

KONTROLL PONT

Kardiometabolikus Prevenció Szakrendelés



A **Kardiometabolikus Prevenció Szakrendelés** olyan működő kórházban vagy rendelőintézetben kialakított központ, ahol a háziorvos által elvégzett szűrővizsgálatok alapján beutalt betegek komplex belgyógyászati (kardiológus, diabetológus, lipidológus, hipertónológus) kezelése történik.

E tevékenység kiegészül:

- dietetikus által végzett tanácsadással és követéssel,
- gyógytornász által végzett állapotfelméréssel és kezeléssel,
- a fenti terápiákat támogató pszichológus munkájával,
- diplomás ápoló gondoskodik
 - a betegek előjegyzéséről,
 - a betegáramlás szakemberek közötti zökkenőmentességéről,
 - a betegek kezelési folyamatának követéséről.

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OBESIT^{LOGIA} HUNGARICA

THE SIGNIFICANCE AND DIAGNOSIS OF VISCERAL OBESITY

Pre-Congress Satellite Meeting
of the 15th European Congress on Obesity

8th Congress of the Hungarian Society for the Study of Obesity

20th–21st April, 2007.

EUROPA CONGRESS CENTER
(1121 BUDAPEST, HÁRSHEGYI ÚT 5-7.)
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Három ász
az Ön kezében



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A ma, terápiaja a jövő, záloga.

EGY ANTIIDIABETIKUM A CV PREVENCIÓBAN

Hatóanyag: 50 mg acarboseum, ill. 100 mg acarboseum tablettként. **Javallat:** Diabetes mellitusban a betegek diétás kezelését kiegészítő terápia. **Ellenjavallatok:** acarbose-zal szembeni túlérzékenység; Mivel gyermekek és fiatalok esetében a hatás és a tolerabilitás tekintetében eddig nem áll rendelkezésre kellő tapasztalat, 18 éven aluli betegeknek alkalmazása kerülendő; Krónikus bélbetegségek, melyek kifejezett emésztési és reszorpciós zavarokkal járnak; Olyan állapotok, melyek a fokozott bélgáz képződés folytán rosszabbodhatnak (Pl. Roemheld szindróma, nagyobb sűrűségű bélszékületek és bélfekélyek). Glucobay nem adható terhességben és szoptatás anyáknak, mivel kellő tapasztalat nem áll rendelkezésre. **Mellékhatások:** Gyakran felfúvódás és bélgöngöl, esetenként hasmenés és hasfájás. Háztartási cukor (nádcukor, répacukor) vagy ilyen tartalmú élelmiszer a kezelés alatt könnyen erős mértékben csökkenteni kell a tablettát, ill. inzulin adagját. Hipoglikémia fellépésekor tekintetbe kell venni, hogy a háztartási cukor (nádcukor, répacukor) a Glucobay-kezelés alatt lassabban emésződik és szívódik fel, így alkalmatlan a hipoglikémia tüneteinek gyors megszüntetésére. Ezért a háztartási cukor helyett ilyenkor szőlőcukrot kell alkalmazni. **Megjegyzés:** **csomagolás:** 50 mg 30 x, 120 x, 100 mg 30 x, 120 x. További információkat a részletes alkalmazási előirat tartalmaz. **Alk. előírás OGYI eng. száma:** 15 336/41/2004

Glucobay 100 mg tablettá 120x	Fogyasztói ár: 4622 Ft.	Normatív támogatás: 2542 Ft.	Normatív térítési díj: 2080 Ft.
Glucobay 100 mg tablettá 30x	Fogyasztói ár: 1496 Ft.	Normatív támogatás: 823 Ft.	Normatív térítési díj: 673 Ft.
Glucobay 50 mg tablettá 120x	Fogyasztói ár: 3312 Ft.	Normatív támogatás: 1822 Ft.	Normatív térítési díj: 1490 Ft.
Glucobay 50 mg tablettá 30x	Fogyasztói ár: 965 Ft.	Normatív támogatás: 531 Ft.	Normatív térítési díj: 434 Ft.


CV Protector

VÉRNYOMÁSCSÖKKENTŐ, BIZONYÍTOTT CV RIZIKÓCSÖKKENTŐ HATÁSSAL

30 retard filmtabletta 28 x
60 retard filmtabletta 28 x

Hatóanyag: Nifedipin. **Javallat:** Szívkezozőri megbetegedés, krónikus stabil angina pectoris (adagolás: 30-60 mg, naponta 1 x), magasvérnyomás kezelése (adagolás: 1 x 30-60 mg naponta, maximum 120 mg naponta). **Ellenjavallat:** Gy.túlérz., grav., lakt., cardiovascularis shock, nifedipinnel kombináció, kock-poch-vaí elő betegek. **Mellékhatás:** Asthenia, fejfájás, lábszárodema, papilláció, vasodilatáció, GI panaszok, szédülés, mellkasi fájás, fájás, a lábakban, rossz közérzet, szív- és érrendszeri panaszok, vázizom elérések, insomniá, idegesség, paraesthesia, aluszékonyság, vertigo, a légzőrendszer megbetegedése, bőrbetegségek, húgy-ivarszervi betegségek, allergiás reakció, mellcsont alatti mellkasi fájdalom, hidegrázás, oedema az arcon, láz, hipesztézia, tremor, látászavar, a szem rendellenessége, a szem fájdalma, vér- és limfatikus rendszeri elérések. **Kölcsönhatások:** Más vérnyomáscsökkentő szerek, béta-receptor blokkolók, citokrom P450 3A4 rendszer gátló v. serkentő szerek, digoxin, fenitoin, kinidin, quinupristin, dalofopristin, cimetid, nifedipin, diltiazem, grapefrutlé, cisaprid, erythromycin, ketoconazol, itraconazol, flucanazol, tacrolimus, carbamazepin, fenobarbitál, valproát sav. További információkat a részletes alkalmazási előirat tartalmaz. **Alk. előírás OGYI engedély száma:** 13 044/41/2004

Adalat GITS 30 retard filmtabletta 28x	Fogy. ár: 2562 Ft.	Normatív támogatás: 626 Ft.	Normatív térítési díj: 1734 Ft.	Emelt indikációhoz kötött támogatás (EU 90: 1/b pont) 2306 Ft.	Emelt indikációhoz kötött térítési díj (EU 90: 1/b pont) 256 Ft.
Adalat GITS 60 retard filmtabletta 28x	Fogy. ár: 4145 Ft.	Normatív támogatás: 2167 Ft.	Normatív térítési díj: 1978 Ft.	Emelt indikációhoz kötött támogatás (EU 90: 1/b pont) 3731 Ft.	Emelt indikációhoz kötött térítési díj (EU 90: 1/b pont) 414 Ft.


ASPIRIN PROTECT 100mg, 300mg

EVIDENCIA A CV RIZIKÓCSÖKKENTÉSBN

100 mg béiben oldódó filmtabletta 20 x 50 x
300 mg béiben oldódó filmtabletta 20 x 50 x

Hatóanyag: Acetylsalicylic acid. **Javallat:** Thrombocytaaggregáció gátlása céljából: instabil angina pectoris (100-300 mg/nap), AMI (100 mg/nap (Az első tbl-t a gyors felszívódás érdekében szét kell rágni)), reinfarktus profilaxis (100 mg/nap), artériás érsebészeti beavatkozások után, TIA és cerebális infarktus megelőzésére (100-300 mg/nap). **Ellenjavallat:** Gy. túlérz. (acetylsalicylic acid, szalicilátok), Haemorrhagiás diathesis, gyomor- és nyombélfekély, szívegtelenség. Grav ill. trim. Gy.-nél fennálló vírusfertőzés esetén. **Mellékhatások:** Emelygés, hányinger, hasmenés, hányás; gastrointestinalális mikrovérzések (kivételes esetben anaemiához vezethetnek), látási zavarok, tinntus; sav-, bázisháztartás zavarai, Na- és vízretenció (oedema), hypoglikémia. **Kölcsönhatások:** Antikoagulánsok, NSAID, antiheumátikumok, orális antiidiabetikumok, metotrexát, litium, barbiturát, digoxin, szulfonamidok és kombinációk, valproát, trijodotironin, alkohol, kortikoszteroidok. Adoszteron antagonisták, kardiuretikumok, antihypertensív szerek, húgysavcsökkentők (probenecid, sulfipyrazon), antacidumok. További információkat a részletes alkalmazási előirat tartalmaz. **Alk. előírás OGYI engedély száma:** 14955/41/2004

Aspirin Protect 100 mg béiben oldódó filmtabletta 50x	Fogy. ár: 1025 Ft.	Emelt indikációhoz kötött támogatás (EU 50: 2 pont): 513 Ft.	Emelt indikációhoz kötött térítési díj (EU 50: 2 pont): 512 Ft.
Aspirin Protect 100 mg béiben oldódó filmtabletta 20x	Fogy. ár: 432 Ft.	Emelt indikációhoz kötött támogatás (EU 50: 2 pont): 216 Ft.	Emelt indikációhoz kötött térítési díj (EU 50: 2 pont): 216 Ft.
Aspirin Protect 300 mg béiben oldódó filmtabletta 50x	Fogy. ár: 1109 Ft.	Emelt indikációhoz kötött támogatás (EU 50: 2 pont): 555 Ft.	Emelt indikációhoz kötött térítési díj (EU 50: 2 pont): 554 Ft.
Aspirin Protect 300 mg béiben oldódó filmtabletta 20x	Fogy. ár: 536 Ft.	Emelt indikációhoz kötött támogatás (EU 50: 2 pont): 298 Ft.	Emelt indikációhoz kötött térítési díj (EU 50: 2 pont): 298 Ft.

