letters to the editor

A) BRIEF PAPERS ON RESEARCH AND CLINICAL EXPERIMENTS

Does Carvedilol Reduce Requirements for Laser Photocoagulation in Diabetic Retinopathy to a Minimum?

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Dear Editor,

Diabetic retinopathy is a microvascular complication of diabetes mellitus and its prevalence is closely related to long term diabetes, chronic hyperglycaemia, nephropathy and hypertension.1 It has been shown that a decrease of blood pressure decreases retinopathy progression.² The benefits of hypotensive treatment for microvascular and macrovascular complications can be seen from blood pressure values <130/80 mmHg. To achieve this it is necessary to use combined therapies.¹

Given the close relationship between diabetic retinopathy, diabetic nephropathy and hypertension, we have studied 63 patients with diabetic nephropathy due to diabetes mellitus type 2 (DM2) who came in to our outpatient offices consecutively. The patients had a mean age of 65.24 + 7.47 years, 26 were women and 37 were men. Statistical analysis was carried out with the statistical software package SPSS 15.0 for Windows.

The patients were divided into 2 subgroups: one group that had not received photocoagulation (n = 26) and another group that had received it (n = 37). The clinical characteristics and the number of patients treated with carvedilol in both subgroups can be seen in Table 1.

In the subgroup of patients that had not received photocoagulation, 35.7% were being treated with carvedilol in comparison with 14.3% in the group who had received photocoagulation (p = 0.017). Since there were no differences in their mean blood pressure, it can be inferred that the effect of carvedilol is independent of its hypotensive effect.

Carvedilol has a blocking effect on beta-adrenergic receptors combined with vasodilator action based on the blockage of alfa-1-adrenergic3 receptors. In experimental studies carvedilol has been shown to have protective and antioxidant⁴ effects on perivascular inflammation.5 These effects could, in part, be explained by the results of our study. Although it is probably limited by the number of patients studied, the finding that the percentage of patients who received carvedilol in the subgroup that did not receive photocoagulation is significantly higher than in the group

that did receive photocoagulation should be taken into account when treating patients suffering from diabetic nephropathy. Subsequent studies should evaluate if carvedilol prevents the development of diabetic retinopathy.

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- Standars of Medical Care in Diabetes-2009. Diabetes Care 2009;32(Suppl 1):S13-S61.[Pubmed]
- UKPDS 38: UK Prospective Diabetes Study Group. Tight blood pressure control and risk of macrovascular and microvascular complications in type 2 diabetes. Br Med J 1998;317:703-13.

Table 1. Subgroups of patients with diabetic nephropathy caused by DM2based on whether they had or not received photocoagulation

	No photocoagulation (n = 26)	Photocoagulation (n = 37)
- Sex (male/female)	11/15	15/22
- Age (years)	66.92 + 8.03	64.83 + 7.25
- BMI (kg/m²)	31.09 + 8.20	31.26 + 7.93
- Time of evolution		
of DM2 (years)	14.48 +6.51	17.78 +8.41ª
- HbA _{1c} (%)	7.25 + 1.17	7.68 +1.79
- ClCr (ml/min)	70.67 + 55.59	57.62 + 21.31°
- MBP (mmHg)	95.31 + 7.58	92.83 + 8.48
- Proteinuria/24 h (n = 14) ^b	1.16 + 1.44	0.95 + 1.42
- Patients with treatment		
with ACEI/ARA-II	5/11	6/22
- Patients with treatment		
combined ACEI + ARA-II	9	9
- Yes/No treatment with carvedilol	6 (85.7%)/ 20 (35.7%)	1(14.3%)/ 36 (64.3%) ^c

 $^{^{\}circ}$ p <0.005 (Student t test for comparison of independent simple mean values)

^b 49 patients did not have microalbuminuria or proteinuria.

^cFisher exact statistical test for the comparison of percentages, bilateral and unilateral exact significance: p = 0.017.

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- Hansson L, Himmielmann A. Carvedilol in the treatment of hypertension-a review of the clinical data base. Scand Cardiovasc J Suppl 1998;47:67-80.[Pubmed]
- Huang H, Shan J, Pan X, Wang H. Carvedilol protected diabetic rat hearts via reducing oxidative stress. Univ Sci B 2006;7(9):725–31.
- Vailati Mdo C, Rocha NS, Matsubara LS, et al. Protective effects of carvedilol on systemic vascular damage induced by

angiotension II: organ-especific effects independent of antihypertensive effects. Med Sci Monit 2010;16(1):6-10.[Pubmed]

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B) BRIEF CASE REPORTS

Renal amyloidosis in common variable immunodeficiency

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Dear Editor:

Common variable immunodeficiency (CVID) is the most prevalent symptomatic primary antibody deficiency, characterized by hypogamaglobulinemia, normal or decreased B-cell number and impaired antibody response leading to chronic and recurrent infections, mostly in the respiratory and gastrointestinal tracts^{1,2}. However, a significant proportion of patients manifest features of immune dysregulation, including polyclonal lymphocytic infiltration, autoimmunity, enteropathy and malignancy³.

Secondary amyloidosis is an extremely rare complication of CVID⁴, mostly reported in middle aged males⁵⁻⁷. This manifestation refers to the extracellular tissue deposition of serum amyloid A (SAA) protein fibrils with β -sheet structure, which could be due to chronic and recurrent infections in this group of patients⁸. The selfassembly by amyloid proteins cannot progress in the soluble condition of dissembled precursor proteins alone, while it is speeded up by seeding with preformed amyloid fibrils9 which described as «seeding mechanism». Also, enzyme inhibitory function against SAA proteins was confirmed in AA type of amyloid formation and deposition¹⁰. All reported CVID cases with amyloidosis had a sever status of infectious disease or underling complications like cor pulmonale, congestive hepatomegaly, bilateral bronchiectasis, severe respiratory failure7 and tuberculosis6. Recurrent infections could he considered as the main cause of the amyloidosis development; although recurrent infections could be as a consequence of inadequate IVIG therapy, long delay diagnosis can also prone patient to chronic and recurrent infections7.

We report herein a 50-year old male with a history of recurrent respiratory tract infections and diarrhea from early childhood. The diagnosis of amyloidosis was made for this patient based on histopathological findings of renal biopsy, once he hospitalized due to edema and massive proteinuria at the age of 48 years. Renal fine needle aspiration biopsy revealed deposition of amorphous pink hyaline eosinophilic material in glomerulus, tubular basement membrane (TBM), interstitial area and vessel walls of arterioles; it was documented by green appearance fibrils under polarized light which stained and

bind with Congo red (figure 1). As the patient experienced several episodes of infections, immunological studies were performed which showed significant decreased in all serum immunoglobulin levels, compatible with diagnosis of CVID (table 1). Regular hypo-osmolar





Figure 1. Renal glomerule with deposition of amorphous pink material proved to be amyloid by Hematoxyline, Eosin staining (A. X400) and special reacting to Congored stain (B. X400).