Insomnia after myocardial infarction

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The heart and the brain appear to be even more closely connected than previously imagined. The damaging effects of myocardial infarction are apparently not confined to the heart, but also affect the brain. In fact, infarction seems to cause neuron loss at the level of the brainstem, which leads to insomnia, notably paradoxical insomnia.

Sleep plays a crucial role in post-infarction remission, as demonstrated by the team of Roger Godbout, Ph.D., his colleague Guy Rousseau and their student Thierno Madjou Bah, investigators at the Research Center of the Hôpital du Sacre-Ceur de Montreal, in a new study published today in the scientific journal *Sleep*.

Although insomnia has long been observed following infarction, to date there have been no studies explaining the phenomenon in scientific terms, apart from the stress that is doubtless brought on by the heart attack. "Thanks to this study, we have been able to show that there is indeed a physiological explanation – the death of cells that play a key role in sleep," says the researcher, who is also a full professor in the Department of Psychiatry at the Université de Montréal.

In the two weeks following a myocardial infarction, not only have periods of paradoxical sleep been observed to be less frequent and of shorter duration, but there are fewer cholinergic neurons in the brainstem, which control paradoxical sleep, due to the phenomenon of self-destruction of cells, known as apoptosis.

Treating insomnia to help the heart heal

A previous study also conducted by the team of Godbout and Rousseau demonstrated that myocardial infarction affected the limbic system, a region of the brain that is responsible for mood, which explains the depression frequently observed after heart attacks. "Since depression is frequently accompanied by insomnia, we wanted to verify whether the neurons in the brainstem were also affected," the investigator explained.

As demonstrated in this study, myocardial infarction, in addition to causing depression, is also associated with the release of factors that provoke the inflammation of tissues, including the brain, and specifically the regions that control sleep, notably the paradoxical sleep phase. The particular function of that phase is to activate regions in the brain that are responsible for integrating our emotions. If that is affected, the risk of depression also increases.

Poor-quality sleep is a known risk factor for cardiovascular disease. Since it can affect remission after an infarction, the risk of complications and recidivism rises and a vicious circle may be set in motion.

Godbout says this study illustrates the importance of rapid intervention in the days following the infarction, before the first signs of insomnia and depression are even apparent. He notes that "any preventive, pharmacological or behavioural treatment is certainly a pathway that should be considered."

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