

1st ANNUAL JOINT CONFERENCE

of the



HELLENIC ASSOCIATION
for the **STUDY** of the
DIABETIC FOOT

and

MEET

MULTIDISCIPLINARY EUROPEAN
ENDOVASCULAR THERAPY



With the
collaboration of:



Hellenic Cardiological Society (HCS)



Hellenic National Center for
the Research, Prevention and
Treatment of Diabetes Mellitus
and its Complications (HNDC)

7-10 July 2011

AKS Hotel
Porto Heli, Greece

**The Conference is accredited with fifteen (15)
Continuous Medical Education credits**

Conference Secretariat:



Final Programme & Abstracts Book



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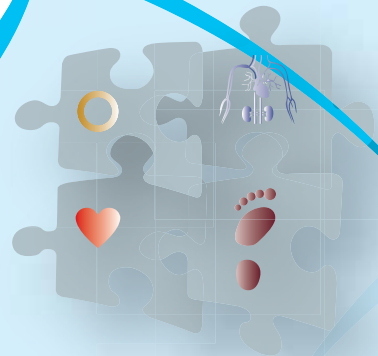
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Invitation



Dear Colleagues,

It is with great pleasure that we invite you to join us to the 1st Annual Joint Conference of the Hellenic Association for the Study of Diabetic Foot Diseases (EMEDIP) and the Multidisciplinary European Endovascular Therapy (MEET). The Meeting will be held in Porto Heli, Peloponnese, Greece, 7-10 July 2011.

This meeting is unique in the way it brings together specialists involved in the care of persons with diabetes, suffering from Diabetic Foot Diseases. This fact creates an environment in which all the aspects and hot topics in the above mentioned field will be presented and analyzed by the eminent speakers.

Since atherosclerotic peripheral vascular disease with the infection is a major factor in the pathology and progression of diabetic foot, special sessions will focus on the diagnosis and treatment of Foot Ischemia of both infected and non infected leg. Apart from the numerous plenary lectures, other review sessions and interactive workshops from renowned scientists have been integrated in the programme and will cover all of the other aspects, from the epidemiology and burden of diabetic foot related problems, to the treatment and rehabilitation.

The vivid exchange of knowledge of all the specialities involved and participating in this Meeting, introducing the concept of the multidisciplinary team approach to the diabetic foot care, will be further established by the discussion of the current status of clinical and basic research, presented orally or in the form of posters by the attendees.

We hope that we will have the opportunity to welcoming you at the excellent seaside resort of Porto-Heli, for the 1st Annual Joint Conference of our Associations and enjoy with you an excellent scientific event.

For the Organizing Committee

Chr. Manes

P. Bergeron

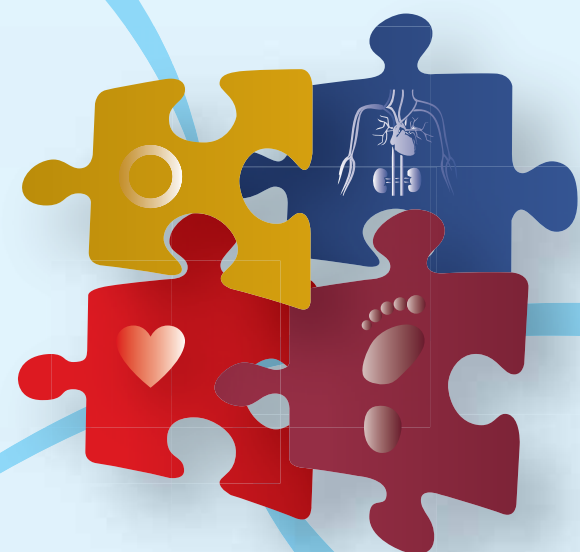
D. Raithel

Organizing Committee

CHAIR:	C. MANES
CO-CHAIRS:	P. BERGERON
	D. RAITHEL
MEMBERS:	K. KALLIGIANNI
	A. KAMARATOS
	T. KATSAROS
	T. MESIMERIS
	A. PAPPAS
	N. TENTOLOURIS

Scientific Committee

V. ANDRIKOPOULOS	N.L. KATSILAMBROS
I. APOSTOLAKIS	D. KISKINIS
P. BALAS	A. KOKKINOS
A. BOULTON	K. KTENIDIS
E. DEMIRI	C. LIAPIS
E. DIAKOUMOPOULOU	S. LIATTIS
A. DIAMANDOPOULOS	K. MAKRILAKIS
A. DIONYSSOPOULOS	G. PARHARIDIS
L. INGLESE	D. PERREA
S. KALLIAFAS	S.A. RAPTIS



Scientific Programme

Scientific Programme

DAY 1

Thursday 7 July, 2011

GRAND BALLROOM II & III

16:00-17:00 Registrations

17:00-18:00 **Workshop I:**
Off loading - dressings

Chair: **C. Loupa**

Speakers: **M. Valsami, L. Thiaspras**

18:00-18:30 **Round Table:**
Epidemiology, pathogenesis and consequences of the diabetic foot

Chair: **P. Balas**

18:00-18:10 Epidemiology of the diabetic foot: **C. Manes**

18:10-18:20 Pathogenesis – socio - economic consequences: **N. Tentolouris**

18:20-18:30 Discussion

18:30-19:00 **Lecture**

Chair: **C. Manes**

The history of crutch through the art: **E. Dounis**

19:00-19:45 **Coffee Break**

19:45-20:30 **Welcome Addresses**

20:30-21:00 **Keynote Lecture**

Chair: **S.A. Raptis**

Mediterranean diet and health: **N.L. Katsilambros**

21:00 **Welcome Reception**

DAY 2

Friday 8 July, 2011

GRAND BALLROOM II & III

09:00-10:00

Workshop II:

Differential diagnosis of ulcers - case reports and open discussion

Chair: **C. Manes**

Speaker: **T. Katsaros**

10:00-11:15

Round Table:

Diabetic cardiovascular disease - multifactorial approach

Chairs: **V.N. Pyrgakis, C. Rokkas**

10:00-10:15

Dyslipidaemia: **K. Makrilakis**

10:15-10:30

Cardiac disease and diabetes: **V.N. Pyrgakis**

10:30-10:45

Surgical treatment of coronary artery disease in diabetic patients:
C. Rokkas

10:45-11:00

Diabetes and thoracic aortic aneurysms: update on endovascular and
hybrid solutions: **D. Raithel**

11:00-11:15

Discussion

11:15-11:45

Coffee Break

11:45-13:15

Round Table:

Infections

Chairs: **A. Giannopoulos, G. Petrikos**

11:45-12:05

Diagnosis - conventional treatment: **D. Plachouras**

12:05-12:25

Radiologic imaging: **A. Bintoudi**

12:25-12:45

Surgical treatment of soft tissue infections: **D. Dimitroulis**

12:45-13:05

Surgical treatment of bone infections: **F. Sayegh**

13:05-13:15

Discussion

13:15-14:00

Satellite Lecture (BMS/AstraZeneca)

Chair: **N. Tentolouris**

The impact of DDP-4 inhibitors in type 2 diabetes treatment in Greece:
K. Makrilakis

14:00-14:30

Satellite Lecture (Roche Diagnostics Hellas S.A.)

Chair: **C. Manes**

Technology at the service of Diabetes Mellitus: **N. Tentolouris**

14:30-16:15

Break

16:15-17:30

Round Table:

Management of the diabetic patient in the hospital

Chairs: **N.L. Katsilambros, C. Manes**

16:15-16:35

Perioperative management: **A. Thanopoulou**

16:35-16:55

The patient in the coronary care unit: **A. Kokkinos**

16:55-17:15

The patient in the intensive care unit: **C. Adamopoulos**

17:15-17:30

Discussion

17:30-17:45

Coffee Break

17:45-19:00

Satellite Symposium (Pharmaserve Lilly)

Aggressive therapeutic treatment when oral antidiabetics combinations fail

Chair: **E. Hatziagelaki**

17:45-18:15

Impact of incretin mimetic based therapy on diabetic vascular disease:
M. Theodorakis

18:15-18:45

Effectiveness of 'analogues' mixes in patients who need insulin therapy:
E. Hatziagelaki

18:45-19:00

Discussion

19:00-19:15

Welcoming from the President and overview of the diabetic arterial disease

C. Manes

19:15-21:00

Round Table:

Diabetic foot patient - multifocal diabetic disease

Chairs: **P. Balas, L. Inglese, I. Tsitouridis**

19:15-19:30

Diabetic foot and coronary artery disease

How should we manage such a patient?: **S. Polymeros**

When to intervene: timing and strategies: **C. Rokkas**

19:30-19:45

Diabetic foot and cerebrovascular disease

How should we manage such a patient?: **D. Raithel**

When to intervene: timing and strategies: **K. Kalligianni**

19:45-20:00

Diabetic foot and renal disease

How should we manage such a patient?: **A. Diamandopoulos**

When to intervene: timing and strategies: **A. Giannoukas**

20:00-20:15

Diabetic foot and aortic disease

How should we manage such a patient: **D. Kiskinis**

When should we operate such a patient?: **P. Bergeron**

20:15-20:30

Indications of operative treatment of arterial disease in the diabetic patient: **C. Liapis**

