[2005] [P2841] Characterisation of an original model of myocardial infarction provoked by coronary artery thrombosis induced by ferric chloride in pig

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Background: Great advances have been made in the prevention of thrombotic disorders by developments of new pharmacological and surgical treatments. Animal models of arterial thrombosis have largely contributed to the discovery and to the validation of original treatments. The purpose of the present work was to develop and validate an original model of acute myocardial infarction provoked in pig by thrombosis of the left anterior descending (LAD) coronary artery induced by topical application of ferric chloride (FeCl3) solution (50% w/v).
Methods: Myocardial infarction, resulting from an occlusive and adherent mixed thrombus formed in the LAD coronary artery, was examined in 10 pigs at macroscopic level using dual staining technique (Evans blue Dye; triphenyltetrazolium chloride) and at microscopic level using conventional histological analyses and immunohistochemical detection of desmin. Biochemical markers (troponin T and ATP), platelet reactivity and standard hemodynamic parameters, such as stroke volume SV, ejection fraction EF, stroke work SW, and cardiac output CO were also evaluated.
Results: Each pig developed LAD coronary artery occlusion, with an average occlusion time of 23.2 ± 1.2 min. In 6 pigs, LAD blood flow linearly decreased to zero, while in the four other animals reperfusion episodes occurred before reaching a complete occlusion within 30 minutes. Estimated as percent of LV mass, risk area was 36.9 ± 2.1%, and mean infarct size was 35.3 ± 2.1%. All animals developed a transmural area of irreversible damage mainly located in the anteroseptal region of the LV. Troponine T level was 0.071 ± 0.031 µg/l at T0 and 0.997 ± 0.106 µg/l at T360. LAD coronary artery occlusion induced a progressive decrease in CO (from 57.8 ± 5.9 ml/sec at baseline to 47.3 ± 4.9 ml/sec at T360, $p < 0.05$), in EF (from 61 ± 8% at baseline to 49 ± 4% at T360, $p < 0.001$), and in stroke work (from 3069 ± 141 mmHg.ml at baseline to 1774 ± 268 mmHg.ml at T360, $p < 0.001$), at unchanged end-diastolic volume. The more progressive development of coronary artery occlusion, as compared to an abrupt ligation, was accompanied by a correspondingly progressive impairment in hemodynamics.
Conclusion: We conclude that this original porcine model of myocardial infarction is quite close to clinical pathophysiological conditions, such as thrombus formation occurring after atherosclerotic plaque rupture. This certainly constitutes a further argument in favour of this model to assess pharmaceutical or mechanical support of an acutely ischemic heart.

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