Effects Of Ischaemic Postconditioning On The Early And Late Testicular Damage After Experimental Testis Ischaemia-Reperfusion

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Ischaemic postconditioning (IPostC) might represent an innovative surgical approach to protect organs from ischaemia and reperfusion injury. We investigated the molecular mechanisms underlying the contrasting effects of IPostC on the early and late damage induced by testicular ischaemia-reperfusion injury (I/R). Testis I/R was induced by occluding the right testicular vessels using a clip. Male rats were divided into the following groups: sham, I/R, I/R+IPostC. In the I/R group, the clip was removed after 60 min of ischaemia, and reperfusion was allowed for 30 minutes, 1 and 30 days. In the I/R+IPostC group, three cycles of 30 sec reperfusion-30 sec ischaemia were performed after 60 min of ischaemia and then reperfusion followed for 30 minutes, 1 and 30 days. Following 30 minutes reperfusion there was an increase of mitogen-activated protein kinases (MAPKs) in I/R rats; after 1 day of reperfusion, IL-6, TNF-a and NF-kB expression were significantly increased; IkB-α expression reduced; and a marked damage in both testes was observed. IPostC inhibited MAPKs, cytokines and NF-kB expression, augmented IkB-α expression and decreased histological damage in testes subjected to I/R. After 30 days of reperfusion, I/R injury activated the apoptosis machinery, caused severe histological damage and reduced spermatogenic activity. By contrast, IPostC did not modify the apoptotic markers, the histological alterations as well as spermatogenic activity following 30 days of reperfusion.
Our data demonstrate that IPostC protects the testis from the early damage induced by I/R injury but it does not protect against the late damage.