20:30-21:00

Discussion

09:00-11:00

Oral PresentationsChairs: **A. Kokkinos, E. Papazoglou****OP1****MICROVASCULAR COMPLICATIONS AND RISK FACTORS, COEXISTENCE, THEIR ROLE IN THE PATHOGENESIS AND THE SEVERITY OF DIABETIC FOOT***D. Skoutas, N. Papanas, Z. Mousleh, S. Giandikidis, G. Tsiantas, S. Georga, Th. Mesimeris, N. Papazoglou, M. Lazaridis, Ch. Manes***OP2****PREVENTION AND MANAGEMENT OF THE DIABETIC FOOT - OUR EXPERIENCE FROM OUR NEW DIABETIC FOOT CLINIC***Bristianou M., Panou Ch., Chatzidakis I., Tsigliou V., Mytis G., Lanaras L.***OP3****OSTEOPROTEGERIN LEVELS CORRELATE WITH SEVERITY OF PERIPHERAL ARTERIAL DISEASE AND PERIPHERAL NEUROPATHY IN TYPE 2 DIABETIC PATIENTS***Eleftheriadou Ioanna, Grigoropoulou Pinelopi, Argiana Vasiliki, Balla Ioanna, Chorepsima Stamatina, Kokkinos Alexander, Perrea Despoina, Katsilambros Nicholas, Tentolouris Nicholas***OP4****SERUM OSTEOPROTEGERIN LEVELS CORRELATE WITH SEVERITY OF LOWER EXTREMITY ARTERIAL CALCIFICATION IN PATIENTS WITH TYPE 2 DIABETES***Eleftheriadou Ioanna, Argiana Vasiliki, Grigoropoulou Pinelopi, Balla Ioanna, Kritikos Constantinos, Arapostathi Christina, Kokkinos Alexander, Perrea Despoina, Katsilambros Nicholas, Tentolouris Nicholas***OP5****POPLITEAL ARTERY PERCUTANEOUS TRANSLUMINAL ANGIOPLASTY FOR CHRONIC CRITICAL LIMB ISCHEMIA AND DIABETES MELLITUS LESIONS. ANALYSIS OF A SINGLE-CENTER EXPERIENCE***Argitis V., Kyriakidis K., Tabakis H, Dervisis K.***OP6****PLEIOTROPIC EFFECTS OF ATORVASTATIN ON CENTRAL AND PERIPHERAL ARTERIES OF PATIENTS WITH TYPE 2 DIABETES***P. Grigoropoulou, I. Eleftheriadou, C. Zoupas, V. Argianna, A. Kokkinos, D. Perrea, N. Katsilambros, N. Tentolouris***OP7****BULLOSIS DIABETICORUM: A NOT-SO-RARE CONDITION CHARACTERISTIC OF DIABETES MELLITUS***C. Loupa, E. Voyatzoglou, A. Donou, N. Souliotis, E. Meimeti, D. Voyatzoglou†***OP8****THE ASSOCIATION BETWEEN DIABETIC AUTONOMIC NEUROPATHY AND CUTANEOUS CIRCULATION IN PATIENTS WITH TYPE 2 DIABETES***Eleftheriadou Ioanna, Grigoropoulou Pinelopi, Argiana Vasiliki, Fardi Panagiota, Kalopita Stavroula, Alexiadou Kleopatra, Kokkinos Alexander, Perrea Despoina, Katsilambros Nicholas, Tentolouris Nicholas*

OP9**ASSOCIATION OF ARTERIAL STIFFNESS AND CARDIOVASCULAR RISK FACTORS IN PATIENTS WITH TYPE 2 DIABETES**

*P. Grigoropoulou, I. Eleftheriadou, C. Zoupas, V. Argianna, I. Balla, A. Kokkinos
D. Perrea, N. Katsilambros, N. Tentolouris*

OP10**VALIDATION OF DIFFERENT RESPONSES OF THE SUDOMOTOR FUNCTION TEST (NEUROPAD) IN IDENTIFYING TYPE 2 DIABETIC PATIENTS WITH PERIPHERAL OVERALL NERVE DYSFUNCTION**

A multicenter study

*Manes Christos, Papanas Nikolaos, Exiara Triada, Papantoniou Stefanos,
Kirlaki Evridiki, Tsotoulidis Stefanos, Kefalogiannis Nikolaos,
Maltezos Efstratios*

OP11**DIFFERENT RESPONSES OF A SCREENING TOOL (SUDOMOTOR FUNCTION TEST) IN IDENTIFYING TYPE 2 DIABETIC PATIENTS WITH SMALL FIBER NERVE DYSFUNCTION**

A multicenter study

*Manes Christos, Papanas Nikolaos, Exiara Triada, Papantoniou Stefanos,
Kirlaki Evridiki, Tsotoulidis Stefanos, Kefalogiannis Nikolaos,
Maltezos Efstratios*

16:00-17:30**Oral Presentations**

Chairs: *I. Apostolakis, A. Kamaratos*

OP12**THE ROLE OF HYPERTENSION AND OBESITY IN THE NEUROPATHIC AND MICROANGIOPATHIC COMPLICATIONS OF DIABETES TYPE 2**

*O. Tsachouridou, S. Tsotoulidis, G. Petridis, K. Grivou, S. Papathoma,
S. Sidiropoulou, A. Matis, A. Tsachouridis, D. Karagiannidou, D. Pavlidou*

OP13**MACROVASCULAR COMPLICATIONS AND RISK FACTORS, COEXISTENCE, THEIR ROLE IN THE PATHOGENESIS AND THE SEVERITY OF DIABETIC FOOT**

*D. Skoutas, N. Papanas, Z. Mousleh, S. Giandikidis, K. Siomos, G. Georgiadis,
V. Souftas, N. Papazoglou, M. Lazaridis, Ch Manes*

OP14**STUDY OF DIABETIC NEUROPATHY AND RETINOPATHY IN PATIENTS WITH DIABETES TYPE 2 IN CORRELATION WITH THE DURATION AND EFFECTIVE MANAGEMENT OF THE DISEASE**

*O. Tsachouridou, S. Tsotoulidis, G. Petridis, K. Grivou, S. Sidiropoulou,
S. Papathoma, A. Tsachouridis, A. Sbardos, D. Mikropoulos, A. Matis,
M. Tsetselidou*

OP15**HEREDITY AND DIABETIC NEUROPATHY AND RETINOPATHY IN PATIENTS WITH DIABETES TYPE 2**

*S. Tsotoulidis, G. Petridis, O. Tsachouridou, K. Grivou, A. Tsachouridis,
S. Sidiropoulou, S. Papathoma, D. Mikropoulos, A. Psarra, M. Tzovanaki*

OP16

**NECROTIZING SOFT TISSUE INFECTIONS OF THE DIABETIC FOOT:
AN EMERGENCY SITUATION**

*C. Loupa, M. Skopeliti, D. Chryssis, M. Fatourou, I. Bakas, E. Voyatzoglou,
A. Donou, G. Marathonitis, D. Voyatzoglou[†]*

OP17

**USE OF VACUUM-ASSISTED CLOSURE (VAC) DEVICE IN A DIABETIC
FOOT CLINIC**

C. Loupa, E. Meimeti, A. Donou, E. Voyatzoglou, I. Bakas, D. Voyatzoglou[†]

OP18

**SUCCESSFUL TREATMENT OF SELECTED CASES OF OSTEOMYELITIS
IN THE DIABETIC FOOT WITHOUT SURGICAL BONE REMOVAL**

*C. Loupa, I. Kotsantis, E. Papadakis, E. Voyatzoglou, M. Skopeliti, A. Donou,
K. Papagiannis, E. Koutsantonidou, S. Lafoyanni*

OP19

**REPRODUCIBILITY OF THE LANARKSHIRE OXIMETRY (LOI) INDEX IN
TYPE 2 DIABETES MELLITUS**

*Papanas N., Kakagia D., Tiaka E., Alexandridou M., Pagkalos A., Kyrgiannaki V.,
Maltezos E.*

OP20

**IS TISSUE OXYGENATION A MAJOR DETERMINANT OF ULCER
OUTCOME IN DIABETIC PATIENTS?**

*C. Manes, Th. Mesimeris, Th Melekos, St. Stefanidou, E. Pertsas, G. Tzatzagou,
D. Mpaltzis, B. Kourkoupas*

DAY 3

Saturday 9 July, 2011

GRAND BALLROOM II & III

08:45-09:30

Debate:

Treatment strategies for the diabetic foot

Chairs: **P. Balas, D. Raithel**

08:45-09:00

Efforts for limb conservation: when and how?: **C. Liapis**

09:00-09:15

Virtual Amputation: a new procedure to salvage the diabetic foot:
E. Calabrese

09:15-09:30

The chair opinion for a consensus: **P. Balas, D. Raithel**

09:30-11:00

Round Table:

Conventional treatment of the diabetic foot

Chair: **E. Bastounis**

09:30-09:45

Radical foot amputations: what are the benefits?: **F. Markatis**

09:45-10:00

Distal by-pass procedures can reduce limb loss: **D. Kiskinis**

10:00-10:15

Can drug - therapy, be efficient in the management of the diabetic foot?
angiogenic growth factors, prostaglandins: **S. Georgopoulos**

10:15-10:30

What is the role of lumbar sympathectomy in 2011, for the management
of diabetic foot lesions?: **A. Giannoukas**

10:30-10:45

Neuro - stimulation of the spinal cord: what is its place in the diabetic
patient?: **K. Ktenidis**

10:45-11:00

Discussion

11:00-11:30

Coffee Break

11:30-14:45

Round Table:

Endovascular limb salvage of the diabetic patient

Chairs: **D. Kiskinis, P. Bergeron**

11:30-11:45

Specificities of diabetic arteries: **U. Meister**

11:45-12:00

Anatomic consideration for successful recanalisation, the tibio - peroneal
runoff scoring and the JENALI collateral score: **R. Pini**

12:00-12:15

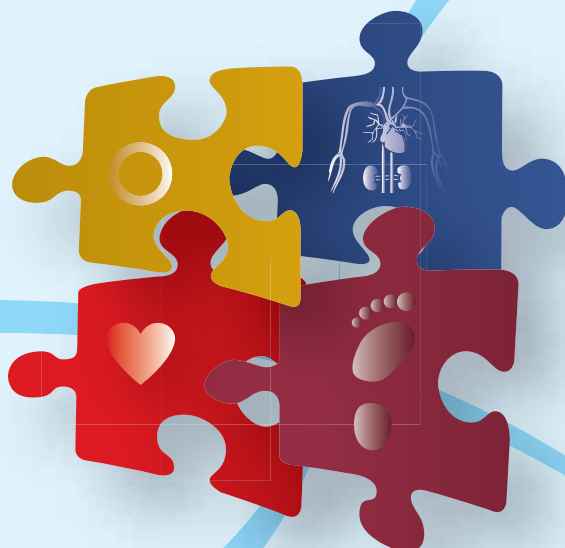
Recanalisation of subtotal occlusions: the role of atherectomy and new
devices (Jetstream): **K. Papazoglou**

12:15-12:30

Recanalisation of chronic total conclusion with subintimal angioplasty:
L. Inglese

