H, CHENTLI F
Endocrine and Metabolic diseases department Bab El Oued Hospital Algiers

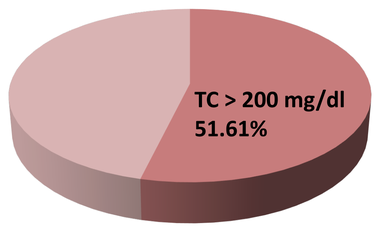
Introduction: Cortisol activates lipolysis in adipose tissue resulting in the release of free fatty acids into the circulation, it also activates cholesterol and triglycerides synthesis. The consequence is an increase in total circulating cholesterol and triglycerides with their inherent risks on the cardiovascular system. The aim of our work was to assess the prevalence and characteristics of dyslipidemia in Cushing syndrome (CS).

Material and methods: This is a retrospective study concerning 51 CS (44F/7M) in whom we looked for the presence of hypertriglyceridemia (triglycerides level ≥150 mg/dl) and/or hypercholesterolemia (total cholesterol (TC) level ≥200 mg/dl). Thereafter we looked for the characteristics of dyslipidemia in CS. Their mean age was 32.34 ± 10.42 years. CS was secondary to cushing disease in 82% and to adrenal adenoma in 18%.

Results: 53.12% of patients have hypertriglyceridemia. Triglycerides (TG) level was between 150 and 199 mg/dl in 41.17% and ≥ 200 mg/dl in 48.83%. 88.23% of hypertriglyceridemic patients have diabetes and hypertension. 51.61% of patients have hypercholesterolemia. 68.75% of hypercholesterolemic patients have hypertriglyceridemia, 87.5% have diabetes and 87.5% have hypertension.



Hypertriglyce ridemia



Hypercholest erolemia

Discussion: Glucocorticoids (GC) have multiple and complex effects on lipid metabolism, dyslipidemia is one of the prominent features of CS. Dyslipidemia is usually associated with other traits of metabolic syndrome ie central obesity, diabetes and hypertension which highly increase cardiovascular morbidity and mortality. Despite improvement in CS treatment mortality remains elevated [1]. GC increase adipogenesis in central fat while they increase lipolysis in peripheral fat. GC promote pre-adipocyte differentiation in central fat. GC enhance the activity of hormone-sensitive lipase and adipose triglycerides lipase in adipocytes. In liver GC increase lipogenesis, triacylglycerides storage, very low density lipoproteins (VLDL) secretion and hepatic steatosis [2]. In CS as observed in our study there is an increase in cholesterol and triglycerides secondary to an increase in VLDL and low density lipoproteins (LDL) [3]. Total cholesterol is reported to be elevated in 25-52% of patients whereas triglycerides are elevated in 7-35% of patients [4]. High density lipoproteins-Cholesterol (HDL-C) levels are variable in CS, while some authors report that they are decreased, others report that HDL-C is either normal or increased [5].

Conclusion: Dyslipidemia is frequent in cushing syndrome. Hypertriglyceridemia and hypercholesterolemia are frequently associated with hypertension and/or diabetes which may explain the increased risk of cardiovascular disease in cortisol excess states.

References:

1°- Etxabe J, Vasquez JA. Morbidity and mortality in Cushing's disease: and epidemiological approach. Clin Endocrinol 1994; 40: 479–484.
2°- Vegiopoulos A, Herzig S. Glucocorticoids, metabolism and metabolic diseases. Mol Cell Endocrinol 2007; 275: 43–61
3°- Arnaldi G, Scandali VM, Trementino L, Cardinaletti M, Appolloni G, Boscaro M. Pathophysiology of Dyslipidemia in Cushing's Syndrome. Neuroendocrinology 2010;92(suppl 1):86–90
4°- Greenman Y. 4°- ArnaManagement of Dyslipidemia in Cushing's Syndrome. Neuroendocrinology 2010;92(suppl 1):91–95
ldi G. High cardiovascular risk in patients with Cushing's syndrome according to 1999 WHO/ISH guidelines. Clin Endocrinol (Oxf) 2004; 61: 768–777.

17 ECE 28-EP
Adrenal
said azzoug
DOI: 10.3252/pso.eu.17ece.2015
Poster presented at: ECE2015 16–20 May 2015 DUBLIN, IRELAND 17th EUROPEAN CONGRESS OF ENDOCRINOLOGY
PosterSessionOnline.com