A mindenkor aktuális árakért és támogatásokért kérjük keresse fel a www.oep.hu weboldalt.

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Scientific Programme

20th April, 2007. (Friday)

18.0 Welcome reception

19.0 Opening

Honorary lecture

Chair: László Halmy

19.00 – 20.00 *Jean-Pierre Després, Ph.D., FAHA (Dir. Research Québec Heart Ins., Hôpital Laval Research Centre, Canada):*
Visceral obesity, metabolic syndrome and global cardiometabolic risk: implications for assessment and management

21st April, 2007. (Saturday)

Diagnostic methods

Chair: Éva Martos

9.00 - 9.30 *David L. Ergun, Ph.D. (Vice President Global Technology – Lunar GE Healthcare, US):*
New Perspectives on Body Composition with DXA

9.30 – 10.00 *László Halmy (IRM Central Hosp., Budapest):*
Multiphase bioimpedance in diagnosis of visceral obesity

10.00 – 10.30 *Lajos Lőcsey, Béla Borbás, Beatrix Szlanka, István Ménes, Anikó Dán, László Szabó, László Asztalos, István Lőrincz (Kenézy Hosp .I. Dept, I. Med. Dept. DEOEC Univ. Debrecen , Hungary):*
Connection between visceral fat surface and cardiovascular complications in chronic renal patients

10.30 – 10.45 COFFEEBREAK

Co-morbidities I.

Chair: György Paragh

10.45 – 11. 15 *Tamás Forster (2 Dept. SZOTE Univ., Szeged):*
Abdominal obesity and cardiac diseases

11.15 – 11.45 *Aladár Rónaszéki (Péterfy Hosp., Budapest):*
The role of obesity in development of congestive heart failure

11.45 – 12.15 *Sándor Alföldi (SE Univ., Budapest):*
Treatment of hypertension in metabolic syndrome: the PPAR-gamma activation in focus

12.15 – 12.45 *Péter Kempler (1st Dept. SE Univ., Budapest):*
Postprandial hyperglycaemia and cardiovascular risk in IGT and diabetes mellitus – Is there a need for early intervention?

12.45 – 13.15 *István Barna (SE Univ., Budapest)*
Novel antihypertensive therapy

13.15 – 14.15 LUNCH – POSTER AND SPONSORS VISIT
Isaeva M.V.¹, Shutov A.M.¹, Isaeva I.N.², Gorbunov V.I.²
(¹Dept Med and ²Dept of Public Health of State University, Ulyanovsk, Russia):
Obesity, kidney dysfunction and severity of asthma

László Halmy¹, Csaba Halmy² (IRM Central Hosp., Budapest¹, Central Military Hosp., Budapest²):
275 kg weight reduction by successful treatment of a female patient weighing 400 kg - Herbalife combined dietary treatment in morbid obesity

Pados Gy, Sztanek F, Koncsos P*, Audikovszky M, Varga É*, Seres I*, Paragh Gy**
 St. Imre Hospital, Budapest, *First Department of Medicine, Medical and Health Science Center, University of Debrecen, Hungary
Effect of low-fat and low-carbohydrate diets on weight LOSS and metabolic parameters

Co-morbidities II.
 Chair: József Pucso

14.15 – 14.45 *László Simon (Tolna M. Teaching Hosp., Szekszárd):*
The GERD in obesity

14.45 – 15.15 *György Paragh (DOTE Univ., Debrecen):*
Lipid-metabolism disorders in abdominal obesity

15.15 – 15.45 *István Kiss (Szent Imre Hosp., Budapest):*
Obesity and renal diseases

15.45 – 16.05 *Katalin Várdi Visy (SE Univ., Budapest):*
Sleep apnoea – Pulmonologist's aspects

16.05 – 16.25 *László Halmy (IRM Central Hosp., Budapest):*
Sleep apnoea in obesity

16.25 – 16.45 COFFEEBREAK

Therapy
 Chair: Antal Czinner

16.45 – 17.15 *János Bende, G. Medgyessy, M. Csiszar, M. Ursu (Dept of Surgery, Peterfy Teaching Hospital, Budapest):*
Treatment of extreme obesity with Laparoscopic Adjustable Gastric Band

17.15 – 17.35 *Eszter Halmy¹, László Halmy² (Hungarian Association for Overweight and Obese¹, IRM Central Hosp., Budapest²):*
Effects of body weight management on risk factors in visceral obesity – The role of step counter in brisk walking

17.35 – 17.50 *Beatrix Sárman (SE Univ., Budapest):*
Treatment of impaired glucose tolerance and diabetes in obesity

17.50 – 18.05 *Gabriella Koroknai (Univ. of Pécs Faculty of Health Sciences Institute of Physiotherapy), Brigitta Hüse (Univ. of Pécs Faculty of Health Sciences Institute of Physiotherapy), László Bajnok (Univ. of Pécs Medical School 1.st Department of Internal Medicine), László Pótó (Univ. of Pécs Faculty of Medicine Institute of Bioanalysis):*
Effects of aerobic and resistance exercises on untrained, obese women

18.05 – 18.20 *N. Lasztity, P. Sebestyén, A. Czinner (Dept. of Pediatrics, Heim Pal Children's Hospital, Budapest):*
Risk factors for coronary heart disease and physical activity in obese children

18.20 – 18.35 *Ágnes Gajdács, László Bólya (MediFat[®] Consulting Institute, Budapest):*
The importance of the determination of the visceral fat area in the perioperative risk stratification

18.35 – 18.45 *László Gyula Halmy¹, László Halmy² (Platon Health Ltd¹, IRM Central Hosp., Budapest²):*
The negative correlation of serum triglyceride and BMI and the positive correlation of CRP and BMI in morbid obesity

19.00 DEPARTURE FOR DINNER BY BUSES

VISCERAL OBESITY, METABOLIC SYNDROME AND GLOBAL CARDIOMETABOLIC RISK: IMPLICATIONS FOR ASSESSMENT AND MANAGEMENT

Jean-Pierre Després, Ph.D., FAHA
Director of Research Québec Heart Institute
Hôpital Laval Research Centre

The prevalence of type 2 diabetes is showing a spectacular progression worldwide, a phenomenon largely resulting from the epidemic proportions reached by obesity in various populations of the world. However, physicians have been puzzled by the heterogeneity of obesity as not every obese patient is characterized by chronic complications such as type 2 diabetes, hypertension and coronary heart disease. In this regard, body fat distribution, especially visceral adipose tissue accumulation, has been found to be a key correlate of a cluster of diabetogenic, atherogenic, prothrombotic and inflammatory metabolic abnormalities now often referred to as the metabolic syndrome. This dysmetabolic profile is predictive of a substantially increased risk of coronary heart disease even in the absence of hyperglycemia, elevated LDL-cholesterol or hypertension. For instance, some features of the metabolic syndrome (hyperinsulinemia, elevated apolipoprotein B, and small LDL particles; the so-called atherogenic metabolic triad) have been associated with more than a 20-fold increase in the risk of ischemic heart disease in middle-aged men of the Québec Cardiovascular Study. From a risk assessment standpoint, we have reported that the "hypertriglyceridemic waist" phenotype (waist circumference ≥ 90 cm combined with triglycerides ≥ 2.0 mmol/L) was associated with a high likelihood (80%) of finding this cluster of metabolic abnormalities resulting from abdominal obesity. Consensus groups emphasize the importance of measuring waist circumference in addition to simple parameters such as fasting triglycerides in the identification of patients likely to have the metabolic syndrome in clinical practice. It is therefore suggested that the hyperglycemic state of type 2 diabetic patients may only represent the tip of a huge dysmetabolic iceberg largely explained by the high prevalence of abdominal obesity in our population. As waist circumference is a useful index of abdominal obesity and of related metabolic complications, it is proposed that waist circumference should be systematically measured in all patients and this information taken into account to optimally assess global cardiovascular disease risk: cardiometabolic risk. Finally, until waist circumference and visceral obesity are identified as additional relevant therapeutic targets, it is proposed that clinicians will not optimally manage global cardiometabolic risk in a large proportion of their patients.