12:30-12:45	Alternative collateral way: K. Ktenidis
12:45-13:00	Efficacy of below the knee (BTK) balloon angioplasty, cryoplasty and drug eluting balloons: K. Kalligianni
13:00-13:15	BTK stenting for tibio-peroneal lesions in critical limb ischaemia patients: W. Ritter
13:15-13:30	Treatment strategies of acute limb ischaemia in the diabetic patient. Thrombectomy, aspiration, thrombolysis: S. Kalliafas
13:30-13:45	New medical treatment in the diabetic foot: negative pressure therapy: A. Dionyssopoulos
13:45-14:00	Treatment with hyperbaric O ₂ in the diabetic foot: T. Mesimeris
14:00-14:15	Combined endovascular and surgical procedures. The “hybrid” era: P. Bergeron
14:15-14:45	Discussion
14:45-16:30	Break
16:30-17:30	Round Table: Update in diabetic neuropathy
	Chairs: N. Tentolouris, T. Katsaros
16:30-16:45	Chronic sensorimotor neuropathy: C. Manes
16:45-17:00	Autonomic neuropathy in diabetic foot: N. Papanas
17:00-17:15	Painful diabetic neuropathy: J. Doupis
17:15-17:30	Discussion
17:30-17:45	Coffee Break
17:45-19:00	Round Table: Contemporary approaches in the treatment of diabetes mellitus and its implications
	Chair: C. Manes
17:45-18:15	Ertapenem in the diabetic foot infections: N. Tentolouris
18:15-18:45	Sitagliptin’s position in the therapeutic algorithm: E. Hatziagelaki
18:45-19:00	Discussion
19:00-19:15	Conclusions P. Bergeron, C. Manes
21:00	Gala Dinner

Speakers Index



INTERNATIONAL FACULTY

- Bergeron P.** Dr., French Surgical Academy Member, Head Department of Cardiovascular and Thoracic Surgery, St Joseph Hospital & Foundation, France, Director of Multidisciplinary European Endovascular Therapy
- Calabrese E.** Diplomate of the American Board of Surgery, Director, International Center for Limb Salvage, G.F.M.E.R.: Geneva Foundation for Medical Education and Research - Geneva, Switzerland, Director, National Center for Limb Salvage, Monza Policlinic - Milan, Italy
- Inglese L.** Dr., Professor of Interventional Radiology IRCCS Policlinic S. Donato, Milan, Italy
- Meister U.** Dr., Chief of the Department of Vascular Surgery, Hospital of Neumarkt, Germany
- Pini R.** MD, Vascular Interventional Radiologist, Department of Vascular and Interventional Radiology, S. Giovanni Battista - Molinette Hospital, Turin, Italy
- Raithel D.** Dr., Professor of Vascular Surgery, Nuremberg Hospital, Germany, Honorary Director of Multidisciplinary European Endovascular Therapy
- Ritter W.O.** Leader of the Department of Interventional Radiology Klinikum Nürnberg Süd, Germany

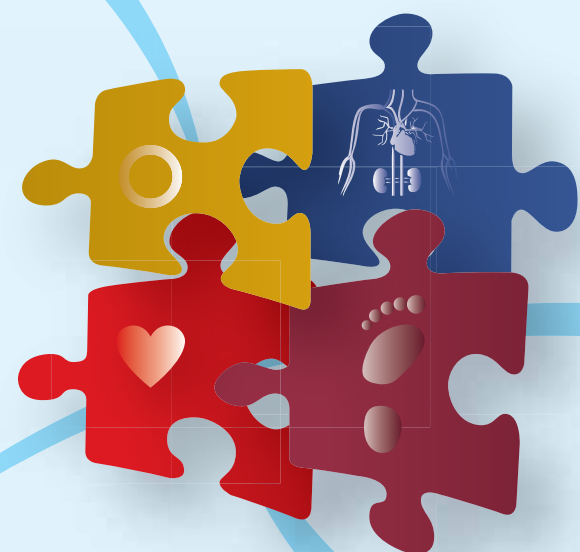
LOCAL FACULTY

- Adamopoulos C.** MD, PhD, Cardiologist, Department of Cardiology, General Hospital "Ag. Pavlos", Thessaloniki
- Apostolakis I.** Physician, Director of 1st Internal Medicine Department, Chief Medical Officer, "Hygeia" Hospital, Athens
- Balas P.** MD, MS (Surg.), FACS (Hon.), Emeritus Professor of Surgery, Athens University Medical School, Athens
- Bastounis E.** Professor Emeritus of Surgery, Athens University Medical School, Vascular Surgeon, Athens
- Bintoudi A.** Consultant Radiologist, MSK Department, "Papageorgiou" Teaching General Hospital, Thessaloniki
- Diamandopoulos A.** Professor of Athens University Medical School, Ex Director of Department of Nephrology, "Ag. Andreas" General Hospital, Patra

Dimitroulis D.	Senior Lecturer in Surgery and Transplantation, 2 nd Department of Propaedeutic Surgery Medicine, Athens University Medical School, “Laiko” General Hospital, Athens
Dionyssopoulos A.	Associate Professor of Plastic Surgery, Faculty of Medicine, Aristotle University of Thessaloniki, Thessaloniki
Dounis E.	MD, F.A.C.S., Orthopaedic Surgeon, Foot and ANKLE Surgery, Athens
Doupis J.	MD, PhD, Director of Internal Medicine and Diabetes Department, Naval Hospital, Salamis Naval Base, Athens
Georgopoulos S.	Assistant Professor of Vascular Surgery, 1 st Department of Vascular Medicine, Athens University Medical School, “Laiko” General Hospital, Athens
Giannopoulos A.	Professor of Surgery, 1 st Department of Surgery, Athens University Medical School, “Laiko” General Hospital, Athens
Giannoukas A.	Professor of Vascular Surgery, Faculty of Medicine, University of Thessalia, Larissa
Hatziagelaki E.	Assistant Professor of Internal Medicine, 2 nd Department of Internal Medicine Propaedeutic - Research Institute and Diabetes Center, Athens University Medical School, University General Hospital “Attikon”, Athens
Kalliafas S.	Vascular - Endovascular Surgeon, Director in the Department of Vascular Surgery, “Hygeia” Hospital, Athens
Kalligianni K.	MD, PhD, Vascular Surgeon, “Hygeia” Hospital, Athens
Kamaratos A.	Assistant Director in the Diabetic Center, “Tzaneio” General Hospital, Piraeus
Katsaros T.	Internist specialized in diabetes, Director in Chief of Endocrinology Department, Athens General Hospital “G. Gennimatas”, Athens
Katsilambros L.N.	Professor of Internal Medicine, M.D., PhD, FACP, University of Athens, Research Laboratory “N.S.Christeas” & “Evgenidion Hospital”, Vice President of the Hellenic National Diabetes Center, Vice President of the Hellenic Society of Internal Medicine, Athens
Kiskinis A.D.	Professor of Vascular Surgery, MD, Director of 1 st Department of Surgical Clinic, Aristotle University of Thessaloniki, General Hospital “Papageorgiou”, Thessaloniki

Kokkinos A.	Lecturer in Internal Medicine, 1 st Department of Propaedeutic and Internal Medicine, Athens University Medical School, “Laiko” General Hospital, Athens
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Loupa C.	Internist, Infectious Diseases Physician, “Amalia Fleming” General Hospital, Athens
Liapis C.	Professor of Vascular Surgery University of Athens, Chairman Department of Vascular Surgery, University General Hospital “Attikon”, Athens
Makrilakis K.	Assistant Professor of Internal Medicine, 1 st Department of Propaedeutic and Internal Medicine, Athens University Medical School, “Laiko” General Hospital, Athens
Manes C.	MD, PhD, Internal Medicine, Chairman of the Diabetic Department of “Papageorgiou” Teaching General Hospital, Thessaloniki, President of the Hellenic Association for the Study of the Diabetic Foot
Markatis F.	Dr., MD, Vascular Surgeon, Clinical Fellow Department Cardio - Thoracique et Gros Vaisseaux, Hospital Saint - Joseph, Marseille, France
Mesimeris T.	Medical Director of Hyperbaric Medicine Department, General Hospital “Ag. Pavlos”, Thessaloniki
Papazoglou E.	General Surgeon, Specialized in Diabetic Foot, Piraeus
Papazoglou K.	Associate Professor of Vascular Surgery, 5 th Department of Vascular Medicine, Aristotle University of Thessaloniki, General Hospital “Hippokration”, Thessaloniki
Papanas N.	Assistant Professor of Internal Medicine, Outpatient Clinic of the Diabetic Foot, 2 nd Department of Internal Medicine, Democritus University of Thrace, University General Hospital of Alexandroupolis, Alexandroupoli
Plachouras D.	Assistant Professor of Internal Medicine - Infectious Diseases, 4 th Department of Internal Medicine, Athens University Medical School, University General Hospital “Attikon”, Athens
Polymeros S.	Dr., “Mitera” Hospital, Ygeia Polis, Consultant Cardiologist, Glasgow, UK

Petrikkos G.	Professor of Internal Medicine - Infectious Diseases, Director of the 4 th Department of Internal Medicine, Athens University Medical School, University General Hospital “Attikon”, Athens
Pyrgakis N.V.	MD, FESC, FACC, Chairman of Cardiology Department, Athens General Hospital “G. Gennimatas”, Former President of the Hellenic Cardiological Society, Athens
Raptis S.A.	Professor M.D., PhD., M.D. (Hon), HMGSIM, HFEFIM, Academician and Senate Member of the European Academy of Sciences and Arts, Internal Medicine, Endocrinology and Gastroenterology, Universities of Athens - Hellas and Ulm - Germany, 2 nd Department of Internal Medicine Propaedeutic - Research Institute and Diabetes Center, Athens University Medical School, University General Hospital “Attikon”, President of the Hellenic National Diabetes Center, Athens
Rokkas C.	Associate Professor of Cardiothoracic Surgery, Director in Chief of Cardiothoracic Surgery Department, Athens University Medical School, University General Hospital “Attikon”, Athens
Sayegh F.	Assistant Professor of Orthopaedics, 3 rd Department of Orthopaedics, Aristotle University of Thessaloniki, General Hospital “Papageorgiou”, Thessaloniki
Tentolouris N.	Assistant Professor of Internal Medicine, 1 st Department of Propaedeutic and Internal Medicine, Athens University Medical School, “Laiko” General Hospital, Athens
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Oral Presentations

MICROVASCULAR COMPLICATIONS AND RISK FACTORS, COEXISTENCE, THEIR ROLE IN THE PATHOGENESIS AND THE SEVERITY OF DIABETIC FOOT

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Introduction: The microvascular complications (diabetic retinopathy and nephropathy), except of course of the neuropathy that constitutes the main reason of diabetic foot, coexist and affect, as microalbuminuria is distinguished, in the development of the Diabetic Foot (DF).

