Right ventricular involvement in anterior myocardial infarction: a translational approach

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Received 7 October 2009; revised 19 February 2010; accepted 12 March 2010; online publish-ahead-of-print 19 March 2010

Time for primary review: 27 days

Aims

The aim of the present study was to evaluate the involvement of the right ventricle (RV) in reperfused anterior ST-elevation myocardial infarction (STEMI).

Methods and results

Left anterior descending (LAD)-perfused area (using thioflavin-S staining after selective infusion in proximal LAD artery, %), infarct size (using triphenyltetrazolium chloride staining, %), and salvaged myocardium (% of LAD-perfused area) in the right and left ventricle (LV) were quantified in a 90-min LAD occlusion 3-day reperfusion model in swine (n = 8). Additionally, we studied, using cardiovascular magnetic resonance, 20 patients with a first STEMI due to proximal LAD occlusion treated with primary angioplasty. Area at risk (T2-weighted sequence, %), infarct size (late enhancement imaging, %), and salvaged myocardium (% of area at risk) in the right and LV were quantified. In swine, a large LAD-perfused area was detected both in the right and LV (30 ± 5 vs. 62 ± 15%, P = 0.001) but more salvaged myocardium (94 ± 6 vs. 73 ± 11%, P = 0.001) resulted in a smaller right ventricular infarct size (2 ± 1 vs. 16 ± 5%, P < 0.001). Similarly, in patients a large area at risk was detected both in the right and LV (34 ± 13 vs. 43 ± 12%, P = 0.02). More salvaged myocardium (94 ± 10 vs. 33 ± 26%, P < 0.001) resulted in a smaller infarct size (2 ± 3 vs. 30 ± 16%, P < 0.001) in the RV.

Conclusion

In reperfused extensive anterior STEMI, a large area of the RV is at risk but the resultant infarct size is small.

Keywords

Right ventricle • Myocardial infarction • Magnetic resonance imaging

1. Introduction

Although the importance of the right ventricle (RV) has been known for many years,1,2 in the field of ST-elevation myocardial infarction (STEMI) attention has been mainly focused on the left ventricle (LV).

The right coronary artery supplies most of the RV.3 As a consequence, studies analysing RV infarction have evaluated those cases resulting from an acute occlusion of this vessel.2–5 Though the anterior wall of the RV is supplied by branches of the left anterior descending coronary artery (LAD)3 and autopsy studies demonstrated that acute LAD occlusion could also provoke a small RV infarction,6,7 this association has been barely analysed so far. More precise characterization of RV involvement should improve our understanding of pathophysiology and permit a more complete assessment of anterior STEMI patients.

Cardiovascular magnetic resonance (CMR) is increasingly used as a standard tool in a comprehensive evaluation not only of the LV6,9 but also of the RV.10 Moreover, in the setting of recent STEMI, a precise quantification of the area at risk, of infarct size, and of the amount of salvaged myocardium is possible using this technique.11–13 Therefore, CMR permits an accurate characterization of the impact of anterior STEMI on the RV. However, this sort of approach has not been carried out so far.

In the present study, we hypothesized that in LV anterior STEMI a significant part of the RV is at risk. We aimed to characterize the area at risk, infarct size, and salvaged myocardium of the RV in two
scenarios: (i) an experimental model of LAD occlusion–reperfusion in swine analysing autopsy specimens and (ii) a series of patients with a large anterior STEMI treated with primary angioplasty and studied with CMR.

