Medical Research Archives





Published: November 30, 2023

Citation: Farsky, S., 2023. Liraglutid and insuline resistance at heart failure patient. Medical Research Archives, [online] 11(11).

https://doi.org/10.18103/mra. v11i11.4614

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DOI:

https://doi.org/10.18103/mra. v11i11.4614

ISSN: 2375-1924

CASE REPORT

Liraglutid and insuline resistance at heart failure patient.

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ABSTRACT

Cardiologists play a key role in the identification and subsequent management of patients with cardiovascular disease and comorbid obesity. In addition to the overall cardiovascular risk profile one must also consider the residual cardiovascular risk in patients in whom, despite properly treated comorbidities, dyslipidemia and hypertension), in connection with ongoing obesity, the chronic inflammatory process of the vascular wall continues, and myocardial fibrosis and the HFpEF phenotype develop. Since last year, it has also been able to indicate modern pharmacotherapy such as GLP-1 receptor agonists (RA) and SGLT2 inhibitors for indications other than DM2T. GLP-1 receptor agonists, specifically liraglutide, regulates appetite by increasing the feeling of satiety while simultaneously reducing the feeling of hunger and the desire to consume more food. This leads to significant weight loss and subsequent reduction in the risk of hypertension, arrhythmias (including atrial fibrillation), ischemic heart disease and heart failure. It can be expected that this type of treatment for patients with the HFpEF phenotype and obesity will gain significant traction in the near future. In this case liraglutid application led to signiffcant weight reduction. This enabled reduction in the extent of pharmacotherapy especially extreme reduction of daily insulin burden dosage together with dramatic decrease blood glucose levels near to normal values. The most probable explanation for this impressive improvement is the insulin resistance reduction together with weight reduction. This was accompanied with improvement of the patient's quality of life, especially of effort dyspnoe reduction and with the renal function improvement too.



Case report

MAY 2013: first examination dated in our database of cardiology care dept. for out patients, 65 years old man, weight 144 kg, height 178 cm, waist circumferrence 143cm. His history: arterial hypertension since 1993, diabetes 2 type since 2008 on peroral treatment, paroxysmal atrial fibrilation since March 2013, diameter of the left atrium 50mm on ECHO, invasive coronarography negat. Therapy: telmisartan, metoprolol, nitrendipin, warfarin, propafenon, digoxin, spironolacton, statin.

NOVEMBER 2013: effect of therapeutic lifestyle changes: regular sinus rhythm, weight 133kg, waist circumferrence 134cm, spironolacton substituted by eplerenon (asymetric gynecomastia), digoxin ex.

MAY 2014: weight 126kg,

SEPTEMBER 2014: atrial fibrilation, propafenon substituted by flekainid, then atrial flutter, switch from flekainid to amiodaron and digoxin, warfarin substituted by apixaban.

FEBRUARY 2015: sinus rhythm, weight 129kg.

DECEMBER 2015: weight 140kg, BP 170/90 mmHg, atrial flutter with a-v blockage 4:1, ankle oedema on both sides, Hb 115g/l, urinary acid 499umol/l, creatinin 119 umol/l, added allopurinol, urapidil, furosemide.

MAY 2017: intensification of the diabetes treatment, added insulin application, dosage escalation during the next years, on ECHO: left atriium diameter 52mm, left ventricle diameter 62mm, systolic function of the left ventricle preserved, moderate mitral and tricuspidal regurgitation.

DECEMBER 2017: sinus bradycardia 46/min, amiodaron and digoxin ex, sick sinus syndrom brady-tachycardia form, hepatomegaly.

JULY 2018: bariatric procedure contraindicated by surgeon because of age and health status.

DECEMBER 2018: creatinin 169umol/l, urinary acid 652umol/l.

OCTOBER 2019: weight 158 kg, hospitalisation for heart failure, confirmed by increased NT-BNP level, therapeutic changes from sartan to ARNI, from warfarin to apixaban, increased furosemid dosage.

FEBRUARY 2021: weight 159kg, O2 peripheral saturation 93%, start of partial fasting (8:16).

MAY 2021: signifficant dyspnoe, NYHA III, ECHO: left atrium diameter 56mm, left ventricle diameter 70mm, EF of the left ventricle 0,50, E/A 0,7.

MARCH 2022: attempt to implantate permanent cardiostimulation device unsuccesfull because of anatomic venous abnormalities. Brain MRI: postischemic frontal area defect right side.

Creatinin 220 umol/l, kalium 5,4 mmol/l, Hb 121 g/l, diabetic nephropathy. Weight 160kg. Added iron supplement. In spite of high insulin dosage (Humalog 3x20 units and basal 2x50 units) 160 units per day the glucose level were around 20mmol/l.

JULY 2022: weight 161 kg, starting of liraglutid daily application of 0,6mg s.c., dosage escalation every week to 1,2, 2,4, 3,0 mg.

OCTOBER 2022: hunger feeling was signifficantly reduced, eating of smaller portions, without craving between main time

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schedule of eating. Weight reduced to 151kg during first 3 months of liraglutid application (cca minus 100g per day), insulin dosage was reduced to 60 units daily (Humalog ex, basal insulin reduced to 60 units) what was accompanied by signifficant glucose level reduction to around 8 mmol/l. Creatinin level was 166 umol/l, acidum uricum level was 323 umol/l.

JANUARY 2023: weight 157kg, fat content 50%, water content 40%. ECHO: left ventricle diameter 60mm, EF 0,5, left atrium diameter 54mm, tricuspidal regurgitation not detected, E/A 0,7. creatinin 128 umol/l, kalium 4,5 mmol/l.

Conclusion

In the LEADER study, liraglutide significantly reduced the incidence of major adverse cardiovascular episodes at diabetic patients by 13 %, HR 0.87 (p = 0.005). GLP-1 receptor agonists reduce CV episodes, including the risk of hospitalization for heart failure, not only in patients with clinical CV disease, but also in patients with increased CV risk without clinically apparent CV disease.

Our patient suffered from the HFpEF disease, which is developing at the obese patients with metabolic syndrome and/or 2nd type diabetes and arterial hypertension. In spite of absence signifficant myocardial ischemia, left heart dilatation and diastolic dysfunction together with increased myocardial fibrosis have led to heart failure. Liraglutid daily application during 4 months of treatment, following by weight reduction and reduced insulin resistance, was effective in attenuation of heart failure symptoms too. In this regard application of liraglutid at the obese patients with heart failure means a new, promising therapeutic approach.



Conflict of Interest Statement:

Funding Statement:

None

None

Acknowledgement Statement:

None



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