Aim: The detection of coexistence and the relation of retinopathy and nephropathy in the pathogenesis and in the severity of DF.

Patients - Methods: Our material of 256 patients (171 male, 85 female) with "diabetic foot" that recorded, monitored and likely hospitalized for this reason. We have complete data concerning only 250 of them, aged $65,31 \pm 10,25$ years, mean HbA1c: $8,65 \pm 1,7\%$, of duration of diabetes $15,84 \pm 9,2$ years and 32% of them are active smokers. The history of the reason which caused the lesion is taken and patients are examined for neuropathy, and peripheral arterial disease (NDS, VPT, ABI are measured).

11,2% of our patients was not detected with nephropathy, while 37,3% of them have microalbuminuria, 39,8% of the patients have macroalbuminuria and 11,6% of the total number has end-stage renal failure.

The existence or not of diabetic retinopathy (DR) was recorded and we have 175/215 patients with DR (81%) that has been examined, by that the 93/234 (40%) had already done laser photocoagulation, while 15 patients are blinded.

Results: 87 (34 %) of our patients have neuropathic lesions, a number of 34 (13,3%) purely ischaemic and another 120 (46,9%) neuroischaemic lesions. The existence of DR appears to have strong cross-correlation with the pathogenesis of DF ($p < 0,021$), after 52,1% of patients with DR have neuroischaemic lesions and 37,3% of them have neuropathic. There is no cross-correlation with the severity of the lesion ($p < 0,121$).

We observe the appearance of retinopathy in 11 years of diabetes in patients with neuropathy or both neuropathy and ischaemia, while in patients with purely ischaemic lesions in 7,5 years. There was no significant difference between the duration of DR and the type of the lesion ($p = 0,269$). The duration of DR does not appear to relate itself with the severity of the ulcer ($p = 0,208$).

The cross-correlation of the type of nephropathy with the pathogenesis of DF ($p < 0,044$) is obvious and especially with neuropathy, since 48,3% of the individuals with nephropathy have neuroischaemic and 38,0% of them have neuropathic lesions, while does not exist cross-correlation with the severity of DF ($p < 0,383$).

Conclusion: The coexistence of microvascular complications in patients with DF is obvious with increased frequency, probably because of poor metabolic control and self-monitoring, as well as the role of the retinopathy and nephropathy in the pathogenesis of DF, particularly among patients with neuropathy.

PREVENTION AND MANAGEMENT OF THE DIABETIC FOOT - OUR EXPERIENCE FROM OUR NEW DIABETIC FOOT CLINIC

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Aim: The classification of the diabetic ulcers of the lower extremities and the epidemiological data of the diabetic patients who visited the Diabetic Foot Clinic of our hospital, a clinic which provides medical services to the public since March 2011.

Materials - Methods: Twenty-one patients participated in this study; these patients had visited the Diabetic Foot Clinic in a 3-month period starting from last March to last June. The patients were registered and the following data were recorded: age, gender, profession, comorbidities, medication as well as the reason for a diabetic foot clinic visit. Complete laboratory tests, examination for peripheral neuropathy and vessel triplex (if it was necessary) were performed.

Results: Of those 21 patients, aged 59 ± 10.5 years and duration of diabetes 12 ± 10.2 years, 7 (33.3%) were women and 14 (66.6%) were men. Of those 7 women, 5 (71.4%) were housewives and only 2 (28.5%) were employed. Of the 14 men, 5 were working in the private sector, 2 were working in the public sector, 2 were farmers, 1 was unemployed and 4 were pensioners. 3 (14.2%) had neuropathic ulcers, 4 (23.8%) neuroischemic ulcers, 8 (38.09%) came for preventive examination and 6 (28.5%) visited the clinic for other reasons (cellulitis, painful diabetic neuropathy, second opinion, curiosity ...).

Conclusion: Although the Diabetic Foot Clinic was a new clinic in our hospital, our small sample shows that the operation of these clinics is essential because they can decrease the morbidity and the mortality of the diabetic foot and they can diminish the amputations and the overall time of hospitalization of the diabetic patients.

OSTEOPROTEGERIN LEVELS CORRELATE WITH SEVERITY OF PERIPHERAL ARTERIAL DISEASE AND PERIPHERAL NEUROPATHY IN TYPE 2 DIABETIC PATIENTS

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Introduction: Increased osteoprotegerin (OPG) levels have been found in diabetic patients with micro- and macrovascular complications. Recent studies have shown that OPG concentrations correlate with severity of peripheral arterial disease (PAD) in patients without type 2 diabetes mellitus (T2DM). However, no data exist on the association between OPG levels and severity of PAD and peripheral neuropathy (PN) in patients with T2DM.

Purpose: The aim of this study was to look for potential association between OPG levels and severity of PAD and PN in patients with T2DM.

Methods: A total of 74 patients with T2DM were recruited (mean age 67.8 ± 9.0 years, duration of diabetes 15.3 ± 10.9 years). Serum OPG levels were measured using ELISA. PAD was diagnosed by means of ankle-brachial index ($ABI \leq 0.9$). Patients with ABI values > 1.3 were excluded from further analysis. Diagnosis of PN was based on neuropathy disability score (NDS) and vibration perception threshold (VPT).

Results: Patients with PAD ($n=27$) had significantly higher serum OPG levels in comparison with those without PAD (18.0 ± 4.9 vs. 14.8 ± 4.8 pmol/l, $p < 0.001$). Patients with PN ($n=36$) had also higher OPG levels than patients without PN (17.1 ± 5.8 vs. 15.0 ± 4.0 pmol/l, $p < 0.011$). OPG levels were significantly associated with ABI ($r = -0.309$, $p < 0.001$), VPT ($r = 0.370$, $p < 0.001$) and NDS ($r = 0.324$, $p < 0.001$). The association between OPG concentrations and ABI remained significant after adjustment for age, diabetes duration, sex and presence of PN. The associations between OPG levels and VPT, NDS remained significant after adjustment for age, diabetes duration, sex and presence of PAD.

Conclusion: Serum OPG levels are increased in diabetic patients with either PAD or PN and are associated independently with the severity of these complications.

SERUM OSTEOPROTEGERIN LEVELS CORRELATE WITH SEVERITY OF LOWER EXTREMITY ARTERIAL CALCIFICATION IN PATIENTS WITH TYPE 2 DIABETES

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Introduction: Recent studies have shown that osteoprotegerin (OPG) concentrations correlate with coronary artery calcification in patients with type 2 diabetes mellitus (T2DM). However, no data exists on the association between OPG concentrations with lower extremity arterial calcification (LEAC) in patients with T2DM.

Purpose: The aim of this study was to look for potential association between OPG levels and severity of LEAC in patients with T2DM.

Methods: A total of 74 patients with T2DM were recruited (mean age 67.8±9.0 years, duration of diabetes 15.3±10.9 years). Serum OPG levels were measured using ELISA. In all patients radiographs were taken of both feet and ankles. LEAC was graded in a scale from 0-5 at 4 locations (posterior tibial and dorsalis pedis arteries bilaterally). The total LEAC score (0-20) at all 4 locations was calculated. Diagnosis of peripheral arterial disease (PAD) was based on the absence of triphasic waveform at the posterior tibial artery, while diagnosis of PN on neuropathy disability score (NDS) and vibration perception threshold (VPT).

Results: Patients without or with less LEAC (grade 0-2 based on the maximum LEAC grade at one out of 4 locations; n=44) had lower OPG levels compared with patients with more severe LEAC (grade 3-5; n=30) (15.0±4.4 vs 18.6±6.0 pmol/l, p<0.001). The total LEAC score was significantly associated with age (r=0.23, p=0.011), pulse pressure (r=0.41, p<0.001), glomerular filtration rate (r= -0.20, p= 0.026), albumin-to-creatinine ratio (r=0.36, p<0.001), VPT (r=0.26, p=0.002) and OPG levels (r= 0.25, p=0.004). The association between the total LEAC score and OPG concentrations remained significant after adjustment for age, GFR, diabetes duration and PN status.

Conclusion: Serum OPG levels are associated with severity of LEAC in patients with T2DM.

POPLITEAL ARTERY PERCUTANEOUS TRANLUMINAL ANGIOPLASTY FOR CHRONIC CRITICAL LIMB ISCHEMIA AND DIABETES MELLITUS LESIONS. ANALYSIS OF A SINGLE-CENTER EXPERIENCE

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Introduction: This study assessed the three year outcome of patients with critical limb ischemia, Fontaine 3 & 4 and diabetic feet with calcific ankle arteries (> 60%) who had undergone balloon PTA.

Purpose: Treat patients with chronic lower limb ischemia and diabetic lesions by using endovascular techniques-balloon PTA.

Methods: Ten patients treated with Ipsilateral access PTA with high pressure low profile balloons (10 Atm) of 2-4mm diameter and 400mm length. Guidewires of 0,014" inches and hydrophilic wires of 0,035 inches.

Results: Three years meta analysis with technical success (no complications by surpassing the occlusion). Primary patency - 88%.

Limb salvage 90%. Tissue loss was a bad predictive sign only for the limb patency but not for the limb surveillance.

Conclusion: PTA appears to be the treatment of choice in patients with popliteal lesions due to chronic obstructive disease and diabetes mellitus. Balloon angioplasty can be successful even in long lesions with the least complications and mortality. Unsuccessfulness of the method does not exclude an open bypass operation moreover it appears to be a repeatable method in case of restenosis.

PLEIOTROPIC EFFECTS OF ATORVASTATIN ON CENTRAL AND PERIPHERAL ARTERIES OF PATIENTS WITH TYPE 2 DIABETES

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Introduction: Dyslipidaemia is a modifiable atherogenic risk factor in patients with type 2 diabetes. Statins have been proved to significantly reduce cardiovascular morbidity in large clinical trials. Increased arterial stiffness, assessed by pulse wave velocity (PWV), has emerged as an independent predictor of cardiovascular events.

Purpose: The aim of this prospective study was to evaluate the effect of atorvastatin administration for 1 year on arterial stiffness in patients with type 2 diabetes.