NEW PERSPECTIVES ON BODY COMPOSITION WITH DXA

David L. Ergun, Ph.D.
Vice President Global Technology – Lunar GE Healthcare

Dual-energy x-ray absorptiometry (DXA) is best known for measuring bone mineral density (BMD) for diagnosing skeletal health. The unique capabilities of Lunar's dual-energy imaging technology allow its clinical utility to be extended to accurately measure body composition of the total body or body regions with low x-ray exposure. The three major compartments of the body: fat mass, lean mass, and bone mineral mass, can easily be measured using a single whole-body DXA scan with high precision and short scanning time.

The underlying technology of Lunar DXA enables this breakthrough. An ultra-stable k-edge filter technique along with an energy-sensitive direct-digital Cadmium-Zinc-Telluride (CZT) x-ray detector provides superior measurement precision. Coupled with a multipoint calibration scheme, measurement accuracy is achieved over a wide anthropomorphic range. A transverse narrow-angle fan-beam scanning technique provides accurate determination of bone area and linear measurements without magnification error. Finally, advancements in software have greatly simplified scan acquisition, automated scan analysis and improved assessment capabilities of DXA.

The comprehensive view of body composition provided by DXA makes it an attractive technique for understanding a variety of disease processes (e.g. link between excess abdominal fat and metabolic syndrome) and therapeutic interventions (e.g. body fat redistribution and HIV antiretroviral therapy). DXA can subdivide soft tissue into lean and fat compartments to make total body or regional (e.g., trunk, arms, legs, abdomen) measurement of body composition. Lunar's automated assessment of fat in the android (abdominal) and gynoid (hip) regions is of special interest for the study of risk of diabetes and cardiovascular disease. Optimized x-ray flux and higher image resolution improve tissue differentiation and quantification. Software analysis provides a unique quantitative %fat color image for visually identifying regions of high %fat.

In summary, the unique capabilities of Lunar's dual energy imaging technology enable its clinical utility to be extended to measure body composition.

MULTIPHASE BIOIMPEDANCE IN DIAGNOSIS OF OBESITY

László Halmy

IRM Central Hospital, Budapest

Obesity means the growth of body fat mass and not the increase of body weight because this way muscle mass or edema would also mean obesity. Nevertheless, obesity and its measure are identified not by the quantity of body fat but by the body mass index all over the world. Moreover, the diagnosis of obesity can be set up by numerous anthropometrical and instrumental examinations.

Gold Standard means an underwater weight measurement for fat identification and the DEXA but their accessibility is not significant because of financial reasons. The last mentioned cannot be used in the case of super obese patients. Although CT and MRI both provide useful data of fat tissue distribution, they provide no detailed information of body fat weight or body fat percentage. In medical practice the bioimpedance measurement is the most useful method. Its more simple methods can only be used for screening for the most but the newer appliances give us exact diagnosis and furthermore, they can be used to establish the scale of muscle mass and fluid compartment.

Bioimpedance shows the resistance of living organism against alternating current. With the usage of eight electrodes, segmental measurements can be carried out from five cylinders which mean the body and the four limbs. The frequency domain between 50 and 500 kHz contains 6 different types of frequency. While the bioimpedance measurement with 50 kHz that was used earlier could only function in extracellular space, the recently used multi-frequency measuring machines are also able to measure in intra-cellular compartments.

The different tissues of the body contain electrolytes of different concentration and this way their electrical conductivity, of which the reciprocal is the bioimpedance, is different as well.

This way fat tissue can be separated from the muscle mass and the fluid space. Another option is the measurement of intraabdominal fat area by InBody 720, which is the newer generation of bioimpedance measurement appliances, that provides an exact diagnose of the abdominal-type obesity. As it is known, during a slimming cure the decrease of intraabdominal adipose tissue is more significant than the decrease of the subcutaneous adipose tissue, which means that the establishment of intraabdominal fat tissue decrease provides a valuable data, since the comorbidity, that goes along with obesity mostly arise in obesity of abdominal origin.

A further option is the diagnosis of sarcopenic obesity, in which muscle mass is not significant and even the great amount of fat still doesn't cause such a great increase of body weight that would reach the limit of obesity. The method can be used to compare the hypertrophying effect of physical activity

on muscles and the effect of very low calory diet treatment on body composition. The evaluation of obesity related risk factors by bio-impedance is more accurate, because it is not hindered by lean body mass.

The presentation shows some illustrative cases and the measured data of 260 cases. It seems to be useful to build the data provided by the bioimpedance examinations in the diagnosis of obesity.

CONNECTION BETWEEN VISCERAL FAT SURFACE AND CARDIOVASCULAR COMPLICATIONS IN CHRONIC RENAL PATIENTS

Lajos Locsey (1), Bela Borbás (1), Beatrix Szlanka (1), Istvan Menes (1), Aniko Dan (2), Laszlo Szabo (3), Laszlo Asztalos (3) and Istvan Lorincz (4)
B.Braun Avitum 10.AKD, Kenezy Hosp.I.Dept (1), Central Lab.(2), I.Surg.Dept. (3), I.Med.Dept.DEOEC.Univ.Debrecen, Hungary

After renal transplantation significant increased BMI, FM. In chronic dialysed program we increased the aging, accelerating atherosclerosis and malnourished complications. Aim: relationship between change of body different compartment (fat and water) and cardiovascular complications (blood and pulse pressure, cardiac events, arterial stiffness). We investigated dialysed pts 79 males, 56 females (67,12±7,26 years) and renal transplanted 81 males, 64 females (45,51±11,27 years). Blood pressure and cardiac events measured with CardioTens (Meditech 04), arterial stiffness with arteriograph (TensioClinic-TensioMed). Bioimpedance analysis (BIA) was performed with multifrequential BIA analyser (InBody720). We compared the LAP-lipid accumulation product-waist (cm) x serum triglyceride (mM/l) and waist/hip ratio in malnourished, and obese pts in dialysis programme and after renal transplantation. After transplantation cystatin C, homocysteine concentrations were 2,1 vs. 5,2mg/l and 15,21 vs. 23,78µmol/l. Dialysed pts have hypertriglyceridaemia, in transplanted pts were high hypercholesterinaemia, LDL and ApoB levels. In dialysis programme was 9,87% malnourished pts, with decreased FM (4,9-12,6kg), visceral fat surface area less-26,9±12,8cm² than normal. In obese dialysed pts were 29,87%, increased FM(16,89±6,71 kg), and VFA (62,78-56,45cm²). Transplanted pts increased BMI, in 63,14% FM, VFA (48,67-178,14cm²). LAP was 72,22±13,74 cm x mM/l (<21kg/m² BMI), and 227,15±41,78 cm x mM/l (BMI>30kg/m²). In obese pts was frequently arrhythmia, non dipper hypertension and negative diurnal index, and pulse pressure increased higher (>70 mmHg) in 38 % of dialysis pts, and 24% of transplanted pts. The augmentations index (AIx) increased in dialysed female 60 %, after transplantation only 18% (>10%). The pulse wave velocity (PWV) was higher in dialysed males 25%, after transplantation 10%(>12m/s). Conclusions: In obese pts increased significantly BMI, FM and VFA, with increased LAP and waist/hip ratio increased blood and pulse pressure with cardiac events.

RELATIONSHIP BETWEEN ABDOMINAL OBESITY AND CARDIOVASCULAR DISEASES

Tamás Forster
2nd Department of Medicine and Cardiology Center, University of Szeged, Hungary

Obesity is a well-known, major risk factor for cardiovascular diseases. Obesity and glucose metabolism abnormality are forecasted to be the epidemic of the XXIst century. It is not exactly decided which factor plays the primary role but these two abnormalities in conjunction with hypertension and hyperlipidemia form metabolic syndrome. Recent guidelines declare metabolic syndrome as a major factor in cardiovascular diseases. Leptin may play an outstanding role in the pathophysiology of the disease. Serum leptin (released from adipocytes) level, increases due to receptor insensitivity, results in decreased vascular reactivity causing finally left ventricular hypertrophy. In experimental settings, leptin increased oxidative stress on endothelial cells, and furthermore initiates smooth muscle cell proliferation and migration. The blood level of inflammatory markers and cytokines (such as CRP, IL-6 and TNF- α) is also increased in obese patients. It is known that these factors also play an important role in atherosclerosis, too, and this fact partly gives the clue between obesity and cardiovascular diseases. Body fat content and BMI have a significant relationship with event free survival of patients. In obesity, hypertension, coronary disease, left ventricular hypertrophy and diabetes can lead to heart failure. ECG and echocardiographic abnormalities are typical in obesity although, following weight loss, these changes could be reversible.

In conclusion, obesity is more than an excessive fat storage of the body and its treatment can be considered essential in relation to cardiovascular risk.

