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CASE REPORT

Premature atherosclerosis in patients with growth hormone deficiency and diabetes mellitus

Bosevski M, Tosev S, Sadikario S

Clinic of Cardiology, Medical Faculty Skopje, Macedonia. **marijanbosevski@yahoo.com**

Abstract: In this article cases of two sisters with premature atherosclerosis have been described. The first one aged 31 with diagnosis of growth hormone deficiency (GHD) and diabetes mellitus presented with calf intermittent claudication as a result of tibial arteries occlusions. The second one aged 34 with impaired fasting glycemia and without any sign of GHD presented with sudden calf pain as a result of tibial posterior arterial acute occlusion. These cases support the hypothesis that in GH deficiency patients is a higher incidence of diabetes mellitus and early atherosclerosis and they are more vulnerable to vascular thrombotic events *(Tab. 1, Ref. 5).* Full Text (Free, PDF) *www.bmj.sk.*
Key words: premature atherosclerosis, growth hormone, diabetes mellitus.

Case 1

A female patient (pt), (31 years old), came to Vascular Lab complaining of calf intermittent claudication after walking distance of 200 meters.

Growth hormone (GH) deficiency was diagnosed in the patient by low serum level of GH and insulin like-growth factor-1. She was treated with GH substitution previously. The patient also suffered of type 2 diabetes treated with 24 units insulin daily (Tab. 1).

Doppler ultrasound presented dampened signals on the dorsal pedal and posterior tibial arteries on both sides, with ankle--brachial indexes values of 0.69 on the right side and 0.65 on the left side. Peripheral angiography showed no significant stenosis of the aorta, iliac arteries, superificial femoral and popliteal arteries. There were occlusions of tibial arteries on both sides. Antiplatelet and statin therapy was initiated and the patient was referred to vascular surgery division.

Case 2

The second patient, sister of the first one, aged 34 years, came to Clinic because of sudden calf pain and coldness. Pathological signal of the left tibial artery was detected by Continuous Doppler with ankle brachial index 0.35. On the right side there was a normal finding and ABI 1.0 was described. Angiography presented left posterior tibial artery with gracile morphology and occlusion in the distal part. No significant changes of right leg arteries were presented.

The patient height was 150 cm but there were no signs of GHD. Impaired fasting glycemia was detected (Tab. 1).

Therapy with low-molecular heparin was initiated with oral anticoagulation continuation. Control Doppler examination showed value of ABI 0.6.

Discussion

Merimee described first case of GH deficiency and premature atherosclerosis in 1973. Five years later in another paper the same author mentioned that patients with GH deficiency and premature atherosclerosis reported a high incidence of diabetes mellitus or glucose intolerance. One of the reported patients had a diagnosis of type 2 diabetes mellitus (1).

GHD patients may account for higher prevalence of vascular disease in coronary, leg and cerebovascular region and increased

Tab. 1. Basic characteristics of patients at hospital admission.

Parameters	1st patient	2nd patient
Glycemia mmol/L	10.2	6.1
Cholesterol	10.6	6.1
HDL	1.2	1.1
LDL	8.4	3.9
Triglycerids	2.1	2.2
Urea	5.2	4.5
Creatinin	145.0	95.0
STH g/ml	14.7	17.0
Insulin IE/ml	160.0	–
Blood pressure	130/80	120/70
Weight (cm)	147.0	152.0

Clinic of Cardiology, Medical Faculty Skopje, Macedonia

Address for correspondence: M. Bosevski, MD, Clinic of Cardiology, Medical Faculty Skopje, Macedonia Vasil Gorgov 20/49, 1000 Skopje, Macedonia.

cardiovascular death rate (2). The exact mechanism for such propensity to atherosclerotic vascular disease is not yet completely clear. It is thought to be the result of changed rhehological factors and high blood viscosity in these patients. But in general these patients reported lipid abnormalities (3). Both patients in our case study reported lipid abnormalities. Study on coagulation factors in our patients was not done.

Possible explanation for the interplay of GHD and atherosclerosis is that hormonal factors may be the initiating mechanism with subsequent secondary metabolic abnormalities rolling as risk factors for vascular disease (4). This seems to be more evident in female hypopituitary patients compared with their male counterparts. Female patients have higher frequency and more pronounced abnormalities of various risk factors as well as surrogate markers of early vascular disease. Our patients were also females.