2. Methods

2.1 Experimental study

The study was approved by the Animal Care and Use Committee of the University of Valencia and it conforms with the Guide for the Care and Use of Laboratory Animals published by the US National Institutes of Health (NIH Publication No. 85–23, revised 1996). Nine juvenile domestic female pigs weighing 25–30 kg were used. Pigs were sedated using IM 8 mg/kg ketamine and 0.1 mg/kg medetomidine and anaesthetized using a 10 mg/kg/h continuous IV infusion of 2% propofol. Pigs were pre-treated with IV amiodarone (150 mg) and lidocaine (30 mg) to reduce life-threatening arrhythmias and with heparin (3000 U). Pigs were mechanically ventilated using a 50% oxygen gas mixture. Continuous electrocardiographic monitoring of heart rate, rhythm, and ST-segment changes was performed. A 6-F sheath was introduced into the jugular vein to administer drugs and fluids if necessary.

A 6-F sheath was introduced into the right femoral artery to measure blood pressure and to access the LAD. A Judkins right 4 catheter and a standard hydrophilic angioplasty wire were used. Ischaemia was induced by inflating a 2.5 × 10 mm balloon (Abbot Vascular, Santa Clara, CA, USA) at four atmospheres in the proximal LAD. Coronary artery occlusion was confirmed by contrast injection and by electrocardiographic ST-segment elevation. After 90 min, the LAD balloon was deflated and restoration of normal coronary flow was documented by angiography. The animals were allowed to recover. No LAD dissection or sustained coronary closure was detected at reperfusion or at 72 h angiogram.

After 72 h, 20 mL of 4% thioflavin-S solution was selectively infused into the proximal LAD using a 2.8-F microcatheter (Progreat, Terumo, Japan). The hearts were then arrested with potassium chloride and excised.

2.1.1 LAD-perfused area, infarct size, and salvaged myocardium

The LV and RV were sectioned into 5-mm thick short-axis slices. In order to detect thioflavin-S stained areas, each slice was viewed from the apical side under ultraviolet light and photographed. Afterwards, slices were incubated in 2,3,5-triphenyltetrazolium chloride 2% solution for 20 min at 37°C; finally, they were viewed under room light and photographed. Images were digitalized and manual definition of endocardial and epicardial borders of all short-axis slices were carried out offline by an independent experienced investigator using the software package MATLAB 6.5 (The Mathworks, Inc., Natick, MA, USA). A ruler was photographed beside myocardial slices in all images and it was used as a reference for determining areas; this along with the pre-defined slice thickness (5 mm) permitted the calculation of volumes.

The LAD-perfused area, both in the RV and LV, was defined as the percentage of the myocardial volume showing thioflavin-S staining. Infarct size was designated as the percentage of the myocardial volume that failed to stain with 2,3,5-triphenyltetrazolium chloride. The salvaged myocardium was regarded as the percentage of the myocardial volume showing thioflavin-S staining (see Supplementary material online, Figures S1 and S2). Lack of thioflavin-S staining in the core of the infarcted area was interpreted as microvascular obstruction (Figure 1).

Five-micrometre sections from paraffin-embedded biopsy specimens of myocardial samples extracted from the RV and LV infarct, peri-infarct (salvaged), and remote regions were submitted for haematoxylin and eosin stains analysis using optical microscopy. Biopsy specimens were taken from the 5-mm thick short-axis slices immediately after the extraction and slicing of the heart.

2.1.2 Right coronary artery-perfused area

In order to analyse the extent of the RV perfused by the right coronary artery as well as to determine whether the LAD truly renders the anterior wall of the RV, we carried out an additional series of three experiments. Anterior myocardial infarction was provoked following the same protocol described above. At 72 h, 6-F sheaths were introduced into the right and left femoral arteries and the left and right coronary arteries were engaged described above. At 72 h, 6-F sheaths were introduced into the right and left femoral arteries and the left and right coronary arteries were engaged with Judkins right 4 catheters. In order to prevent the entrance of thioflavin-S into the LAD territory, a 2.5 × 10 mm balloon was inflated in the proximal LAD; coronary occlusion was confirmed by contrast injection. After 15 min, 20 mL of 4% thioflavin-S solution was selectively infused into the proximal right coronary artery using a 2.8-F microcatheter. The hearts were then arrested with potassium chloride and excised. The right coronary artery-perfused area, both in the RV and LV, was defined as the percentage of the myocardial volume showing thioflavin-S staining (see Supplementary material online, Figures S1 and S2). The extent of ischaemic RV myocardium during LAD occlusion was regarded as the percentage of the RV myocardial volume not showing thioflavin-S staining.
In this series of experiments, we also monitored LV and RV regional systolic function before and 5 min after LAD occlusion (see Supplementary material online, Figure S3) using the equipment Vivid S5 Cardiovascular Ultrasound Systems (GE Healthcare, WI, USA).