THE ROLE OF OBESITY IN THE DEVELOPMENT OF CONGESTIVE HEART FAILURE

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The obesity as a coronary risk factor may provoke ischaemic congestive heart failure (CHF) by facilitating other risk factors and accelerating the atherosclerotic process. In the Framingham Heart Study it was shown that obesity itself (and not just the associated clinical conditions) can lead to CHF, since in this large community-based sample, increased body-mass index was associated with an increased risk of CHF. The pathophysiological consequences of obesity, like left ventricular hypertrophy (LVH), hypervolaemia, hypertension, potentiation of increased sympathetic activation may aggravate the symptoms of CHF and require considerations in therapeutic strategy. Left ventricular anatomical remodeling (LVH) in obese patient is followed by electrical remodeling which predispose for life-threatening arrhythmias and sudden death. Changes in the right heart in obese patients are related to obstructive sleep apnoe and/or obesity hypoventilation syndrome, which produce pulmonary hypertension and right ventricular hypertrophy and dilatation, progressive dysfunction and finally right heart failure. In obese patients the CHF that develops is often biventricular.

Given the high prevalence of obesity in the developed countries, strategies to promote optimal body weight may reduce the population burden of CHF.

TREATMENT OF HYPERTENSION IN METABOLIC SYNDROME: THE PPAR-GAMMA ACTIVATION IN FOCUS

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The metabolic syndrome is characterized by the clustering of insulin resistance, dyslipidemia, and hypertension and is associated with increased risk of cardiovascular disease and type 2 diabetes mellitus. Older antihypertensive agents such as thiazide diuretics and beta-blockers, however, have potentially adverse effects on glucose and lipid metabolism and may even exacerbate the metabolic syndrome and increase risk of type 2 diabetes. Recent clinical trials have suggested that antihypertensive agents that inhibit the renin-angiotensin system and the alpha1-adrenergic receptors may reduce risk for new-onset type 2 diabetes, but only a few of these studies were placebo controlled, and in most cases, the absolute antidiabetic effects were relatively modest. Evidence is accumulating that telmisartan, in addition to blocking the angiotensin II type 1 receptor, activates the peroxisome proliferator-activated receptor (PPAR)-gamma a well-known target for treatment of the metabolic syndrome and diabetes. By contrast, other angiotensin-receptor blockers are largely devoid of activity on PPAR-gamma. Telmisartan is a partial agonist of PPAR-gamma and has a better tolerability without causing the fluid retention and edema associated with full agonists of PPAR-gamma such as the insulin sensitizer antidiabetic glitazones. Recent studies have indicated that in addition to antidiabetic properties, PPAR-gamma activators may also provide protection against atherosclerosis and coronary events.

POSTPRANDIAL HYPERGLYCEMIA AND CARDIOVASCULAR RISK IN IGT AND DIABETES MELLITUS – IS THERE A NEED FOR EARLY INTERVENTION?

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The estimated number of known diabetic patients by now is about 95 million. The number of unknown, unreported cases is similar while the number of patients with IGT (impaired glucose tolerance) is 300 million. According to estimates of the International Diabetes Federation (IDF) the number of patients with diabetes until 2025 will increase by 72 % and will be 324 million. According to the FEND-IDF data the mean prevalence of diabetes is 7.5 % in the EU countries, while the current prevalence in Hungary is 9.7 %. Abdominal obesity increases the risk of developing diabetes while there is a clear relationship between weight gain in adulthood and the risk of Type 2 diabetes mellitus. The prevalence of obesity in the USA increased by 61% between 1991 and 2000. Diabetes is one of the most relevant medical complications of obesity. Epidemiological data indicate that glucose should be considered as a continuous risk factor for cardiovascular mortality. 33 years follow-up data of the Whitehall Study indicate that CHD mortality increased from 4.6 mmol/l postprandial glucose levels. The „clock start ticking” very early: for microvascular complications from the onset of hyperglycemia while for macrovascular complications in the prediabetic phase as well. The risk for the development of diabetes among IGT persons correlates linearly with two hour postprandial glucose levels. Data of large prospective epidemiological studies including the DECODE Study, the Honolulu Heart Study, the Diabetes Intervention Study, the Whitehall Study and the Paris Prospective Study indicate that postprandial hyperglycemia is a stronger predictor for cardiovascular mortality compared to fasting glucose levels. Moreover, postprandial hyperglycemia is the first event in the course of diabetes mellitus.

Acarbose reduces postprandial hyperglycemia and is a first line drug in the therapy of Type 2 diabetes mellitus. According data of the STOP NIDDM Trial acarbose may prevent the development of Type 2 diabetes, among patients with IGT the incidence of new cases was reduced by 36 %. Even more importantly, new cases of hypertension were reduced by 34 %, the risk for the development of any cardiovascular event was reduced by 49 %, while the risk of myocardial infarction was reduced by 91 %. According to the metaanalysis of cardiovascular events among Type 2 diabetic patients treated with acarbose, the risk of any cardiovascular events was reduced by 35 %, while the risk of myocardial infarction was reduced by 34 %. Beside diminishing postprandial hyperglycemia, acarbose reduces LDL oxidation, reduces serum cholesterol, triglyceride, LDL, VLDL and chylomicron levels, CRP levels, just as postprandial pro-inflammatory NFκB activity and improves endothelial dysfunction. All these beneficial properties contributes to the vasoprotective effect of acarbose.

NOVEL TREATMENT OF HYPERTENSIVE PATIENTS

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The use correct blood pressure measurement (,) with validated device is important correct diagnosis and treatment. Hypertension Syndrome - It's More Than Just High Blood Pressure, treat endothelial and neurohumoral dysfunction, abnormal glucose and lipid metabolism too. Diabetes, obesity, smoking and age are strongest cardiovascular risk and causes of target organ damages. We have to prevent with optimal antihypertensive treatment the heart, kidney and cerebral dysfunctions (left ventricular hypertrophy, end stage renal failure, and stroke)..

The initiation of treatment blood pressure and risk factors, treatment we cardiovascular diseases. The optimal antihypertensive treatments are safe and have few side effects.

The between modern therapies (CCBs, ACE-inhibitors, ARBs) versus older (beta-blockers, diuretics) in new cases were more then 20%. The potential mechanisms of beta-blocker-induced weight gain are the metabolic rate and postprandial thermo genesis, lipolysis, and increas insulin resistance.

ARB treatment can reduce microalbuminuria and this is the best clinical sign of improvement of endothelial dysfunction (PRIME, RENAL -studies). ARBs are optimal in heart failure, too (VALUE). ARBs increase more 4 times the insulin sensitivity. muscle blood flow sympathetic activity, ionic changes (K⁺, Mg⁺⁺). They act on adipose tissue (FFA, adiponectin, adipogenesis). The effect on insulin cascade signalling and GLUT4 glucose transporters and partial PPAR-γ activity (PPAR-γ modulation: telmisartan, irbesartan, EXP-3174) well known.

The most important message: for the optimal treatment we have to win the compliance of the patients, and treat them with the less side effects.

OBESITY AND GASTRO-ESOPHAGEAL REFLUX DISEASE

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Introduction. Several factors – including changing dietary habits and increasing body weight - can be held responsible for the worldwide rising of incidence and prevalence of gastro-esophageal reflux disease (GERD). Association between obesity and GERD symptoms has been reported, however it is not known whether obesity is an independent risk factor or not.

Epidemiology. Several studies examined the association of body mass index (BMI) with GERD symptoms. Most of them found statistically significant associations of BMI with erosive esophagitis, and esophageal/gastric cardia adenocarcinoma. On the other hand, an US National Examination Survey proved that overweight, but not high dietary fat intake increases risk of gastroesophageal reflux disease hospitalization.

Pathogenesis. The mechanism by which obesity increases the risk of GERD is unknown. Obesity may affect the development of GERD by:

- **Dietary intake:** Some important observations suggest that the positive association between obesity and GERD symptoms remained unchanged after adjustment for various dietary factors.
- **Mechanical factors:** abdominal obesity leads to an increase in intragastric pressure, increased frequency of transient lower esophageal sphincter (LES) relaxations, and/or formation of hiatal hernia.
- **Changes in gastric motility:** The significance of these GI motility alterations in obesity is not fully understood, but they have been considered as potential contributing factors in the development and maintenance of obesity and changed eating behavior.
- **Humoral factors:** as insulin, leptin. Ghreline and growth factors or estrogen may affect risk for GERD.

Clinical relations and treatment. Several reasons support to extend clinical studies for investigations of positive association of obesity and GERD. From among them,

- Possible relations between *Helicobacter pylori* infection, obesity and reflux disease,
- Differences of clinical symptoms and extraesophageal manifestation's appearance of GERD and obesity,
- Priority of therapeutic modalities of obesity or GERD

may have special importance.

Actually, individually determined, adequate proton pump inhibitor (PPI) drugs can be effectively applied in relieving GERD symptoms, but correct decreasing of BMI must not be neglected.