Methods: A total of 54 patients were recruited, 31 patients were assigned to 12-months atorvastatin therapy (10 mg daily) and a low fat diet, and 23 patients to low-fat diet only. The two groups were comparable at baseline in terms of lipids, body mass index, diabetes duration, age and HbA1c. The main inclusion criteria were LDLc ≥ 100 mg/dl and absence of macrovascular disease. PWV was determined by applanation tonometry (SphygmoCor Vx, AtCor Medical, Sydney, Australia) along the carotid-femoral (PWVcf) and the carotid-radial (PWVcr) arteries. Plasma lipids, HbA1c, and PWV were evaluated prospectively at baseline, 3, 6, and 12 months in both groups.

Results: Total cholesterol and LDLc declined significantly in the atorvastatin-treated group at the examined time intervals. In the same group a significant reduction in PWVcr and PWVcf was observed at 3 months (4.59 %, $p < 0.001$, and 9.18 %, $p < 0.001$), which was maintained at 6 (6.55 %, $p = 0.007$, and 12.99 %, $p < 0.001$) and 12 months (9.9%, $p = 0.01$, and 12.43%, $p = 0.003$, respectively). In the diet treated group no significant changes of PWVcr and PWVcf were found.

Conclusion: A low dose of atorvastatin improves arterial stiffness in patients with T2DM.

BULLOSIS DIABETICORUM: A NOT-SO-RARE CONDITION CHARACTERISTIC OF DIABETES MELLITUS

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Introduction: Bullosis diabeticorum is considered a rare and relatively harmless skin manifestation with painless, tense, superficial blisters appearing rapidly and abruptly in diabetic persons, usually on the feet and lower legs, without preceding trauma. The bullae arise from a noninflamed base, are usually multiple, and vary in size from 1 to several cm. The bullae rupture in approximately 1 week, leaving a deep, painless ulcer that forms a firmly adherent crust. They commonly heal in 2-6 weeks without scarring, but complications such as secondary bacterial infection or hemorrhage may occur. Many patients never have another episode, whereas others have recurrences. Most papers report only a few cases and the cause of the blisters is unknown, but is possibly ischemic.

Patients: We describe 6 patients with bullosis diabeticorum, 4 men/2 women, all with T2DM. Characteristics of these patients are summarized in the following Table.

Px	Sex	Age	Type of DM	HbA1c (%)	Duration of DM (yrs)	Tx of DM	Localization of bullae	Size (cm)	Abx
1	M	79	2	5	17	I	Foot	7,5	Y
2	M	59	2	5,8	10	I	Lower leg	3	Y
3	M	60	2	9,6	20	I	Foot	2,5	Y
4	M	72	2	7,5	25	I	Lower leg	3,5	N
5	F	90	2	6,8	4	OA	Lower leg	2	Y
6	F	77	2	10,1	0	-	Upper leg	2,5	N

I: insulin, OA: oral hypoglycemic agents, Y=yes, N=no, Tx=treatment, Abx=antibiotics

In all 6 patients, bullae either ruptured spontaneously or were perforated in the clinic, and clear yellowish sterile fluid came out. In 2 out of 6 patients antibiotics were prescribed because of superinfection, and 2 more patients were already on antibiotics because of another ulcer complicated with osteomyelitis. There was complete healing of lesions in all patients.

Conclusion: We have experienced that the lesions are not so rare. Although the containing fluid is sterile, it is very easy that a secondary infection occurs after rupture, so antibiotic treatment is necessary in many cases. Bullae heal completely in several weeks' time.

THE ASSOCIATION BETWEEN DIABETIC AUTONOMIC NEUROPATHY AND CUTANEOUS CIRCULATION IN PATIENTS WITH TYPE 2 DIABETES

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Introduction: Transcutaneous oxygen tension (TcPO₂) reflects the cutaneous microvascular perfusion status and TcPO₂ levels are reduced in patients with type 2 diabetes mellitus (T2DM).

Purpose: The aim of this study was to examine the potential effect of cardiac autonomic neuropathy (CAN) on cutaneous circulation assessed by determination of TcPO₂ in patients with T2DM.

Methods: A total of 100 patients with T2DM were recruited (mean age 66.5±8.7 years, duration of diabetes 14.0±10.8 years). TcPO₂ was measured using a TCM30 system and the electrode was placed on the dorsum between the first and second metatarsal heads. Diagnosis of CAN was based on the battery of the four autonomic function tests proposed by Ewing. Diagnosis of peripheral arterial disease (PAD) was based on the absence of triphasic waveform in the posterior tibial artery, while of peripheral neuropathy (PN) on neuropathy disability score (NDS) and vibration perception threshold (VPT).

Results: A total of 20 subjects had CAN. Subjects with CAN had more often PAD and PN and lower TcPO₂ levels in comparison with subjects without CAN (43.6±15.0 vs. 49.1±11.1 mmHg, p=0.033). Univariate linear regression analysis showed that diabetes duration, HbA1c values, presence of PAD, presence of PN and presence and severity of CAN were significantly and negatively associated with TcPO₂. Model 1 of the multivariate analysis demonstrated that after adjustment for gender, age, duration of diabetes and HbA1c levels, severity of CAN and presence of PAD were significantly and negatively associated with TcPO₂. Model 2 showed that after controlling for gender, age, diabetes duration and HbA1c values, severity of CAN and presence of PN were significantly associated with lower TcPO₂ levels.

Conclusion: Presence and severity of CAN is associated with reduction in cutaneous perfusion irrespective of the presence of PAD or PN.

ASSOCIATION OF ARTERIAL STIFFNESS AND CARDIOVASCULAR RISK FACTORS IN PATIENTS WITH TYPE 2 DIABETES

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Introduction: Increased arterial stiffness represents the link between diabetes and increased cardiovascular disease. Aortic pulse wave velocity (aPWV) has emerged as an independent predictor of cardiovascular morbidity and mortality in patients with diabetes.

Purpose: Aim of our study was to evaluate the association between aPWV and cardiovascular risk factors (new and classic) in patients with type 2 diabetes.

Methods: A total of 163 patients (74 men, 89 women, mean age 62,59±8,97 yrs) with type 2 diabetes and no apparent clinically macrovascular disease were assessed. We assessed basic clinical and demographical characteristics. Plasma glucose, creatinine, HbA1c, fibrinogen, plasma osteoprotegerin (OPG), hsCRP, GFR and urine albumin/creatinine ratio (A/Cu) were evaluated. aPWV was determined by applanation tonometry (SphygmoCor Vx, AtCor Medical, Sydney, Australia) along the carotid-femoral.

Results: In the total patients enrolled, 127 (65.8%) had plasma LDLc >100 mg/dl, 81 (42%) patients were under statin therapy, 120 (62.2%) had hypertension, 39 (20.2%) patients suffered from peripheral neuropathy, and 34 (33%) were smokers. Linear regression analysis showed that aPWV was significant associated with age ($p<0.001$), BMI ($p=0.025$), diabetes duration ($p=0.006$), OPG ($p=0.002$), A/Cu ($p=0.006$), plasma creatinine ($p=0.009$), calculated GFR ($p<0.001$), presence of peripheral neuropathy ($p<0.001$) and pulse pressure ($p<0.001$). No significant association was observed between aPWV and arterial pressure, smoking, plasma lipids, HbA1c, waist circumference, waist-to-hip ratio and hsCRP.

Conclusion: In patients with type 2 diabetes, beyond classic risk factors, new factors, such as peripheral neuropathy and OPG are associated with arterial stiffness.

VALIDATION OF DIFFERENT RESPONSES OF THE SUDOMOTOR FUNCTION TEST (NEUROPAD) IN IDENTIFYING TYPE 2 DIABETIC PATIENTS WITH PERIPHERAL OVERALL NERVE DYSFUNCTION

A multicenter study

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Objective: To investigate the utility of different responses of the indicator test for Sudomotor function (SFT) for the detection of overall fiber dysfunction in diabetic patients.

Research Design and Methods: The study included 1010 type 2 diabetic patients. Assessments of overall nerve fiber dysfunction were diagnosed and graded clinically using the Neuropathy Disability Score (NDS). The SFT was applied for 10 minutes on the plantar aspect of the feet and results were recorded as pink, patchy (blue/pink), and blue.

Results: Patients with blue SFT results were older, had a longer duration of Diabetes, and expressed more severe overall fiber dysfunction compared to those with patchy and normal SFT response. The abnormal SFT result defined as patchy and/or blue had 94.9% sensitivity and 70.2% specificity for neuropathy (overall fiber dysfunction). The abnormal SFT result defined as only blue had 64% sensitivity and 96% specificity for overall neuropathy while the positive predictive value was 82%.

Conclusion: The present study showed that SFN blue response performs more specific results in order to identify diabetic patients at risk of FU. In addition SFT patchy and or blue response has better performance for screening purposes in order to identify diabetic patients with overall nerve fiber dysfunction.

DIFFERENT RESPONSES OF A SCREENING TOOL (SUDOMOTOR FUNCTION TEST) IN IDENTIFYING TYPE 2 DIABETIC PATIENTS WITH SMALL FIBER NERVE DYSFUNCTION

A multicenter study

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Background: Small-fiber neuropathy is considered as an earlier manifestation of chronic sensory motor neuropathy the main initiating factor for foot ulceration.

Objective: To investigate the utility of different responses of the indicator test for Sudomotor function Neuropad® (SFT) for the detection of small nerve fiber dysfunction in diabetic patients.

Research Design and Methods: The study included 1010 type 2 diabetic patients. Assessments of small nerve fiber dysfunction were diagnosed and graded clinically using the Neuropathy Disability Score (NDS). The SFT was applied for 10 minutes on the plantar aspect of the feet and results were recorded as pink, patchy (blue/pink), and blue.

Results: Patients with blue SFN results were older, had a longer duration of Diabetes, and expressed more severe small fiber dysfunction compared to those with patchy and normal SFT response. The abnormal SFT result defined as patchy and/or blue had 85.6% sensitivity, 71.2% specificity for small fibre dysfunction respectively. The abnormal SFT result defined as only blue had 64% sensitivity and 96% specificity for overall neuropathy while the positive predictive value was 82%.

Conclusion: The present study showed that SFT blue response has more specific results in order to identify diabetic patients with small fiber dysfunction. In addition SFT blue response had better performance for screening purposes.