2.2 Study in patients

This investigation conforms with the principles outlined in the Declaration of Helsinki for the use of human subjects. The local Ethics Committee approved the research protocol. Informed consent was obtained from all subjects.

From January 2008 to December 2008, we prospectively included 27 consecutive patients admitted to a tertiary university hospital with a first STEMI due to proximal LAD occlusion and treated with primary angioplasty. Heparin (100 U/kg) was administered to all patients before the revascularization procedure. Patients with contraindications to CMR \( (n = 2) \), death \( (n = 1) \), re-infarction \( (n = 1) \), severe clinical instability \( (n = 1) \), or failed angioplasty [Thrombolysis in myocardial infarction (TIMI) flow grade 0–1, \( n = 2 \)] were not included. Accordingly, the study group comprised 20 patients.

Troponin I (ng/mL) was measured using a highly sensitive immunoassay (Dimension, Dade Behring, Inc., Newark, NJ, USA) upon patient arrival and at 6, 12, 24, 48, and 96 h after reperfusion. The peak value was recorded.

The association of inferior STEMI secondary to right coronary occlusion with the occurrence of RV infarction has been very well established.\(^2\)–\(^5\) In order to confirm this finding we analysed, using the CMR methodology applied in the present study, 10 consecutive patients admitted to our hospital with a first STEMI due to proximal right coronary occlusion and treated with primary angioplasty (see Supplementary material online, Figure S4).

2.2.1 Cardiovascular magnetic resonance

CMR (1.5-T scanner, Sonata Magnetom, Siemens, Erlangen, Germany) was performed \( 4 \pm 1 \) day after STEMI in accordance with our laboratory protocol\(^6\),\(^12\),\(^14\) and current recommendations.\(^15\) Steady-state free-precession sequences were used for cine and first-pass perfusion imaging, a dark-blood T2-weighted short-tau inversion-recovery turbo-spin echo sequence was applied for determining the area at risk (oedema) and a segmented inversion recovery steady-state free-precession sequence was used for late enhancement imaging. Further details regarding the technical approach applied can be consulted elsewhere.\(^6\),\(^12\),\(^14\) All images were acquired by a phased-array body surface coil during breath-holds and were ECG triggered.

CMR studies were analysed offline by an experienced observer blinded to all patient data using customized software (QMASS MR 6.1.5, Medis, Leiden, The Netherlands). The 17-segment model for the LV and the nine-segment model for the RV\(^16\) were applied.

In cine images, end-diastolic volume index (mL/m\(^2\)), end-systolic volume index (mL/m\(^2\)), ejection fraction (%), and left ventricular mass (g/m\(^2\)) were quantified by manual definition of endocardial and epicardial borders of all short-axis slices. A wall motion score ranging from 1 to 4 to describe normokinesia, hypokinesia, akinesia, and dyskinesia, respectively, was assigned to all LV and RV segments; a wall motion score index (WMSI) was calculated as the ratio of the sum of wall motion score over total segments.\(^15\) This same WMSI was applied in the echocardiographic subanalysis in swine.

T2-weighted images were used to quantify the area at risk in the RV and LV. Area at risk was defined as the percentage of ventricular mass with signal intensity two standard deviations above the mean signal obtained in the remote non-infarcted myocardium (posterior wall).\(^11\),\(^12\) Dark areas located in the middle of a region displaying high signal intensity were also included in the area at risk. Increased signal intensity from the blood pool adjacent to the endocardium was excluded (Figure 2).