Conclusion. Overweight and obesity are strong independent risk factor of GERD symptoms and esophageal erosions, possibly development of esophageal adenocarcinoma. The prevalence of these disorders in obesity seems to

progressively increase with increasing body weight, and therefore more effective health education and social preventive activity regarding this field seems to be mandatory.

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LIPID METABOLISM DISORDERS IN ABDOMINAL OBESITY

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Obesity is a major cause of cardiovascular morbidity and mortality, and it is frequently associated with cardiovascular risk factors. One of these is hyperlipidemia. The characteristic lipid abnormalities are hypertriglyceridemia, low HDL and increased small dense LDL levels. What is the biochemical background for these alterations? Obesity is commonly accompanied by insulin resistency. The insulin resistency reduces the glucose uptake of the liver and skeletal muscle cells, which leads to hyperglycemia. The high glucose level stimulates the pancreatic beta cell insulin secretion, consequently resulting in hyperinsulinemia. Reduced glucose uptake of adipocytes increases lipolysis in these cells leading to the production of high free fatty acid levels in the sera. Hyperglycemia, hyperinsulinemia, and high levels of free fatty acids promote hepatocyte VLDL production. VLDL is the marker of endogenous lipid production. This particle contains high amounts of triglycerides and cholesterol. Overproduction of this particle leads to hypertriglyceridemia and hypercholesterolemia. VLDL-triglyceride is metabolised by the lipoprotein lipase. In visceral obesity, lipoprotein lipase activity is decreased and these abnormalities also contribute to hypertriglyceridemia. High triglyceride levels also modify other lipoprotein particles, like HDL, IDL and LDL. The cholesterol ester transfer protein activity of the triglyceride-rich HDL is increased, and results in a reduced cholesterol ester content of HDL. The HDL that is rich in triglycerides and poor in cholesterol ester becomes smaller, thereby enhancing its metabolism in the liver via the kidney cubulin and megalin receptors. The cleavage of triglyceride-rich particles decreases the number of those compounds that make up the HDL. These metabolic alterations lead to low levels of HDL. In visceral obesity, triglyceride-rich food intake promotes postprandial hypertriglyceridemia. In this case, the overproduction of endogenous triglyceride-rich VLDL particles exhaust the lipoprotein lipase activity that has already been decreased. The triglyceride-rich VLDL and IDL are metabolised to small dense LDL.

In all, the metabolic changes in visceral obesity increase triglyceride and reduce HDL, increase small dense LDL levels and postprandial hypertriglyceridemia being present. These lipid abnormalities are characteristic for visceral obesity. To reduce the cardiovascular morbidity and mortality of visceral obesity, we need to selectively decrease the amount of the visceral fatty tissue.

OBESITY AND CHRONIC KIDNEY DISEASE

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Obesity is one of the most common disorders in the world and has reached epidemic prevalence. We have growing number of evidences suggesting that obesity initiates a cascade of disorders including hypertension, diabetes, atherosclerosis and chronic renal disease. The 65-75% risk of essential hypertension is connected to excess weight gain and approximately 90% of them have type II diabetes, which is closely linked to excess weight gain. Recent studies suggest that it is also a major risk factor for end-stage renal disease through diabetes and hypertension.

Obesity-induced renal injury are not fully understood at the moment but is likely to involve a combination of hemodynamic and metabolic abnormalities. Obesity raises blood pressure by increasing renal tubular reabsorption, impairing pressure natriuresis, causes volume expansion due to activation of the sympathetic nervous system and renin-angiotensin system. In chronic obesity there may be a loss of nephron function too. Obesity increases the risk for proteinuria. Patients with obesity-associated proteinuria show focal segmental glomerulosclerosis and glomerulomegaly in their biopsy. The pathophysiology of obesity-associated proteinuria is unclear but may include hyperfiltration, increased renal venous pressure, glomerular hypertrophy, hyperlipidemia and increased synthesis of vasoactive and fibrogenic substances, including angiotensin II, insulin, leptin, prostaglandin I-2alpha, tumor necrosis factor-alpha and transforming growth factor-β1, all of which are adipocyte-derived factors.

Visceral obesity leads to the compression of the kidneys and to increased intrarenal pressures. Several mechanisms suggests to mediate sympathetic nervous system activation in obesity, including hyperinsulinemia, increased fatty acids, angiotensin II, increased central chemoreceptor sensitivity, impaired baroreflex sensitivity and hyperleptinemia.

The hypertension and metabolic abnormalities associated with obesity contribute to the development of chronic renal disease and a synergistic relationship may exist between the metabolic abnormalities and the increased glomerular pressure in causing chronic renal vascular disease and nephron loss. Severe obesity may enhance the progression to end-stage renal disease of preexisting nephropathies such as IgA nephropathy.

Several factors associated with greater cardiovascular mortality in the general population show a paradoxical relationship in patients on dialysis therapy. This dialysis-risk paradox is reported for high blood pressure, serum lipid levels, and body mass, but is more consistent for obesity.

Higher body mass index and hypertension independently increase the long-term risk of renal-cell cancer.

Obesity is not only a cardiovascular risk factor but is a renal risk factor, too. The special consideration for obese patients, in addition to adequately controlling the blood pressure, include correction of the metabolic abnormalities and protection of the kidney from further injury. The obese patients need more aggressive non-pharmacological and pharmacological therapy for decreasing body weight and cardiovascular-renal risk reduction. Significant weight reduction is believed to be the most important therapeutic approach to the management of this condition.

OBSTRUCTIVE SLEEP APNOEA SYNDROME AS SEEN BY A CHEST PHYSICIAN

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Obstructive sleep apnoea syndrome is characterized episodic repetitive desaturation and arousals. It's main symptoms are antisocial snoring and excessive daytime sleepiness (EDS).

The condition was first observed in the XIXth century. In early times a close link between obesity day time somnolence and snoring was supposed.

After a debate in this issue the syndrome was called as Pickwickian syndrome after Dickens famous novel. The syndrome in itself was described by Jung and Gastaut in 1965. Our modern view on sleep related breathing disorders is arising the glourishig computer technology which in the last two decades gave us the opportunity of understanding the neurophysiologic mechanism of sleep and respiratory drive as well as the diagnosis, treatment and thorough research of al aspects of sleep and the sleep related breathing disorders (SRBD)

OSA is classified as intrinsic sleep disorder by the international coding of sleep disorders (ICSD), which is prominent and severe form of SRBD. Other sleep related breathing disorders are central sleep apnoea/hypopnoea syndrome, alveolar ventilation, antisocial snoring, upper airway resistance syndrome and nocturnal form asthma.

The main diagnostic criteria of sever OSA is a cessation or decrease of ventilation of more than 30/h. This is called apnoea/hypopnoea index. S a result of this desaturation over 3% is a typical finding. On the neurophysiology side parallel to change in breathing pattern and/r snoring arousals are seen on the EEG. This is to happen as a result of the muscle tone change in the upper airways which is finally narrows or closes the pharynx at any level. As a consequence of this process regular change in sleep and awake state arises causing stress and extra sympathetic activity. On the other hand there is a major decrease in the intrathoracic pressure due to the closure of the upper airways and the unsynchronise respiratory muscle effort. These two processes ends up in endothelial damage which increasing vascular risk factors. In group of patients there is a higher rate of acute myocardial infarct and stroke. In 2005 OSA was classified as major risk factor of stoke.

The repetitive split in sleep and awake state and the enormous negative intrathoracic pressure in the beginning f the disease causing non-diper type blood pressure profile which ends up in hypertension. Lately the AHA named OSA as most widely spread form of secundaer form of hypertension. Again the intrathoracic pressure change can be either the origin or aggravate the injury of the lower esophageal sphincter and gastroesophageal reflux disease.

The most frightening socioeconomic is EDS. Daytime hypersomnolence is leading cause of car accidents among both professional and non-professional drivers.

SLEEP APNOE IN OBESITY

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Some degree of OSA is common finding in 5 – 20 % of adult men although only about one in five of these individuals will have associated daytime sleepiness (OSAS). The main cause the upper body obesity especially a large neck circumference although subtle abnormalities in craniofacial anatomy that also narrow the pharynx are important. OSA has an estimated prevalence of 0.3 – 4 % depending on population studied and criteria used. BMI significantly independently correlates with apnoea/hypopnoea index but after controlling for BMI and age, waist circumference correlated more closely with AHI than neck circumference among males while the opposite is true among females.

Obesity is the main risk factor for developing OSA and 40% to 90% of obese individuals are affected. OSA patients have greater serum levels of leptin. Total sleep deprivation decreases diurnal rhythm of leptin. Short sleep is associated with high ghrelin level which hormone increases the appetite. Sleep loss is a risk factor for developing insulin resistance and type-2 diabetes. These consequences are very common in OSA patients. Obesity is associated with short sleep duration and short sleep duration is a risk factor for diabetes and heart diseases. In persons sleeping less than 8 hours increased BMI was proportional to decreased sleep. In animals deletion of ob gene and/or gene product contributes to obesity and disrupts the temporal organisation of sleep.