THE ROLE OF HYPERTENSION AND OBESITY IN THE NEUROPATHIC AND MICROANGIOPATHIC COMPLICATIONS OF DIABETES TYPE 2

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Background: Increased body weight in patients with diabetes type 2, as well as the co-existence of aggravating factors such as arterial hypertension, may accelerate the onset of diabetes complications, such as diabetic neuropathy and retinopathy.

Aim: Aim of this study was to investigate the existence or not of correlation between hypertension and Body Mass Index (BMI) with these two complications of diabetes type 2.

Patients - Methods: 342 patients (149 males and 193 females) with diabetes type 2 participated in this study. The mean age was 66 years old (65 in males, 67 in females), while the mean duration of diabetes was 10.4 years for women and 7.5 for men. Vibration perception in the big toe, the ankle and the lower part of the shin was measured and recorded, as well as the sensation (touch, pain, cold). In all patients the release of tendon reflexes (patellar reflex, Achilles tendon reflex) was examined. Moreover, medical history of hypertension, Body Mass Index (BMI) at the first visit and the existence of retina damage were recorded.

Results: 250 patients (73,09%) suffered from hypertension and already receiving medication. 59 were overweight (24 males, 35 females), while 251 were obese (118 males, 133 females). There is statistical significance between high BMI and retinopathy ($\chi^2=50,904$, $df=24$, $p<0.01$). There is statistical significance between high BMI and the pathologic release of the patella tendon reflex ($\chi^2=208,654$, $df=52$, $p<0.01$), as well as Achilles tendon reflex ($\chi^2=76,181$, $df=52$, $p<0.05$). No correlation between obesity and the different sensation dysfunctions of the lower limbs. Hypertension predicts independently the onset of neuropathic and microangiopathic disorders in the univariate statistical analysis.

Conclusion: It seems that body weight affects the function of peripheral nerve fibers, as well as other factors such as hypertension. Though, more perspective studies are necessary to confirm this finding.

MACROVASCULAR COMPLICATIONS AND RISK FACTORS, COEXISTENCE, THEIR ROLE IN THE PATHOGENESIS AND THE SEVERITY OF DIABETIC FOOT

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Introduction: Macrovascular complications and risk factors, such as hypertension, dyslipidaemia, except of course of the Peripheral Arterial Disease (PAD) that constitutes sovereign factor in aetiopathology of the diabetic foot, all these also affect in the development of Diabetic Foot.

Aim: The detection of coexistence and the relation of macrovascular complications and risk factors in the pathogenesis and severity of Diabetic Foot.

Patients - Methods: Our material consists of 256 patients (171 males, 85 females) with “diabetic foot” that recorded, monitored and likely hospitalized for this reason. We have complete data concerning 250 of them, aged $65,31 \pm 10,25$ years, mean HbA1c: $8,65 \pm 1,7\%$, of duration of diabetes $15,84 \pm 9,2$ years and 32% of them are active smokers. The history of the reason which caused the lesion is taken and patients are examined for neuropathy, and peripheral arterial disease (NDS, VPT, ABI are measured). Dyslipidaemia is observed in 29,3% (68/232) of our patients, while 79,4% (185/233) of our patients suffers from hypertension. In 40,2 % (101 patients) there is known coronary heart disease, while the 10,6% (27 patients) had a history of stroke.

Results: 87 (34 %) of our patients have neuropathic lesions a number of 34 (13,3%) purely ischaemic and another 120 (46,9%) has neuroischaemic lesions.

The presence of coronary heart disease (CHD) has an important statistical cross-correlation with the pathogenesis of the diabetic foot ($p < 0,002$), as we recognize that 60,4% of our patients with CHD are having neuroischaemic lesions, 16,7% of them are having ischaemic lesions and only 22,9% of them has neuropathic lesions. There is no cross-correlation with the severity of diabetic foot ($p = 0,086$). The presence of hypertension does not appear to relate itself with the pathogenesis and the severity of the lesion ($p < 0,133$ and $p < 0,577$ respectively). However, there is a significant difference among the patients with hypertension, which coexist in the 84,5% of neuroischaemic lesions, in opposite of the 72,9% of neuropathic only.

The presence or not of dyslipidaemia does not appear to statistically relate itself neither with the pathogenesis nor with the severity of lesion ($p < 0,413$ and $p < 0,218$ respectively). We have similar results with the presence of heart failure of relating itself with the pathogenesis ($p < 0,018$) and 66% of the patients with heart failure have neuroischaemic lesions and 13,2% of them purely ischaemic. However there is no cross-correlation with the severity of diabetic foot ($p < 0,751$).

The history of a stroke does not related neither with the pathogenesis nor with the severity of the diabetic foot ($p < 0,910$ and $p < 0,434$ respectively).

The duration of smoking also is related with the type of lesion ($p < 0,001$).

Conclusion: The coexistence of macrovascular complications, hypertension, dyslipidaemia and smoking in patients with Diabetic Foot is obvious as well as their role into pathogenesis of the Diabetic Foot, especially among those patients who already have ischaemia.

STUDY OF DIABETIC NEUROPATHY AND RETINOPATHY IN PATIENTS WITH DIABETES TYPE 2 IN CORRELATION WITH THE DURATION AND EFFECTIVE MANAGEMENT OF THE DISEASE

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Background: Diabetic neuropathy and retinopathy consist two of the most severe complications of diabetes mellitus type 2. Numerous studies implicate bad control and longer course of the disease for the more severe outcome of these implications.

Objectives: This study investigates the correlation of diabetic neuropathy and retinopathy with the disease control and duration

Patients - Methods: 342 patients (149 males and 193 females) with diabetes type 2 participated in this study. The mean age was 66 years old (65 in males, 67 in females), while the mean duration of diabetes was 10.4 years for women and 7.5 for men. Vibration perception in the big toe, the ankle and the lower part of the shin was measured and recorded, as well as the sensation (touch, pain, cold). In all patients the release of tendon reflexes (patellar reflex, Achilles reflex) was examined. Additionally, all of them underwent fundoscopy (to confirm the existence of retinopathy or sclerosis of the retina vessels) and measurement of glycated hemoglobin in blood at their first visit.

Results: 21.2% of the patients had retinopathy damages, while 47.8% of them suffered from sclerosis of the retina vessels. From these patients, 75.3% had bad control of blood glucose ($HbA1C > 7$), though there was no statistically significant correlation between $HbA1C$ and retinopathy (Fisher's exact test, $p = 1.00$) during the first screening test of the patients. More severe retinopathy was observed in the majority of the patients who suffered from diabetes type 2 for more than 10 years. There is statistically significant correlation between the time course of the disease for more than 10 years and the disorder of the patella tendon reflex. ($\chi^2 = 5.358$, $df = 2$, $p < 0.05$), with similar observation in the Achilles reflex ($\chi^2 = 6.875$, $df = 2$, $p < 0.05$). There is no statistical significance between the duration of the diabetes and the pathologic perception of pain ($\chi^2 = 8.810$, $df = 3$, $p < 0.05$), as well as in the perception of cold ($\chi^2 = 20.014$, $df = 3$, $p < 0.01$) and vibrations ($\chi^2 = 10.059$, $df = 3$, $p < 0.05$). There is statistically significant dependence of retinopathy from the time course of the disease (Fisher's exact test, $p < 0.01$).

Conclusion: The duration of the disease and bad control of blood glucose may affect the onset and the severity of neuropathic and microangiopathic complications of diabetes type 2.

HEREDITY AND DIABETIC NEUROPATHY AND RETINOPATHY IN PATIENTS WITH DIABETES TYPE 2

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Background: The obtaining of a fully detailed medical record and the regular follow-up of the patients with diabetes type 2 may contribute beneficially in the prevention and control of the complications of the disease, anticipating its heavier consequences.

Objectives: Aim of this study is the investigation of the correlation between the heredity and those two complications of diabetes type 2.

Patients - Methods: 342 patients (149 males and 193 females) with diabetes type 2 participated in this study. The mean age was 66 years old (65 in males, 67 in females), while the mean duration of diabetes was 10.4 years for women and 7.5 for men. Vibration perception in the big toe, the ankle and the lower part of the shin was measured and recorded, as well as the sensation (touch, pain, cold). In all patients the release of tendon reflexes (patellar reflex, Achilles reflex) was examined. Moreover, positive family history of diabetes (at least one relative suffering from diabetes), Body Mass Index (BMI) and the existence of retina damage were recorded.

Results: 210 patients (61.4%) mentioned positive family history for diabetes type 2 at least in one relative person). 88,75% of the patients with tendon reflex disorder had positive family history. There is statistical significant correlation between positive family history and the release of the patella tendon reflex ($\chi^2=5,138$, $df=2$, $p<0.05$). 59,2% of the patients with disorder in the sensation in the lower limbs had a positive family history. The relation between positive family history for diabetes and dysfunction of the large nerve fibers is statistically significant (vibration disorder, $\chi^2=7,122$, $df=2$, $p<0.05$). 80% of the patients with retinopathy had a positive family history for diabetes, though there was no statistical significance among these two factors.

Conclusion: The existence of a positive family record for diabetes type 2, may comprise a significant factor for the onset of complications of the disease, mainly the neuropathic disorders, pointing out the importance of the obtaining a fully detailed medical personal and family history for the optimum management of patients in risk of diabetic neuropathy and retinopathy manifestation.

NECROTIZING SOFT TISSUE INFECTIONS OF THE DIABETIC FOOT: AN EMERGENCY SITUATION

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Introduction - purpose: Diabetes mellitus and peripheral angiopathy (PAD) are predisposing factors for necrotizing soft tissue infections (NSTIs), which are characterized by fulminant destruction of tissue, severe and continuous pain, systemic signs of toxicity, and a high rate of mortality. Despite the various different names that have been used, early diagnosis and appropriate treatment (antimicrobial therapy - including clindamycin - and surgical intervention) must be performed. We describe 2 cases of NSTI in diabetic patients with PAD.

Case 1: A 60-year-old man with T2DM (on insulin), coronary artery disease and PAD presented with NSTI (necrotizing fasciitis / myonecrosis) with fever (38°C), chills and brownish malodorous drainage, because of infected extended ulcer (PEDIS 4) of left ankle. He reported use of hydrocolloid dressings for the previous week. After admission, he was treated with extended surgical debridement and iv antibiotics (pip/tazo, metronidazole, clindamycin). Alginate wound dressings with daily change were used. Culture grew *Citrobacter freundii* and *Streptococcus uberis* (*streptococcus pyogenes*). Patient's outcome was satisfactory.