Figure 2 Cardiovascular magnetic resonance study in patients. Example of a patient with a large anterior myocardial infarction. Left panels: T2-weighted sequence demonstrated a large area at risk (hyperenhanced myocardium, oedema) both in the right (arrowheads) and in the left (arrows) ventricle. Right panels: infarct size in late enhancement imaging (hyperenhanced myocardium, necrosis) was smaller in the right (arrowhead) than in the left ventricle (arrows) as a consequence of more salvaged myocardium. Severe microvascular obstruction (asterisk) was observed in the middle of the left ventricular infarcted area. Abbreviations: a, anterior; LV, left ventricle; p, posterior; RV, right ventricle.
Microvascular obstruction in the infarcted area was defined as the presence of hypoenhancement compared with the remote non-infarcted segments at the end of the acquisition period in first-pass perfusion imaging

Late enhancement imaging was used to define infarct size in the RV and LV. Infarct size was regarded as the percentage of ventricular mass with signal intensity two standard deviations above the mean signal obtained in the remote non-infarcted myocardium (posterior wall). Dark areas located in the middle of a region displaying late enhancement were regarded as zones of severe microvascular obstruction and they were also considered as infarcted tissue (Figure 2). The salvaged myocardium was regarded as the percentage of the area at risk without late enhancement.

In our laboratory, inter-observer variability for all CMR indexes analysed is less than 5%. Specifically, for assessing the reproducibility of T2-weighted and late enhancement imaging in detecting the area at risk and infarcted tissue, respectively, 20 control patients free of ischaemic heart disease were scanned using the same protocol applied in the present study. Images were analysed in a blinded manner by the observer who was not aware whether studies belonged to STEMI or control patients. Area at risk and late enhancement was correctly ruled out in all control cases.

2.3 Statistical analysis

Continuous data were expressed as the mean ± standard deviation and were compared by the unpaired and paired t-test. Dichotomic data were expressed as percentages and were compared by the χ² statistic; the Fisher exact test was used when appropriate. Statistical significance was considered for two-tailed P < 0.05. SPSS 11.0 (SPSS, Inc., Chicago, IL, USA) was used throughout.

3. Results

3.1 Experimental study

LAD occlusion was conducted in nine pigs, one of them died during balloon inflation due to refractory ventricular fibrillation. Experiments were successfully finished in the other eight cases although electrical ventricular fibrillation was needed in four of them during LAD occlusion. No significant complications were recorded over the 72 h reperfusion period.

Thioflavin-S staining (LAD-perfused area) was detected both in the RV and in the LV in all cases; absence of 2,3,5-triphenyltetrazolium chloride staining (infarcted tissue) was detected in four cases in the RV (50%) and in eight (100%) in the LV (P = 0.04). A certain amount of microvascular obstruction was always detected in the core of the infarcted areas (Figure 1).

A large area of the anterior wall of the RV (30 ± 5%) was perfused by LAD. However, owing to a big quantity of salvaged myocardium (94 ± 6% of the LAD-perfused area), the resultant right ventricular infarct size (2 ± 1%) was small (Figures 1 and 3).

In comparison with the RV, the LV displayed a larger LAD-perfused area (62 ± 15%, P < 0.001), less salvaged myocardium (73 ± 11% of the LAD-perfused area, P < 0.001), and a bigger infarct size (16 ± 5%, P < 0.001) (Figures 1 and 3).

Haematoxylin and eosin stains (Figure 4) of the remote areas demonstrated no significant structural alterations. Contraction bands were observed in the peri-infarct (salvaged) areas. In the RV and LV infarct regions generalized cardiomyocyte necrosis and massive infiltration of mononuclear leucocytes migrating outside capillaries and accumulating within interstitial spaces were appreciated.