Morbidly obese men are at extremely high risk for sleep apnoea (71-90 %). OSA patients after dietary treatment had good weight maintenance only in 3 % after 5 years. Those patients who had bariatric surgery had better results. The continuous positive airway pressure (CPAP) has good effect in the treatment of morbid obesity and together with the bariatric surgery.

In western societies where chronic sleep restriction is common changes in appetite regulatory hormones with sleep curtailment may contribute to obesity.

TREATMENT OF EXTREME OBESITY WITH LAPAROSCOPIC ADJUSTABLE GASTRIC BAND

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Extreme obesity has become a national health issue in Hungary, since most of the health expenses are spent to treat diseases connected to extreme obesity. Without normalizing the patient's body weight these diseases can only be treated, but not cured. The authors' opinion is that body weight of patients above the a BMI of 40 can only be reduced by surgical methods. In their presentation the authors share their experiences of the surgeries of 750 such patients. The analyze the experiences of the past 8 years, the results of the follow up with the aim of proving the surgical methods to be a necessary accessory of gastroenterological treatment. The number of operations showed an exponential growth rate: 2001:17 pat. 2002: 40 pat. 2003: 51 pat. 2004: 94 pat. 2005: 196 pat. 2006: 323 pat.

No patients have been lost in the post-operative phase. There were altogether 491 female and 259 male patients. The ages varied between 16-62 with a mean of 39. The BMI values varied between 40-91 kg/m² with a mean of 52 kg/m². The highest body weight of a patient was 267 kg-s. 457 (61%) suffered from Hypertension, 308 (41%) of Type II Diabetes, 270 (36%) sleep apnea, 426 (57%) GERD, 727 (97%!) psychological disorders, mainly self-esteem problems, 62 young female patients had raro- or amenorrhea or infertility.

Results:

If the patient lost 55% of the Excess Wight (EWL) there is a 90% probability to cure from Type II Diabetes. After 50% EWL the Hypertension normalized in 65% of the patients. After 40 % EWL the sleep apnea was cured. After the normalization of the weight loss 22 children were born from earlier infertile women. (2 twin births and 4 female patients are currently pregnant)

The patients appear for control on a monthly basis at first, later – depending on the weight loss or certain symptoms – in every 2 or 3 months. Usually the doctors used 2 ml of fluid to tighten the band every time an adjustment was due. In average 3-4 tightening sessions were enough to reach a satisfactory result or intake restriction. During a control session there is a swallow test (X-ray), lab test and a satisfaction survey is filled out by the patient.

Due to side effects (penetration into stomach, psychological intolerance, early or late achalasia) the authors had to remove the band in 10 cases. In 5 cases re-operation was needed due to slippage, in each case the problem was solved laparoscopically without removing the band. Band breakage occurred in 2 cases, correction was re-implantation.

We believe that the adjustable gastric banding is a very effective, low risk procedure to successfully treat and cure extremely obese patients. The costs of

the surgery and the band return in a few months. The patients have a chance to live a better life, and have a much higher life expectancy.

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EFFECTS OF BODY WEIGHT MANAGEMENT ON RISK FACTORS IN VISCERAL OBESITY – THE ROLE OF STEP COUNTER IN BRISK WALKING

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Objectives: Our aim was to determine the effect of a new complex treatment on the change of body fat, visceral fat area (VFA) and risk factors and to study the usefulness of step counter in the base of complex treatment.

Subjects: 146 overweight and obese inpatients (male:72, female:74) BMI: 0: 41.59 kg/m² (SD:8.28) (28-69) median:37,3, age: 44.9y (SD:10.64)(17y-72y), time of treatment 0:22.66 (SD: 12.55) median:19.0 days.

Method: After clinical examination daily underwater exercise +gymnastic +brisk walking +diet (800-1500 kcal/day), every day control. Bio-impedance by InBody720 for VFA, anthropometric parameters and risk factors were measured. Walking distance was measured in every case by Omron HJ-113-E.

Results: before and after treatment values 0 (SD)

Body weight (kg)	BMI (kg/m ²)	waist circ (cm)	hip circ (cm)
119.46 (SD:25.88)	41.27 (SD:8.34)	123.36 (SD:18.31)	128.28 (SD:17.15)
112.94 (SD:24.23)	39.06 (SD:7.86)	117.3 (SD:17.69)	124.09 (SD:16.44)

FB%	Fat mass (kg)	VFA (cm ²)	RRsyst (mmHg)
42.57 (SD:7.95)	52.2 (SD:18.23)	177.27 (SD:37.09)	147.86 (SD:19.69)
41.16 (SD:8.39)	47.87 (SD:18.25)	165.45 (SD:49.18)	123.5 (SD:11.93)

RRdiast (mmHg)	glucose (mmol/l)	cholesterol (mmol/l)	LDL - chol (mmol/l)
90.43 (SD:12.67)	6.27 (SD:2.09)	5.32 (SD:1,14)	3.18 (SD:0.96)
76.98 (SD:7.44)	4.96 (SD:1.03)	4.34 (SD:1.06)	2.66 (SD:0.88)

HDL - chol (mmol/l)	triglyceride (mmol/l)	hCRP (mmol/l)
1.32 (SD:0.33)	2.47 (SD:2.77)	8.80 (SD:8.2)
1.17 (SD:0.25)	1.69 (SD:1.14)	4.99 (SD:4.44)

steps/day	walking distance/day	energy expenditure/day
7954 (SD:5697)	4.5 km/day (SD:3.2)	380,81 (SD:251,29)
18571 (SD:8980)	10.2 km/day(SD:5.7)	851,76 (SD:460,69)

Mean values of daily step-count, walking distance, energy expenditure 0 (SD) min-max

daily mean of total steps	walking distance, daily mean	energy expenditure, daily mean
11531 (SD:11731)	6,14 (SD:4.39)	514,14 (SD:366,94)
357 – 118563	0,3 – 28,2	32,6 – 2662,3

Significant difference has been observed between all before and after treatment values.

Conclusions

The newly elaborated complex weight reduction treatment proved to be effective in decreasing body mass, fat mass and VFA and the cardiovascular risk factors. Walking distance was 2,5 times longer after treatment, due to improved fitness. Under-water training provided the possibility for patients with locomotor diseases to participate in the program. Our results persuaded us to promote and suggest our complex weight reduction program as a national program, directed by HSSO and related civil organizations and offices.

TREATMENT OF IMPAIRED GLUCOSE TOLERANCE AND DIABETES IN OBESITY

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The world is facing a rapid increase in the prevalence of obesity and diabetes. Body fat distribution rather than total body fat correlates with the metabolic and cardiovascular complication of being overweight. An accumulation of visceral fat is associated with insulin resistance and other metabolic abnormalities that increase the risk of impaired glucose tolerance, type 2 diabetes and cardiovascular disease. About 70-80 percent of patients with type 2 diabetes are overweight or obese.

In obese diabetic patients therapy is more complex than blood glucose management alone. Therapy involves the prevention of further weight gain, lifestyle modification, combining "diabetic diet" and calorie restrictions and in most cases medical treatment of both diabetes and obesity.

First steps in therapy are life style modifications and weight reduction. Weight loss improves glycemic control and metabolic risk factors including low HDL, high triglyceride and high blood pressure in obese patients with impaired glucose tolerance or diabetes.

Pharmacological management of bodyweight is one of the current challenges in diabetic patients. Numerous anti-diabetic agents (ie, metformin, acarbose, exenatide, pramlintide) used in the treatment of hyperglycemia seem to have beneficial effects on obesity as well. In contrast agents used in the management of obesity (sibutramine, orlistat) appear to improve glycemic control and other metabolic alterations found in diabetes.

Several new therapies to treat obesity may also be advantageous in the management of diabetes. New oral agents may include serotonin reuptake inhibitors, topiramate and rimanobant.

This lecture summarizes current therapies and future therapeutic options in the management of impaired glucose tolerance and type 2 diabetes in obese patients.

EFFECTS OF AEROBIC AND RESISTANCE EXERCISES ON UNTRAINED, OBESE WOMEN

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Introduction: The aim of the present study was to investigate whether preventive and complex exercise therapy combined with dietary consultation affects participants' anthropometric parameters and physical activity.

Methods: We started our six-month exercise program with 20 untrained, obese female patients. 15 of whom (BMI: $32,1 \pm 4,7$ kg/m², mean age $52,5 \pm 4,1$ years) completed the therapy. Eight of them had associated diseases: 7 had hypertension, 1 of them had hypertension and diabetes. The dietary advice given five times during the program contained no specific energy restrictions.