Case 2: A 80-year-old man with T2DM (on insulin) and PAD (70-75% stenosis) presented with PEDIS 3 infection (left 1st toe extending to mid-metatarsal) following trauma that occurred 2 months before. He reported recent antibiotic therapy (cefuroxime/clindamycin). He had no systemic symptoms, but there was severe local pain. After admission, patient was treated with iv ampicillin/sulbactam. On day 2 clindamycin was added because of localized necrotic areas. Apart from arterial calcification, X-ray was otherwise normal. Surgical incision and debridement of lateral side of toe was performed and patient's condition improved. Toe nail came out spontaneously after 2 weeks. Patient's outcome was satisfactory.

Conclusion: Diabetic patients with PAD deserve increased level of suspicion for NSTIs, which consist a medical emergency. Immediate treatment with surgical debridement and antibiotic regimen including clindamycin is imperative.

USE OF VACUUM-ASSISTED CLOSURE (VAC) DEVICE IN A DIABETIC FOOT CLINIC

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Introduction: Negative pressure wound therapy, otherwise vacuum-assisted closure (VAC), is a relatively new (mid-'90s) adjunctive therapy for open wounds that applies subatmospheric pressure to the wound surface. VAC has been utilized in the treatment of wounds from acute injury (eg, trauma, burns, surgical debridement), and diabetic foot ulcers. It has several advantages over traditional wound management, including simplification of wound care and accelerated wound healing, with subsequent shortening of patient hospitalization.

Purpose: To record all diabetic patients with foot ulcers treated with VAC in our foot clinic. We studied patient outcome but also cost effectiveness of VAC.

Patients - Methods: Retrospective study including 13 diabetic patients with diabetic foot ulcers treated with VAC as adjunctive therapy. These patients, 8 men / 5 women, age 44-76 years, presented at diabetic foot clinic of A. Fleming General Hospital from January to December 2009.

Results: There was successful outcome (complete healing) in all 13 patients with diabetic foot ulcers, and they are still seen in our diabetic foot clinic. Unfortunately, 3/13 patients presented again with ulcerations occurring in the same or different location, because of inappropriate footwear and poor glycemic control.

Conclusion: VAC has successfully been utilized in the management of diabetic foot ulcers. Its use reduces edema, increases blood flow, stimulates granulation tissue and accelerates wound healing, thus decreasing hospital stay. But considering its increased costs {hospital price 450 EUR for each application (foam dressing, semiocclusive adhesive cover, fluid collection system) and usually 2-3 applications per patient are needed}, VAC has to be applied only in selected cases of diabetic foot ulcers, when conventional treatment has failed: in deep, complicated and nonhealing ulcers, in younger patients with satisfactory performance status and, most important, with good compliance regarding glycemic control, offloading and appropriate footwear.

SUCCESSFUL TREATMENT OF SELECTED CASES OF OSTEOMYELITIS IN THE DIABETIC FOOT WITHOUT SURGICAL BONE REMOVAL

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Introduction - purpose: Early diagnosis and culture-based or empirical antibiotic therapy for osteomyelitis (OM) of the diabetic foot (DF) is needed. Traditionally, surgical therapy (bone resection, or even amputation) is considered essential for treatment of chronic OM. Recently, some authorities have disputed the routine need for surgical therapy. We describe 2 cases of successful treatment of DF OM without surgical bone removal. Diagnosis was based on clinical grounds and imaging techniques. Patients were successfully treated with antibiotics, alginate dressings (daily change), LMWH, buflomedil, offloading and tight glucose control. Treatment success was based on clinical examination, blood tests and radiology.

Case 1: A 36-year-old man with T1DM with history of foot ulcer 2 years before, peripheral neuropathy (PN) and HbA1c=11,8% presented with multiple infected ulcers (PEDIS 3) of plantar surface of the right foot and swollen inguinal lymph nodes, because of inappropriate footwear during summer holidays. X-ray showed osteolysis of 1st metatarsal, and MRI showed extended inflammation with osteomyelitis. Culture grew MSSA, Enterococcus, CNS, Corynebacterium, Bacteroides, Prevotella, & anaerobic peptococcus. Targeted antibiotic therapy was given for a total of 3.5 months (iv for the first 5 weeks). Patient's condition remains satisfactory at 6 months' follow-up.

Case 2: A 72-year-old woman with T2DM on insulin with history of toe amputation 2 years before, PN, peripheral angiopathy and HbA1c=9% presented with worsening of pre-existing (for 4 months) ulcer of 2nd toe ('sausage toe'). X-ray showed 1st metatarsal absorption. Culture was unreliable, and empirical antibiotics were given for 6 months (iv for the first 2 weeks). Although x-ray at 4 weeks showed worsening, radiology at 15 weeks showed significant improvement. She was in good shape at 2 months' follow-up.

Conclusion: Medical therapy of DF OM, without surgical bone removal, needs persistent and long-term treatment, but it can be successful in selected cases, despite the contrary beliefs of some doctors, especially surgeons. Consequently, surgical treatment and especially amputation is not necessary in many cases.

REPRODUCIBILITY OF THE LANARKSHIRE OXIMETRY (LOI) INDEX IN TYPE 2 DIABETES MELLITUS

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Introduction: Lanarkshire Oximetry Index (LOI) is an alternative diagnostic tool for peripheral arterial disease (PAD).

Purpose: The aim of the present study was to evaluate the reproducibility of LOI in patients with type 2 diabetes mellitus (T2DM).

Methods: This study included 10 patients with T2DM (5 men, 5 women, aged 53-77 years). LOI was measured by a pulse oximeter (Nellcor Puritan Bennett, NPB-295, Nellcor Inc) and a sphygmomanometer. For finger pressure, the pulse oximeter sensor was placed on the index finger and the sphygmomanometer cuff around the patient's upper arm. For hallux pressure, the pulse oximeter sensor was placed on the hallux and the sphygmomanometer cuff around the patient's calf. Measurements were performed on the right. LOI was calculated by dividing hallux pressure by finger pressure. To assess intra-observer reproducibility, patients were repeatedly examined by the same operator. Inter-observer reproducibility was assessed through examination by a second operator.

Results: Intra-observer variability was low, as evidenced by the % Coefficient of Variance (mean CV%: 2.89%, range: 1.66-4.58%). On examination by the two operators, mean difference in LOI was 0.043 (range 0.03-0.06).

Conclusion: These results suggest that LOI has excellent reproducibility in T2DM and encourage its further exploration as an alternative diagnostic tool for PAD.

IS TISSUE OXYGENATION A MAJOR DETERMINANT OF ULCER OUTCOME IN DIABETIC PATIENTS?

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Background: Tissue hypoxia is an important factor in the occurrence of diseases of the lower extremities in diabetic patients. Therefore, the oxygen at high pressure (increased O₂ diffusion in the tissues) has been proposed as an adjunct therapy to address these problems.

Aim: The purpose of this study were a) to assess the therapeutic effects of hyperbaric oxygen therapy combined with conventional therapeutic approaches to lesions in the "diabetic foot" and b) the search for factors that might predict the outcome.

Patients - Methods: The study included 23 diabetic 9,74 and duration±patients (type-approval2 n=19, males n=16), aged 58,87 8,5 years. ±of disease 13 The clinical examination included assessment of the severity of a) ulcer (classification by Wagner) b) of diabetic neuropathy (nerve dysfunction index determination - NDS and vibration perception threshold - VPT) and c) peripheral vascular disease (clinically and by determining the ABI Tissue oxygenation. was assessed by transcutaneous PO₂ in normal conditions (1 ATA) and hyperoxia (O₂ breathing at 2.4 ATA).

Results: a) Ulcers (by Wagner) degree (4) n = 4, (3) n = 6, (2) n = 10, (1) n = 3, b) neuropathic ulcers n=8, neuroischemic n=12, ischemic n=3. Healing of ulcers was achieved in 14 (61%) - group A, improvement in 5 (21%) - Group B and failure in 4 (18%) - group C. There were no differences between group A and group B regarding age, disease duration, blood glucose, NDS, VPT, PO₂ (normal conditions 65±40 mmHg vs 69±26 p=ns) and PO₂ in hyperoxia (465±275 mm Hg vs 402±135 mm Hg, p=ns). Comparing groups A and C was found higher PO₂ in hyperoxia in group A (465± 275 mm Hg vs 203± 106 mm Hg p <0,05), while other factors did not show any differences.

Conclusion: The administration of hyperbaric oxygen is a useful therapeutic approach (complementary) in diabetic foot disease. Patients with reduced tissue oxygenation in hyperoxia did not respond to therapeutic approach.