In order to analyse the extent of the RV perfused by the right coronary artery as well as to determine the presence, extent and location of RV ischaemic myocardium during LAD occlusion we carried out an additional series of 3 experiments in which thioflavin-S was selectively infused into the proximal right coronary artery during simultaneous occlusion of the LAD (see Supplementary material online, Figure S1). On average, 65 ± 8% of the RV and 30 ± 6% of the LV were perfused by the right coronary artery (see Supplementary material online, Figure S2). LAD occlusion provoked ischaemia in 33 ± 5% of the RV, especially in the mid and apical anterior areas, this coinciding with the zones where RV infarction occurred (see Supplementary material online, Figure S1).

In comparison with systolic function at rest, LAD occlusion induced systolic dysfunction (P < 0.05) both in the LV (WMSI 1 ± 0 vs. 1.7 ± 0.1) and in the RV (WMSI 1 ± 0 vs. 1.3 ± 0.1) (see Supplementary material online, Figure S3).

3.2 Study in patients

Characteristics of the study group are displayed in Table 1. Acute occlusion of the proximal LAD was confirmed in all cases. TIMI flow grade 2–3 was restored by means of stent placement. TIMI 3 flow was present in the right and left circumflex artery in all patients.

All cases analysed displayed a certain amount of area at risk in T2-weighted imaging both in the RV and in the LV. Infarcted tissue (late enhancement) was detected in eight cases in the RV (40%) and in 20 (100%) in the LV (P < 0.001). Clinical, ECG, and angiographic characteristics were similar in patients with and without RV infarction (see Supplementary material online, Table S1).

A large area of the anterior wall of RV was at risk (34 ± 13%). Owing to a big amount of salvaged myocardium (94 ± 10% of the area at risk), the final RV infarct size (2 ± 3%) was small (Figures 2 and 3).

In comparison with the RV, the LV displayed more myocardium at risk (43 ± 12%, P = 0.02). However, owing to less salvaged myocardium (33 ± 26% of the area at risk, P < 0.001), the resultant infarct size was bigger (30 ± 16%, P < 0.001) (Figures 2 and 3).

WMSI was slightly altered in the RV (1.2 ± 0.2) but it was more preserved than in the LV (1.7 ± 0.2, P < 0.001).

A subgroup of 10 patients with inferior STEMI secondary to proximal right coronary occlusion was also analysed using the pre-defined CMR protocol (see Supplementary material online, Figure S4). All cases displayed a certain amount of myocardium at risk in T2-weighted imaging both in the RV and in the LV. Infarcted tissue (late enhancement) was detected in five cases in the RV (50%) and in nine patients (90%) in the LV. Baseline and CMR data of this subgroup are displayed in Supplementary material online, Table S2.

4. Discussion

The main finding of the present study is that in the setting of extensive anterior STEMI due to proximal LAD occlusion, a large area at risk exists in the anterior wall of the RV. Owing to a large extent of salvaged myocardium after LAD reperfusion, the resultant infarct size is small.

Although in 1616 Sir William Harvey was the first to describe the importance of RV function, for many years emphasis in cardiology was placed on LV physiology overshadowing the study of the RV. Over the last decades, the importance of RV involvement in STEMI has been recognized. A recent meta-analysis found that RV
Figure 3 Scheme of results in swine and in patients. Left panel: results in swine. Right panel: results in patients. Abbreviations: a, anterior; LAD, left anterior descending artery; LV, left ventricle; p, posterior; RV, right ventricle.

Figure 4 Optical microscopy analyses. Left panels: right ventricle. Right panels: left ventricle. In the infarcted regions, multiple abnormalities were appreciated including generalized cardiomyocyte necrosis and massive infiltration by mononuclear leucocytes. Contraction bands (arrows) were detected in the peri-infarct (salvaged) areas, but structure was much more preserved in these regions. No significant structural alterations were detected in the remote areas. Abbreviations: LV, left ventricle; RV, right ventricle.

