ATRIAL FIBRILLATION CAN CAUSE MAJOR HYPERGLYCEMIA

V. RIGALLEAU (1), L. BAILLET (1), M. HOCINI (2), H. GIN (1)

SUMMARY - We report the case of a 66 years old woman with a well controlled, insulin-treated, type 2 diabetes, who experienced a ten-fold increase of her daily insulin needs (from 21 to 215 U/day) after the onset of a symptomatic atrial fibrillation. Check-up for another cause of insulin resistance was negative, and insulin doses could be decreased to preceeding values only after electric cardioversion. Symptomatic atrial fibrillation should be considered as a potential cause of hyperglycemia.

Key-words: atrial fibrillation, type 2 diabetes, hyperglycemia.

RÉSUMÉ - Fibrillation auriculaire : cause possible d’une hyperglycémie majeure.
Nous rapportons le cas d’une femme de 66 ans, diabétique de type 2, bien équilibrée sous insuline, dont les besoins insuliniqques quotidiens ont décuplé (de 21 à 215 U/j) après la survenue d’une fibrillation auriculaire symptomatique. La recherche d’une autre cause d’insulino-résistance a été négative, et les doses d’insuline n’ont pu être ramenées aux valeurs initiales qu’après réduction par choc électrique. Une fibrillation auriculaire symptomatique peut entrainer une hyperglycémie majeure.

Mots-clés : fibrillation auriculaire, diabète de type 2, hyperglycémie.
Atrial fibrillation (AF) affects about 3% of subjects over 65 years of age [1]. Diabetic subjects are twice more prone to stroke when AF is present [2]. AF is not known to affect glucose homeostasis. We report a ten-fold increase of insulin needs in an insulin-treated type 2 diabetic woman at the onset of AF, which resolved after cardioversion.

A 66-year-old woman with type 2 diabetes since 1990 had been managed for three years with low daily doses of insulin and a good metabolic control (HbA1C: 5.6 to 7.7%). Her past medical history included situs inversus, arterial hypertension since 1990, nephrectomy for a renal carcinoma (January 1998), and moderate chronic renal failure (serum creatinin 160 µmol/L). Repeated electrocardiograms were normal during these years.

On July 1999, atrial fibrillation and a moderate heart failure (NYHA class II) was found after a lipothymia, and she was hospitalized in a cardiologic ward. Plasma glucose level was increased (15 mmol/L). Medical treatment (amiodaron, digoxin, furosemide and oral anticoagulation) reduced ventricular rate to 65/min, and she went back home. Capillary glucose levels remained very high (15 to 20 mmol/L) despite insulin doses were increased (from 21 to 70 U/day, as shown on the figure 1).

Ten days later, persistant hyperglycemia led to the admission in the diabetologic unit. Plasma glucose levels were still high (15 mmol/L), without ketoacidosis nor hyperosmolarity. Extensive check-up for an intercurrent cause of hyperglycemia was negative: body weight was stable, serum potassium, thyroid hormons, urinary catecholamines were in the normal range. There was no biological inflammatory syndrom, no urinary infection, serum creatinin was stable (176 µmol/L). Regular insulin was added before each meal, and doses were increased until plasma glucose levels became acceptable, needing 215 U insulin/day. Clinical signs of heart failure were not present, furosemide was withdrawn for 5 days. This did not modify insulin requirements. Atrial fibrillation persisted.

Cardioversion was then successfully performed by electric shock. Insulin needs immediately began to decrease, and they were back to prevailing values (26 U/day) one month later. Two years later, metabolic control remains fair with similar insulin doses.

Our patient presented a transient state of insulin resistance, as demonstrated by a ten-fold increase of her insulin needs that reached 3U/kg body weight. This occurred soon after the onset of a symptomatic AF, and quickly resolved after cardioversion. No other cause of hyperglycemia was found, and moderate doses of insulin allowed a good metabolic control three years before and two years after the arrythmia. Congestive heart failure impairs insulin sensitivity [3], however in our patient clinical signs of heart failure disappeared with digoxin and furosemide, whereas insulin requirements remained high until cardioversion. Digoxin has a steroid structure, however glycemia increased before its introduction in our patient. Although AF is not associated with increased plasma catecholamine levels in patients with congestive heart failure [4], sympathetic tone is increased at the onset of tachyarrhythmia [5], which may influence insulin action. AF, at least when it is symptomatic, should be considered as a possible cause of hyperglycemia in diabetic patients.

**REFERENCES**


© Masson, Paris, 2002