

Hormone shot boosts prospects of quick recovery from heart attack

A single shot of erythropoietin or EPO, a hormone, administered just after a heart attack can drastically reduce the degree of damage to the organ's muscle fibres, according to a study by Japanese scientists.

EPO limits such damage by eliminating apoptosis or programmed cell death. 'We wanted to see if the area of cell death following acute coronary occlusion (partial or complete obstruction of blood flow in coronary arteries) could be reduced by a single dose of EPO,' said H. William Strauss, physician at the Nuclear Medicine Service at Memorial Sloan Kettering Cancer Centre and co-author of the study.

'Although other drugs to inhibit apoptosis have been studied, none appears nearly as effective as a single dose of EPO,' Strauss said.

'Cells deprived of blood quickly begin to die. By administering 99mTc-annexin V, a radiotracer with a high affinity for apoptotic cells, we were able to view the effects of EPO on heart cells immediately following the restriction of blood flow that occurs during myocardial infarction (heart attack),' he said.

In the study, 18 Wistar rats were randomised into two groups. In both groups, arteries were blocked to induce a heart attack; 20 minutes later, they were unblocked, according to a release of the Society of Nuclear Medicine.

Immediately afterward, one group (treatment) received an EPO shot and the other group saline (non-treatment). Both groups were then injected with 99mTc-annexin V, and their hearts were examined using autoradiography to evaluate the distribution of the radiotracer.

In the treatment group, EPO therapy caused a 2.7-fold reduction of tracer accumulation, indicating a reduction in apoptosis (programmed cell death) and, therefore, less damage to heart tissue.

The reduction in damage to the heart was also demonstrated by measurement of regional cardiac function, which was significantly better in the EPO-treated group. These findings suggest that EPO may be useful to prevent long-term heart damage and dysfunction after a heart attack.

Co-authors of the study include Katsuichi Ohtsuki and Tomoki Doue, Kyoto Prefectural University of Medicine, Kyoto (Japan).

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