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Clinical aspects of melatonin in the acute coronary syndrome.

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This review considers the actions of an endogenously produced molecule, melatonin, on heart diseases. Recent research has shown that inflammation plays a key role in coronary heart disease (CHD) and other manifestations of atherosclerosis. Immune cells dominate early atherosclerotic lesions, their effector molecules accelerate progression of the lesions and activation of inflammation can elicit acute coronary syndromes (ACS). Scientific evidence from the last 15 years has suggested that melatonin has positive effects on the cardiovascular system. The presence of vascular melatoninergic receptor binding sites has been demonstrated; these receptors are functionally linked to vasoconstrictor or vasodilatory effects of melatonin. It has been shown that patients with CHD have a low melatonin production rate, especially those with higher risk of cardiac infarction and /or sudden death. Similarly to other organs and systems, the cardiovascular system exhibits diurnal and seasonal rhythms, including those in the heart rate, cardiac output and blood pressure. The suprachiasmatic nuclei of hypothalamus and, possibly, the melatoninergic system modulate the cardiovascular rhythms. The melatonin attenuates molecular and cellular damages resulting from cardiac ischemia/reperfusion in which destructive free radicals are involved. Anti-inflammatory and antioxidative properties of melatonin are also involved in the protection against vascular disease, i.e. atherosclerosis. The current brief summary of the literature provides an overview on the role of melatonin in the ACS.

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