Figure 5 The moderator band as a source of collateral coronary flow. Selective infusion of thioflavin-S in the left anterior descending artery stained the anterior wall of the right and the left ventricles. Through the moderator band (arrow), part of the right ventricular posterior wall was also stained (arrowhead). Abbreviations: a, anterior; p, posterior.
The association of anterior LV with RV infarctions has been demonstrated in pathological studies, but evidence is very limited. Clinical data in patients are even scarcer. Using scintigraphy, Tobinick et al. did not detect RV systolic dysfunction in 24 anterior infarction patients. Marmor et al. studied LV and RV systolic function with radionuclide ventriculography in 22 patients with anterior infarction. There was persistent regional and global impairment of LV function but only transient impairment of the RV, suggesting transitory RV stunning after anterior infarction.

Taking into account the limited and controversial data regarding RV involvement in anterior infarctions, we aimed to clarify this issue by means of a translational approach using two models which permit a reliable and complementary assessment: an experimental model of LAD occlusion—reperfusion in swine, analysing autopsy specimens, and a series of patients with a large anterior STEMI treated with primary angioplasty and studied in vivo with CMR (see Supplementary material online, Figure S5). The similarity of anatomico-pathological and CMR images in swine was confirmed in one experiment using, immediately after euthanasia and extraction, the same T2-weighted sequence applied in humans to determine oedema (see Supplementary material online, Figure S6).

### 4.2 Experimental study

The experimental model applied has been solidly validated. Lack of staining with 2,3,5-triphenyltetrazolium chloride is the gold standard for the macroscopic analysis of myocardial infarction in autopsy specimens. Moreover, the selective infusion of thioflavin-S in the proximal LAD allowed us for a clear quantification of the myocardium irrigated by this vessel (LAD-perfused area, at risk during occlusion). In order to avoid coronary thrombosis and to induce the same therapeutic effect expected in patients, heparin (100 U/kg) was administered both in experimental studies in swine and in primary angioplasty procedures in patients.

RV thioflavin-S staining was detected in all experiments and it involved around one-third of the RV, this proving previous anatomical knowledge regarding RV perfusion. In spite of this large quantity of myocardium perfused by LAD, infarction only occurred in four cases (50%) and, on average, affected 2% of the RV myocardium. These results suggest that, although RV infarction can happen during LAD occlusion, the resulting necrosis is small. Optical microscopy analyses confirmed the presence of necrotic tissue as well as of massive inflammatory infiltration in the infarcted areas of the RV and LV anterior walls.

In an additional series of experiments, in which thioflavin-S was selectively infused into the proximal right coronary artery during

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<td>Salvaged myocardium (% of area at risk)</td>
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**Events**

- VF/VFVT within the first 24 h (%)  
  - 2 (10)
- VF/VFVT after the first 24 h (%)  
  - 1 (5)
- Atrioventricular block (%)  
  - 0 (0)
- Atrial fibrillation (%)  
  - 1 (5)
- Death (%)  
  - 0 (0)
- Re-infarct (%)  
  - 0 (0)
- Re-admission for heart failure (%)  
  - 1 (5)
- Follow-up (week)  
  - 50 ± 11

**TIMI**, thrombolysis in myocardial infarction; **VF/VFVT**, ventricular fibrillation-ventricular tachycardia.
simultaneous occlusion of the LAD (see Supplementary material online, Figures S1 and S2), we demonstrated that around one-third of the RV myocardium, especially in the mid and apical anterior areas, became ischaemic. Areas of ischaemia coincided with those regions where RV infarction occurred (see Supplementary material online, Figure S1). These results truly reveal the presence as well as the extent and location of RV ischaemic myocardium during LAD occlusion.

4.3 CMR study in patients

CMR is being increasingly used as a standard tool in the evaluation of RV structure and function and it has become the state-of-the-art imaging technique for evaluating the consequences of infarction, not only on the LV but also on the RV.