Resting wake-up pulse rate, body weight, BMI, waist and hip circumference, as well as body fat content with monophasic bioimpedance device were monthly controlled. At the beginning and the end of the program patient's blood pressure was measured and their physical fitness was assessed with a 2 km walking test. We also tested the hands' grip strength and the endurance of the abdominal muscles through sit-up test. The mood of the participants was checked on a Visual Analog Scale.

Results: The following parameters significantly decreased at the end of the therapy: body weight decreased from $81,4$ kg \pm $15,7$ to $78,1$ \pm $15,8$, the BMI from $32,1$ kg/m² \pm $4,66$ to $30,90$ \pm $4,72$, body fat from $40,1$ % \pm $5,7$ to $37,9$ \pm $6,6$, waist circumference from $101,7$ cm \pm $11,7$ to $94,9$ \pm $12,1$ and hip circumference from $114,8$ cm \pm $11,7$ to $108,8$ \pm $11,3$ and resting wake up pulse rate from $69,7$ bpm \pm $7,8$ to $66,1$ \pm $7,3$ ($p < 0,01$), while right grip strength from 299 N \pm 50 to 333 \pm 49 , sit-up test from 20 \pm 5 to 27 \pm 4 / 30 sec and calculated VO_{2max} from $7,70$ ml \times min⁻¹ \times kg⁻¹ to $11,94$ significantly increased ($p < 0,01$).

The mood of the participants also considerably improved.

Conclusions: Our results indicate that in obese women aerobic and progressive resistance training can be effectively combined with mild dietary modifications in order to favourably change anthropometric parameters and physical fitness.

RISK FACTORS FOR CORONARY HEART DISEASE AND PHYSICAL ACTIVITY IN OBESE CHILDREN

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There is a rationale for early prevention of cardiovascular diseases (CVD) in children with clustering risk factors. Decreased physical activity seems to play an important role in development of metabolic syndrome and CVD. The aim of this study was to analyze the associations between physical activity and different CVD risk factors in obese children.

Patients and methods: We measured serum lipids (total cholesterol, triglycerides, HDL-cholesterol), apolipoproteins A-I, B, C II and C III (immunturbidimetry, Randox), fasting blood sugar and insulin levels in a cross-sectional study of obese children (n:40, aged:10-18 years). Twenty of them had repeated examinations because of having two additional metabolic risk factors (hypertension, glucose intolerance, fatty liver disease, dyslipidaemia). Body composition was determined by segmental multi-frequency impedance analysis (Inbody 3.0), oral glucose tolerance test and blood pressure monitoring was performed. We determined the physical activity with accelerometer (Actical, Mini Mitter Inc., OR, USA) worn at the hip for 3 days at home.

Results: Alterations of apolipoprotein levels were detected in overall 8/40 obese children, while dyslipidaemia was presented in 15/40. The strongest correlation was found between triglyceride and apo C III levels. Obese children spent $121,5 \pm 54,2$ minute/day with moderate physical activity. Frequency of moderate physical activity showed significant correlation with serum triglyceride and apo B levels ($r:0,41$ and $r:0,75$, $p < 0,05$). There was no correlation with cholesterol and HDL-cholesterol levels. High level of moderate physical activity (>99 minute/day) was associated with a lower incidence of glucose intolerance (5/21 vs 8/11 children, $p < 0,01$) and lower fastin insulin leveles ($8,98 \pm 7,73$ vs $23,57 \pm 18,96$, $p < 0,05$). We found no correlation between physical activity and blood pressure.

Conclusion: The promotion of physical activity has a role in prevention of metabolic alterations in obese children. Measurement of daily activity could be a useful tool in the development of optimal therapeutic strategies and dietary regimes.

THE IMPORTANCE OF THE DETERMINATION OF THE VISCERAL FAT AREA IN THE PERIOPERATIVE RISK STRATIFICATION

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Objective: Due to the epidemic character of obesity the perioperative care of overweight patients is now a routine task of the medicinal care as an interdisciplinary work. The present essay scanned the incidence of the metabolic syndrome in the preoperative period. This was completed by the investigation of the size of the visceral fat area (VFA) besides the analysis of body composition and the state of nourishment and their correlation with the perioperative complications, using an Inbody 720 precision body composition analyser.

Method: Examination of 98 overweight (BMI over 25 kg/m²) patients waiting for an elective general surgery in the period from Jan 1st to Oct 30th, 2006 was completed. Patients were divided into two groups, group A patients (N=35) participated in the MediFat®'s body weight reduction programme, group B patients (N=63) were the control group. 1st examination was made in 8-10 weeks before the surgery and the 2nd examination was made in 1 week before the surgery. The incidence of the complications was assessed 2 weeks after the surgical intervention. During the study the incidence of the metabolic syndrome was stated, all components of the body were weighed (body composition), the distribution of the body fluid compartments, the state of nourishment, the amount of excess weight and the size of VFA were determined with the assistance of a Multifrequency-Bioelectrical Impedance Analyser (DSM-BIA). During the postoperative period the incidence of the general, anaesthesiological and surgical complications was studied.

Result: 32% of all examined patients were overweight, 68% were obese. Incidence of hypertension and dyslipidaemia were over 80%, disorder in the carbohydrate metabolism were existing in 52% of the patients. Incidence of the abdominal type obesity was 78% and in the VFA measurement an average level of 123 cm³ was stated (interval between 88-200 cm³). Occurrence of general perioperative complications has a significant correlation with the size of the VFA and with the age. In group A the VFA reduction due to the reduction of the body weight decreased the incidence of the complications. The incidence of surgical complications were correlated with the amount of the subcutaneous fat (SCF) besides the VFA and the occurrence of diabetes mellitus.

Conclusion: In cases of elective surgeries a longer period of provision may enable the aimed preoperative obesity-related management of the obese patient for the purpose of the avoidance of surgery postponing and for the reduction of the general, surgical and anaesthesiological complications. In our study the size of the VFA was correlated with the incidence of the perioperative complications, so the determination of the VFA is an important tool for the perioperative risk stratification. Reduction of the VFA may affect positively the components of the metabolic syndrome and may reduce the perioperative risk.

THE NEGATIVE CORRELATION OF SERUM TRIGLYCERIDE AND BMI AND THE POSITIVE CORRELATION OF CRP AND BMI IN MORBID OBESITY

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Hypertriglyceridemic effect of obesity is generally acknowledged. Recently increased production of inflammatory factors has been found in obesity, like for instance the increase of hCRP. It is not clear, however, whether there is correlation between serum hCRP and triglyceride levels.

Material and method

The two parameters were examined in obese inpatients before treatment. Patients with endocrine disorders or diabetes mellitus have been excluded. BMI was between 30 and 70 kg/m² (mean: 42.08, SD: 8.87, median: 41.5, n:260). Patients were classified according to WHO BMI groups, level 1: 30-34.9, level 2: 35-39.9 and morbid obesity above 40 kg/m². Body mass, body fat, body fat percent, abdominal fat area and lean body mass values have been measured by multiphase bio-impedance (InBody 720). Serum triglyceride and hCRP were measured by standard clinical method.

Results

Mean serum triglyceride level was higher than normal (1.7mmol/l with the method), it was 2.17 mmol/l. Triglyceride levels are shown in table.

BMI (kg/m ²)	30-34.9	35-39.9	30-39.9	40-
Triglyceride (mmol/l)	2.04±1.57	1.96±1.09	2.00±1.35	2.29±1.54

Fat mass and serum triglyceride showed no correlation in level 1 and 2 obesity. Surprisingly, triglyceride showed negative correlation in morbid obesity ($r = -0.174$; $p = 0.018$; $n = 144$).

While in level 1 and 2 obesity there was no correlation between fat mass and hCRP levels, in morbid obesity they showed positive correlation ($r = 0.461$; $p = 0.0003$; $n = 53$).

Conclusion

The two parameters showed different results in morbid obesity. It is presumable that morbid obesity is not only different from lower levels in quantity, but the disease may have different metabolic pathways and reactions.

OBESITY, KIDNEY DYSFUNCTION AND SEVERITY OF ASTHMA

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Background/Aims. Obesity increases the prevalence and possibly severity of asthma, and also influences asthma control and the response to standard asthma therapeutics. Obesity significantly increased the risk chronic kidney disease. The aim of this study was to investigate the association between body fat mass, kidney dysfunction and severity of asthma.

Methods. 107 patients (65F, 42M, mean age was 48±16 (18-85) years) with asthma were studied. All patients receive therapy concordantly of GINA, 2005. The skinfold thickness at four sites (triceps, biceps, subcapsular, suprailiac) was measured. The BMI for each individual was calculated and percentage of body fat was measured. Glomerular filtration rate was calculated by MDRD formula.

