MicroRNA 103 as a potential diagnostic marker and promising therapeutic target for myocardial infarction

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Introduction

MicroRNAs are considered as a small non-coding RNA which regulate expression of gene by preventing the translation including destruction of specific mRNA. Necroptosis is a form of regulated necrosis and is dependent on a signaling pathway involving receptor interacting protein kinase (RIPK). Fas-associated protein with death domain (FADD) is a negative regulator for necroptosis. MicroRNAs play a critical role in the pathogenesis of cardiovascular diseases. The current study aimed to investigate the role of cardiac-specific MicroRNA in acute myocardial infarction (MI) and focused mainly on its influence on cell death.

Methods



12 mice

- **Control group** (Normal saline ,s.c)
- **MI group** (Isoprenaline HCl 100 mg/kg; s.c)

- Blood sampling
- Heart isolation

- **Heart**
  - One half for histopathological examination of heart tissue
  - Other half for analysis of expression level of:
    - miR 103
    - FADD
    - RIPK
    - IL-6
- **Serum**
  - Determination of cardiac troponin-I level

Results and discussion

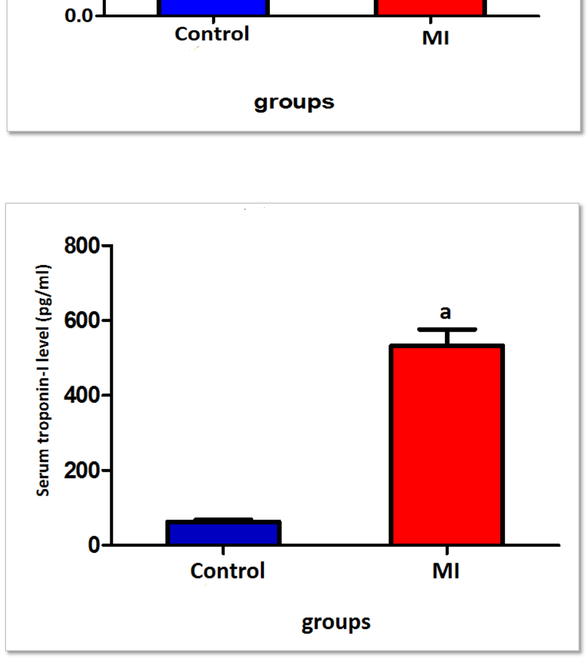
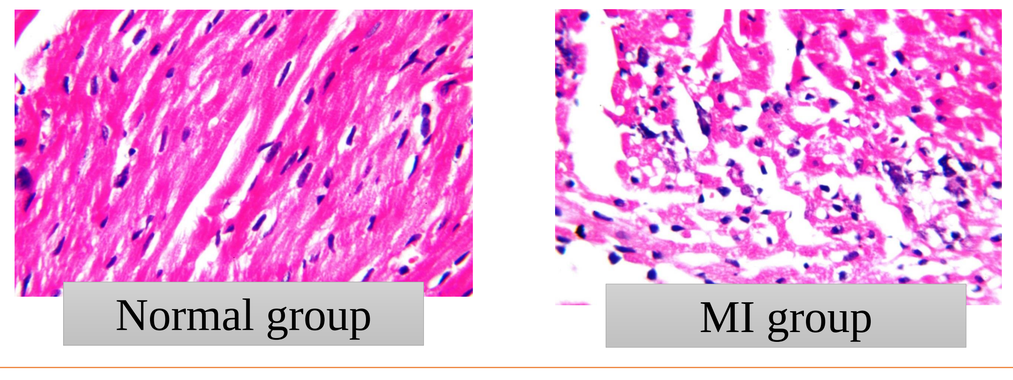


Figure 1: Biochemical analysis of miR103, mRNA expression of FADD, RIPK and IL-6 in heart tissue as well as the significant negative correlation between miR103 and FADD.

Data are presented as mean ± S.E

a: significant difference from control group at $P \leq 0.05$



Normal group

MI group

Figure 1: Histopathological examination:

Inflammatory cells infiltration with myo-fibroblasts proliferation were detected in the vacuolar degenerated fatty changed atrophied myocardium of the ventricles in isoprenaline induced MI group

Conclusion

In conclusion, miR103 and its signaling pathway may be considered novel potential biomarkers for myocardial infarction and its modulation provides a promising new approach for treatment of MI.

References

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