In order to confirm the well-established repercussion of inferior infarctions on the RV by means of our CMR approach, we evaluated an additional series of 10 patients with a first inferior STEMI. CMR solidly demonstrated the extent of area at risk, salvaged, and infarcted myocardium in both ventricles (see Supplementary material online, Figure S4 and Table S2). This approach represents an interesting but so far unexplored tool for the understanding of RV involvement in the context of anterior STEMI; this constituted the main objective of our study in patients.

In parallel to the experimental study, T2-weighted imaging revealed that in the clinical setting around one-third of the RV mass is at risk following proximal LAD occlusion. In agreement with previous pathological observations and our own experimental data not all patients displayed RV necrosis: late enhancement was present in 40% of patients and on average it only affected 2% of RV mass. Similar to the experimental results, RV-WMSI was less depressed than LV-WMSI but not totally normal, suggesting a certain amount of RV myocardial stunning following anterior STEMI.

Area at risk, salvaged myocardium, and infarct size of the RV were almost identical in the clinical and in the experimental settings. With respect to the LV, the area at risk was smaller in humans; this could be caused by more distal thrombotic occlusions (close to the mid-LAD) in same cases, whereas in experiments thioflavin-S was selectively infused into the most proximal part of the LAD. The LV infarct size was smaller in experiments, this probably being a consequence of the complete and earlier reperfusion in this scenario.

A number of reasons could underlie the preservation of the anterior wall of the RV myocardium despite transient severe ischaemia of a large LAD-perfused area at risk: (i) its low oxygen consumption. (ii) Its ability to increase oxygen extraction. (iii) In this study, the complete LAD reperfusion probably contributed to preserve RV myocardium. (iv) The extensive collateral system, especially from the moderator band artery, a branch of the first septal perforator that originates from the LAD. Actually, staining of part of the posterolateral wall through this band was uniformly detected in our experiments (Figure 5). However, the fact that around one-third of the RV became ischaemic during prolonged LAD occlusion (as observed in the additional series of experiments), suggests that collateral circulation from the right coronary artery can contribute but it is not the only factor to explain the small RV infarct size provoked by LAD occlusion.

4.4 Clinical implications

Our data strengthen the importance of CMR for comprehensively characterizing the consequences of anterior infarction on the RV. In our study, clinical data and ST-segment resolution were similar in patients with and without RV infarct. The small size of RV infarctions after complete LAD reperfusion along with the fact that this area is overshadowed by the potent LV vectors probably impedes the detection of anterior RV necrosis by means of surface ECG.

The role of small areas of necrosis in the RV in the genesis of ventricular arrhythmias after anterior infarctions is a potentially interesting subject which can only be taken into consideration after the awareness that RV infarction can occur in patients with anterior LV infarcts.

4.5 Limitations

On the basis of our results many questions such as the role of coronary dominance or the presence of right coronary artery disease on RV infarct size, the usefulness of the clinical presentation and of more available non-invasive techniques for the diagnosis of anterior RV infarction or the long-term prognostic implications of concomitant LV and RV anterior infarction might arise. Regarding all these issues, our data are hypothesis generating. Further studies with larger series of patients will be needed to clarify these and other relevant points.

4.6 Conclusions

In reperfused extensive anterior STEMI, around one-third of the RV myocardium is at risk. As a consequence of a great deal of myocardial salvage, the resulting RV infarct size is small. The significant involvement of the RV in large anterior LV infarctions appears as a novel factor to take into consideration for a better understanding of the pathophysiology and the clinical course of patients with anterior infarctions.

Supplementary material

Supplementary material is available at Cardiovascular Research online.

Acknowledgements

We are indebted to Mr Jose Benavent for his assistance in the preparation of samples for histological analyses.

Conflict of interest: none declared.

Funding

This work was supported by the Spanish Government (PI08128 and HERACLES grants) and the Spanish Society of Cardiology (FEC grant).

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