Results. The prevalence of obesity, based on a BMI ≥ 30 kg/m², was 39 (36,4%). BMI was higher in severe persistent asthma, than mild/moderate persistent asthma (29.3±6.0 vs 26.2±5.0 kg/m², resp., p=0.02). Patients with severe asthma have higher fat mass (26.8±10.0 vs 20.7±8.6 kg, resp., p=0.005), but lean mass (51.8±11.3 vs 54.5±10.4 kg, resp., p=0.3) and waist circumference did not differ (104.5±14.1 vs 97.8±14.9 cm, resp., p=0.1). GFR was lower in severe asthma, than mild/moderate asthma (73.6±19.3 vs 87.3±21.3 ml/min/1.73m², resp., p=0.005). The prevalence of GFR < 30 ml/min/1.73m², was 18 (16.8%). GFR was an inverse associated with FM (r = -0.32; p=0.001), but there was positive association with lean mass (r=0.31; p = 0.002). Patients with GFR < 30 ml/min/1.73m² have lower hemoglobin concentrations (13.0±1.5 vs 14.0±1.3 g/dl, resp., p=0.01).

Conclusion. Severity of asthma is associated with increase of fat mass. Increase fat mass and decrease lean mass and hemoglobin concentration is associated with decrease of GFR in patients with asthma. Understanding the relationship between obesity, kidney dysfunction and asthma may lead to new therapeutic strategies for treatment of asthma.

(Poster)

275 KG WEIGHT REDUCTION BY SUCCESSFUL TREATMENT OF A FEMALE PATIENT WEIGHING 400 KG - HERBALIFE COMBINED DIETARY TREATMENT IN MORBID OBESITY

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History

A 41 year old female patient, weighing 400 kg, was transferred to hospital by firemen. The patient was septic, had extended skin necrosis, suffered from NYHA IV. cardiac dysfunction, hepatic dysfunction, acute pyelonephritis, extreme hypothyroidism, ascites, generalized edema, diabetes mellitus, iron deficiency anemia, osteoporosis and depression.

2 years before her admission to hospital she fell in her flat and became immobile because of waist and shoulder pain. Her weight increased by 300 kg in two years. She refused medical treatment. Allergy to pain killers caused extended dermatitis with desquamation, bacterial and fungal superinfection. She couldn't move her extremities due to cardiac failure and muscle weakness. She was lifted out from her second-floor flat by a construction industry machine after removing the window.

The main cause of her co-morbidities was super-obesity. Its treatment seemed to be the major task. A 1000 kcal + fruit diet was started, supplemented with Shapeworks Formula 1 (Herbalife) products and 10 g/day Shapeworks Formula 3 personalized protein concentrate because of hypalbuminemia.

Co-morbidities were treated with anti-inflammatory drugs, antibiotics, thyroid hormones, Ursofalk, anti-depressants, diuretics, spironolactone and panthenol tablets for alopecia.

Reeducation was started when pressure ulcers and skin ulcers healed. Muscle strength improved gradually. She became able to perform minimal movements in bed and later on to sit up with help on the edge of the bed.

During the 15-month treatment body weight decreased by 275 kg, the ascites and the edema disappeared, her hair started to grow, her skin healed completely. She reached a mental balance. Laboratory parameters of cardiac, hepatic, kidney, thyroid gland and erythropoietic functions showed improvement or were normalized. She is facing complete mobilization and she makes plans for the future.

Our following task is the surgical removal of the redundant skin-fat flaps and the abdominal dermolipectomy.

(Poster)

EFFECT OF LOW-FAT AND LOW-CARBOHYDRATE DIETS ON WEIGHT LOSS AND METABOLIC PARAMETERS

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BACKGROUND: According to certain publications diets restricted in carbohydrates with high glycemic indexes exceed the weight reducing and metabolic benefits of low-fat diets.

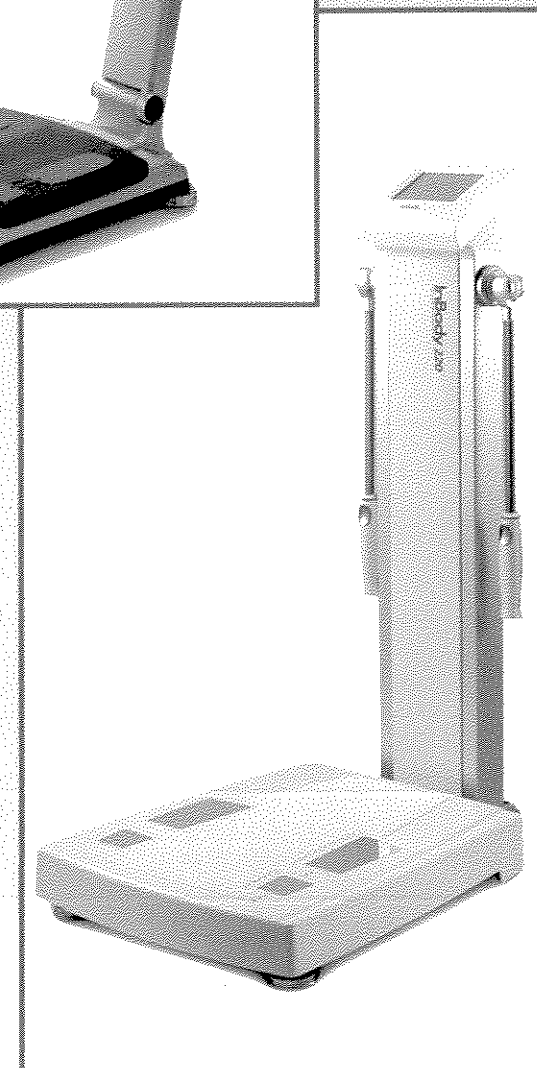
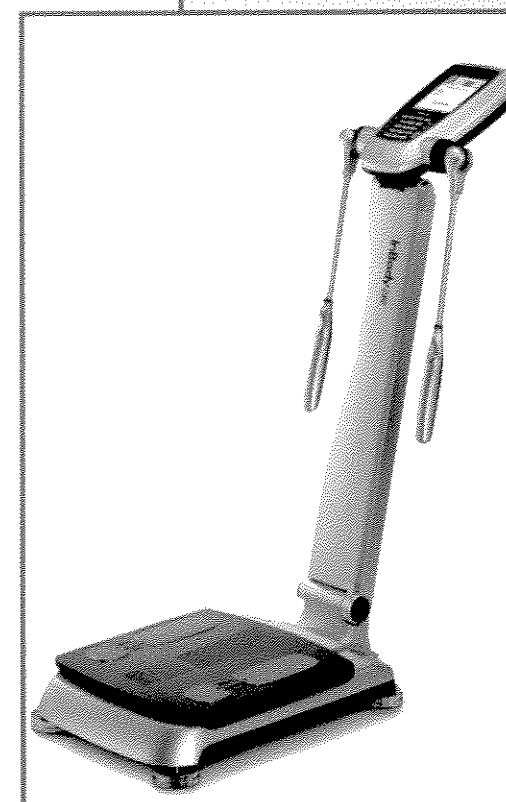
OBJECTIVES: Our aim was to compare the influence of low-carbohydrate and low-fat diets on anthropometric, metabolic parameters and other biochemical markers.

METHODS: We started a non-pharmacological weight-reducing treatment program for obese female patients with metabolic syndrome with a 600 kcal/day hospital treatment, then 91 patients received a 1200 kcal/day low-carbohydrate (low glycemic index) diet, while 30 patients consumed a low-fat diet with similar energy intake for 3 months.

RESULTS: Low-carbohydrate diet resulted in significantly higher ($p=0.02$) weight loss of a mean 8.56 kg (from 102.33 ± 18.64 to 93.77 ± 18.1 kg; -8.4%), than low-fat diet (6.06 kg weight loss, from 97.06 ± 21.48 to 90.6 ± 17.4 kg, -6.6%). Similar changes were observed in the low-carbohydrate group compared to the low-fat one in abdominal circumference (-8.03% vs. -2.69%, $p=0.023$), in BMI (-8.39% vs. -4.33%, $p<0.0001$), in body fat percentage (-7.57% vs. -2.46%, $p=0.013$) as well. Lipid profile improved significantly in the low-carbohydrate group (Chol: -5%, $p=0.0128$, Tg: -23.2%, $p<0.0001$), similarly to carbohydrate metabolism (glucose: -12.58%, $p<0.0001$, HOMA: -37.25%, $p=0.03$). We observed a significant decrease in the inflammatory marker CRP in the low-carbohydrate group ($p<0.05$). Serum leptin produced by adipocytes decreased ($p<0.0001$), as adiponectin increased significantly with weight loss ($p<0.05$). In the low-fat group these parameters (HOMA, CRP, leptin, adiponectin) did not change significantly, and lipid peroxidation elevated substantially ($p=0.016$).

CONCLUSIONS: This study indicated that low-carbohydrate diets are more effective than low-fat ones with the same calorie intake in weight loss, improving anthropometric parameters, glucose and lipid status, and other metabolic markers. (poster)

Az InBody testösszetétel analízátorok pontos értékelést adnak a testet alkotó összetevőkről



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