

IPERTENSIONE ARTERIOSA 93

FISIOPATOLOGIA DELL'IPERTENSIONE ARTERIOSA (*IPERTENSIONE ARTERIOSA*)
MONITORAGGIO DELLA PRESSIONE AMBULATORIALE (*IPERTENSIONE ARTERIOSA*)
TRATTAMENTO DELL'IPERTENSIONE ARTERIOSA (*IPERTENSIONE ARTERIOSA*)
INFIAMMAZIONE E IMMUNITÀ (*ATEROTROMBOSI*)

**MELATONIN AND OXIDATIVE STRESS IN THE PREVENTION OF
CARDIOVASCULAR DISEASE**

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BACKGROUND. Arterial hypertension, especially if not well-controlled, is one of the main risk factors predisposing to fatal cardiovascular diseases (CVDs). Moreover, the diagnosis of essential hypertension are increasing, therefore oxidative stress and chronic inflammation have also been identified as potential responsible for the development of endothelial damage. Among all the molecules, melatonin (MT) was chosen for its role as a powerful antioxidant and anti-inflammatory endogenous molecule.

PURPOSE. This trial aims to evaluate the early intervention at the base of the inflammatory and oxidative cascade (that results in the development of hypertension), to restore an oxidative balance leading to positive results even at the endothelial and vascular level using MT in addition to anti-hypertensive therapy.

MATERIALS AND METHODS. The trial is randomized, prospective and monocentric control. We enrolled 23 patients with hypertension in absence of other cardiovascular or autoimmune diseases that could alter the oxidative background, from March 2018 to April 2019 (recruitment period). Patients were randomly assigned to 2 groups: "melatonin group" (in which 16 patients add 1 mg/day of melatonin for a year, to their already settled therapy), and a "control" group (consisting of 7 patients with no changes in their therapy). The average follow-up was 1 year from randomisation. Patients were evaluated before and after a period of 1 year through MT plasma concentration and serum antioxidant capacity (TAC) by specific quantitative ELISA method. Therefore endothelial dysfunction and arterial stiffness were evaluated too (using the non-invasive methods of EndoPAT and SphygmoCor).

RESULTS. In "melatonin group" arterial stiffness index statistically decreased (p 0.022), according to a significant increase in plasma melatonin values (p 0.003) and significant decrease in TAC levels (p 0.041) despite the "control" group. The improvement of endothelial function was not significant (p 0.688). Blood pressure had not a significative improvement too (p 0.401).

CONCLUSIONS. Data obtained could confirm the hypothesis of activation of plasma antioxidant system against a situation of altered oxidative balance. In fact, it is possible to hypothesize a correlation between TAC and arterial stiffness that confirm the antioxidant role of MT. The combination between antihypertensive therapy and antioxidant supplementation is able to improve the vascular stiffness. Data obtained are still preliminary and present some limitations but we can think of proposing this trial as a future basis for other extensive and prolonged studies.