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Immunity participation in the Hypertension Pathology

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Abstract

Research that have led to Immune System participation in the pathology of Hypertension are relatively new. Association between HTA and renal disease was reported on 1879 and the first description of autoimmunity as producing morbid situation appeared on 1904 when the antibodies description causing hemolytic anemia in the crio hemoglobin paroxysmic where the end of the stated situation by Paul Erlich who established that the organism didn't hurt himself (horror autotoxicus) Pioneer observations about participation in experimental models in HTA started to re appear at the end of the last century , however it has been in the last few decades when an increasingly number of researches have led to stablish in an unmistakable manner the critical role of autoimmunity in the complex etic pathogenic mechanism which results in the elevation of HTA

Inflammation as manifestation of the immune reactivity of hypertension arterial

Inflammation as a demonstration of the immune reactivity of Hypertension Arterial. It is very well established that the kidneys inflammation, in the arterial wall and the central nervous system helps development and increases severity of the hypertension.

The potential hypertensive of the inflammation in these target organs has been demonstrated in the studies in which the inflammation has been reduced with a range of immuno suppressors treatments which result in the prevention or improvement of hypertension in practically all of the hypertense ceps of mice. Even more, the experimental induction of the renal inflammation is associated with the increase of the arterial hypertension

Renal inflammation induces hypertension as a result of the reduction of the natriuresis by pressure, which is the response of the renal adaptation to a sodium positive balance. The reduction of the natriuretic response by the increase of the pressure of renal perfusion which is caused by the tubule interstitial inflammation of the release of oxidative stress with reduce of nitric oxide, increase of the angiotensin activity and the effects profibrotic with losses peritubular capillary. In the arterial wall, inflammation increases the local produce of reactive species of oxygen, increases the vasoconstrictor tone and suppresses the endothelial vasodilatation response. In the central nervous system, the inflammation of the areas in the third ventriculus helps lymphocyte migration to the arterial wall (originator of the vascular inflammation) and stimulates the activity of the sympathetic nervous system which carries not only the increase of the vasoconstrictor tone, cardiac expense and the reabsorption tubular of sodium, but also, induces the stimulation of various aspects of the immune system.

Lymphocytes participation in the pathology of hypertension arterial

Lymphocytes paper in the pathology of hypertension was initially found in pioneer experiments of Svendsen, who demonstrated that the malnourished mouse didn't develop hypertension depending of salt in the model DOCA (deoxycorticosterone). Hypertension development capacity in this phase of the experimental model was recovered with the lymphocytes transfer. Guzik et al. demonstrated that induced hypertension by angiotensin II was surprised in mice Rag 1 -/-, lacking lymphocytes, and that the response to angiotensin was restored with the adoptive transfer of lymphocytes T. same resistance to hypertension was demonstrated in mice Dahl nulls for Rag 1. Lymphocytes B paper was demonstrated by Chan et al., who used mice BAFFR -/- (lacking B cell activation factor-receiver).

Biography

Irene Burguillo, Clinical Nurse Specialist with over 17 years of experience. Born in Barcelona in 1981, with a Bsc Degree in Nursing from UEX, graduated in 2003. Master´s Degree in Occupational Health and Safety and Nursing prescribing course among several postgraduate education. Having worked in Spain, UK and Qatar in the private sector at Qatar Airways, Optima Health among others and in the public sector in the NHS. Moved into Management as Team Manager, Service Manager and Director of Nursing. Excellent leader and coach. Able to create and manage projects and services, creating policies and procedures delivering high quality care. Expert in Occupational Health, primary care and community nursing. Broad experience delivering conferences, health care, health promotion, case management, health surveillance, service management, and risk management. Enthusiastic, motivated, creative, analytical, visionary, empathic always with a positive attitude making an amazing contribution in every role.

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