Patients with growth hormone deficiency presented with early atherosclerosis and vulnerability for vascular thrombotic events. The sustained improvement of the adverse lipid profile and body composition suggests that GH replacement therapy may reduce the risk of cardiovascular disease and the premature mortality seen in hypopituitary patients with untreated GHD (5).

These cases support the hypotheses that GH deficiency patients suffer more frequently from diabetes mellitus and early atherosclerosis and vulnerability for vascular thrombotic events. It remains to be shown by future studies whether growth hormone treatment in adult patients or risk factors prevention may ameliorate vascular disease.

References

1. Merimee TJ. A follow-up study of vascular disease in growth-hormone-deficient dwarfs with diabetes. New Engl J Med 1978; 298 (22): 1217—1222.

2. Moreno Esteban B, Garrido M, Rejas J, Lorenzo L, Rodriguez P, Tresguerres JA. Growth hormone-releasing hormone: Clinical aspects 1993; 6 (3—4): 311—316.

3. Abdu TA, Neary R, Elhadd TA, Akber M, Clayton RN. Coronary risk in growth hormone deficient hypopituitary adults: increased predicted risk is due largely to lipid profile abnormalities. Clin Endocrinol 2001; 55 (2): 209—216.

4. Elhadd TA, Abdu TA, Clayton R. Hypopituitarism and atherosclerosis. Ann Med 2001; 33 (7): 477—485.

5. Abs R, Feldt-Rasmussen U, Mattson AF, Monson JP, Bengtsson BA, Goth MI, Wilton P, Koltowska-Haggstrom M. Determinants of cardiovascular risk in 2589 hypopituitary GH-deficient adults – a KIMS database analysis. Europ J Endocrinol 2006; 155 (1): 79—90.

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SCIENTIFIC AND CLINICAL INFORMATION

The problems of pediatric glaucoma

Busanyova B, Gerinec A

Department of Pediatric Ophtalmology, Children's Teaching Hospital, Comenius University in Bratislava, Slovakia

Summary

The authors in the work define and introduce classification of the pediatric glaucomas and analyze the new opinions on the clinical and genetic aspects of this affection and their significance for ophthalmologic practice. Also they have dealt with recommended process in diagnostics. They analyse modern therapeutical problems of glaucoma in children. Authors have followed long-term the collection about 300 different pediatric glaucomas. Its purpose is by securing of reduction of intraocular pressure to preserve the satisfactory visual acuity and the visual field, eventually to reduce the progress of optic nerv damage. They introduce the brief survey about the newest pharmacological therapy in the conservative treatment of pediatric glaucoma and also they recommend the algorithm of surgical treatment, which is valid at this time and applied in their own praxis.

Key words: pediatric glaucoma

Pathogenesis of glaucomatous damage

Evaluation of IOP is caused by increased resistance to aqueous outflow in drainage channels

Congenital glaucoma

Most causes are sporadic, in 10% the inheritance is autosomal recessive

Aqueous outflow is impared as a result of trabeculodysgenesis which is defined maldevelopment of the trabeculum, which is not associated with any other ocular anomalies

Clasification:

- True congenital glaucoma. IOP becomes elevated during i.u.life
- Infantile laucoma disease becomes menifest prior to the childs third birthday
- Juvenile glaucoma- pressure rise develops after the third birthday but before the age of 16 years

Clinical features

- Corneal haze – is caused by epithelial oedema and may be associated with lacrimation, photophobia and blefarospasm
- Buphthalmos large eye as a result of elevated IOP
- Breaks in Descemet membrane is associated with endothelial decompensation and a sudden influx of aqueous intio the corneal stroma
- Optic disc cupping cup disc ratio is greater than 0.3

Therapy

- The managment is always surgical – trabeculectomia
- Is drainage procedure lowers IOP by creating a new channel for aqueous outflow between the anterior chamber and subtenon space
- The initial evaluation should be performed under general anesthesia.
- Examination of optic disc should be undertaken first, followed by measurement of IOP and corneal diameters and gonioscopy.

This study was presented on the Meeting of the Slovak Medical Society, on the March 3, 2008 in Bratislava.