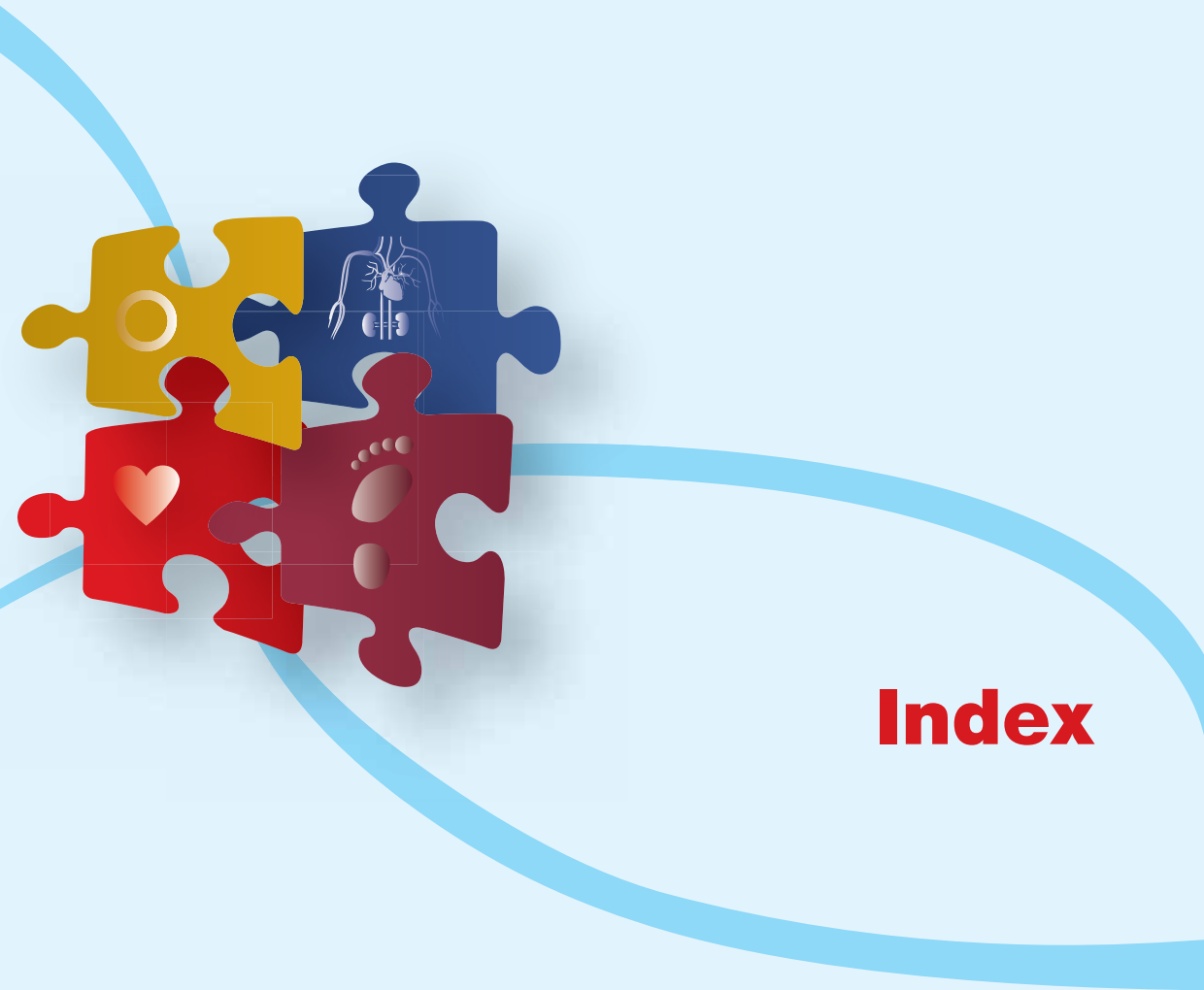


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General Information

Conference Date

July, 7-10, 2011

Conference Venue

AKS PORTO HELI HOTEL****

Conference Center

Porto Heli, 21061, Argolis, Peloponnese

Tel.: +30 27540 - 98073

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Website: www.akshotels.com/porto-heli-hotel.html

Official Language

The official languages of the Conference are Greek and English. Simultaneous translation will be provided.

Certificate of Attendance

All registered participants will receive a Certificate of Attendance with fifteen (15) CME Credits, from the Secretariat desk, at the closing session of the Conference.

Name Badges

Each participant will receive a name badge upon registration. For their convenience, participants are requested to wear their badge at all times during the Conference.

Exhibition Hall

The commercial exhibition will be held at the Porto Heli Conference Center of the AKS Hotel. Access to the exhibition is free for registered participants. The exhibition will be open on 7 - 9 July 2011, during Conference hours.

Registration Fees

Up to 31 st May 2011	200€
June 1 st , 2011 - On site	250€

(a 23% VAT will be added to the above mentioned prices)

Registration fees cover:

- Access to the scientific sessions/workshops and exhibition
- Meeting material (bag, book of abstracts, certificate of attendance)

Acknowledgements

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Αξιόπιστες λύσεις για κάθε ασθενή



ΠΕΡΙΛΗΨΗ ΧΑΡΑΚΤΗΡΙΣΤΙΚΩΝ ΤΩΝ ΠΡΟΪΟΝΤΩΝ Humalog KwikPen, Humalog Mix25 KwikPen, Humalog Mix50 KwikPen

ΟΝΟΜΑΣΙΑ ΤΟΥ ΦΑΡΜΑΚΕΥΤΙΚΟΥ ΠΡΟΪΟΝΤΟΣ: Humalog 100 U/ml KwikPen, ενέσιμο εναιώρημα, Humalog Mix25 100 U/ml KwikPen, ενέσιμο εναιώρημα & Humalog Mix50 100 U/ml KwikPen, ενέσιμο εναιώρημα. **ΠΟΙΟΤΙΚΗ ΚΑΙ ΠΟΣΩΤΙΚΗ ΣΥΝΘΕΣΗ:** Ένα (1) ml διαλύματος περιέχει 100U (που αντιστοιχούν σε 3,5 mg) ινσουλίνη lispro (παράγεται από βακτηρίδια E. coli με την τεχνολογία του ανασυνδυασμένου DNA). Η Humalog Mix25 περιέχει 25% ινσουλίνη lispro και 75% εναιώρημα πρωταμικής ινσουλίνης lispro, ενώ η Humalog Mix50 50% και 50% αντίστοιχα. Κάθε φυσιολογικό περιεχόμενο 3 ml που αντιστοιχούν σε 300U ινσουλίνης lispro. **Θεραπευτικές Ενδείξεις:** Για τη θεραπεία ενήλικων και παιδιών με σακχαρώδη διαβήτη, στους οποίους απαιτείται χορήγηση ινσουλίνης για τη διατήρηση της φυσιολογικής ομοιοστασίας της γλυκόζης. Η Humalog ενδείκνυται επίσης για την αρχική σταθεροποίηση του σακχαρώδους διαβήτη. **Δοσολογία και τρόπος χορήγησης:** Η δοσολογία πρέπει να καθορίζεται από τον ιατρό ανάλογα με τις ανάγκες του ασθενούς. Η Humalog, Humalog Mix25 & Humalog Mix50 μπορούν να χορηγηθούν αμέσως πριν τα γεύματα ή μετά τα γεύματα όταν κρίνεται απαραίτητο. Τα σκευάσματα Humalog πρέπει να χορηγούνται υποδορίως ή με αντλία συνεχούς υποδόριας έγχυσης και παρόλο που δεν συνιστάται, είναι δυνατόν να χορηγηθούν και με ενδομυϊκή ένεση. Εάν κρίνεται απαραίτητο, η Humalog μπορεί να χορηγηθεί και ενδοφλέβια, για τον έλεγχο των επιπέδων της γλυκόζης αίματος σε καταστάσεις όπως κατόχληση, σοβαρή νόση ή σε προ-ή μετεγχειρητικές περιόδους. Οι Humalog Mix25 & Humalog Mix50 πρέπει να χορηγούνται μόνο με υποδόρια ένεση. **Αντενδείξεις:** Υπερευαίσθηση στην ινσουλίνη lispro ή σε κάποιο από τα συστατικά της. **Αντενδείξεις ενέχυσης:** Η υπηλίκωση είναι η συχνότερη ανεπιθύμητη ενέργεια κατά την ινσουλινθεραπεία διαβητικού ασθενούς. Τοπικές αλλεργικές αντιδράσεις είναι συχνές (1/100 έως <1/10). Ερυθρότητα, οίδημα και κνησμός μπορεί να εμφανιστούν στο σημείο της ένεσης της ινσουλίνης. Τα συμπτώματα αυτά συνήθως υποχωρούν σε μερικές ημέρες έως μερικές εβδομάδες. Η συστηματική αλλεργική αντίδραση, η οποία είναι σπάνια (1/10.000 έως <1/1000), είναι δυνατόν επικίνδυνη. Στην περίπτωση αυτή υπάρχει γενικευμένο εξάνθημα σε όλο το σώμα, δύσπνοια στην αναπνοή, δύσπνοια (συστακτικό τίμπος), υπόταση, ταχυκαρδία και επιφύσεις. Η συστηματική αλλεργική αντίδραση μπορεί να είναι επικίνδυνη για τη ζωή του ασθενούς. Η καταστολή της απάντησης της ένεσης είναι άσχετη (1/1000 έως <1/100). Έχουν αναφερθεί περιπτώσεις οίδηματος με ινσουλινθεραπεία, ειδικά στις περιπτώσεις που ο προηγούμενος ανεπαρκής μεταβολισμός έλεγχο βελτιώθηκε με εντατικοποιημένη θεραπεία με ινσουλίνη. **ΜΟΡΦΕΣ/ΤΙΜΕΣ:** Humalog KwikPen 5X3ml X.T.: 31,93€, N.T.: 27,78€, A.T.: 45,91€, Humalog Mix25 KwikPen 5X3ml X.T.: 32,42€, N.T.: 28,20€, A.T.: 46,61€, Humalog Mix50 KwikPen 5X3ml X.T.: 32,44€, N.T.: 28,22€, A.T.: 46,64€ **ΚΑΤΟΧΟΣ ΤΗΣ ΑΔΕΙΑΣ ΚΥΚΛΟΦΟΡΙΑΣ:** Eli Lilly Nederland B.V., Grootslag 1-5, 3991 BA Houten, Ολλανδία. **ΑΡΙΘΜΟΣ ΑΔΕΙΑΣ ΚΥΚΛΟΦΟΡΙΑΣ:** EU/1/96/007/031, EU/1/96/007/033 & EU/1/96/007/035 για τις Humalog, Humalog Mix25 & Humalog Mix50 αντίστοιχα 5 x 3 ml 100 U/ml KwikPens. **ΗΜΕΡΟΜΗΝΙΑ ΠΡΩΤΗΣ ΕΓΚΡΙΣΗΣ/ΑΝΑΝΕΩΣΗΣ ΤΗΣ ΑΔΕΙΑΣ:** Ημερομηνία πρώτης έγκρισης: 30 Απριλίου 1996. Ημερομηνία τελευταίας ανανέωσης: 30 Απριλίου 2006. **ΗΜΕΡΟΜΗΝΙΑ ΑΝΑΒΕΒΡΗΣΗΣ ΤΟΥ ΚΕΙΜΕΝΟΥ:** Απρίλιος 2011. **Χορηγούνται με ιατρική συνταγή. ΕΠΙΧΟΡΗΓΗΣΗ ΑΠΟ ΤΑ ΤΑΜΕΙΑ:** 100% για τα πλήρη ΠΟΥ σχετικά με τα προϊόντα απευθύνονται στην εταιρεία.



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