The association between admission hyperglycaemia and the no-reflow phenomenon in STEMI patients undergoing primary percutaneous coronary intervention

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Introduction

No-reflow phenomenon is not uncommon in acute myocardial infarction patients treated by primary percutaneous coronary intervention (PPCI). It is associated with poorer left ventricular systolic dysfunction and higher mortality in such patients.[1] Diabetes was linked to increased incidence of no-reflow in PPCI.[2] We hypothesised that acute admission hyperglycemia, rather than diabetes, is responsible for this complication.

Materials and methods

We prospectively studied 120 consecutive ST-elevation myocardial infarction (STEMI) patients presenting to two PPCI centres over a period of 6 months. We included all the patients eligible for PPCI according to the European Society of Cardiology (ESC) guidelines.[3] We excluded patients with previous PCI and stent thrombosis and patients with previous coronary artery bypass grafting (CABG). The local research ethics committee has approved the study protocol and we followed the Helsinki declaration of research ethics in human beings including informed consents. The patients were divided into two groups based on the coronary flow post-PPCI (normal flow and no-reflow). No reflow is defined as the absence of coronary TIMI 3 flow post PCI without mechanical obstruction.[1] A professional statistician did the analysis using IBM SPSS 21.0 software.

Results and discussion

The incidence of no-reflow was 17.5% (n=21). There was no significant difference between the two groups regarding the clinical characteristics and the different cardiovascular risk factors including diabetes (Table 1). The median of admission random plasma glucose (RPG) level was significantly higher in the no-reflow group (15.5 vs 8.3 mmol/L; p=0.001; Fig 1). A possible explanation is that hyperglycemia increases leucocytes' adhesion molecules, causing microvascular obstruction and Elastase-induced endothelial damage.[4] This augments thrombus formation and impairs ischaemic preconditioning.[5] The study was limited by the small study population size and the narrow geographical area of recruitment.

Table 1. The clinical characteristics of the studied groups

Parameter	Normal flow group	No-reflow group	P value
Number of patients	99	21	
Age, years, mean (standard deviation)	56.3 (10.3)	62.3 (7.9)	0.014
Men, n (%)	75 (75.8)	13 (61.9)	0.19
Non-diabetic, n (%)	62 (62.6)	9 (42.9)	0.094
Diabetic of insulin, n (%)	9 (9.1)	2 (9.5)	1.00
Diabetic of oral diabetic medications, n (%)	28 (28.3)	10 (47.6)	0.084
Hypertension, n (%)	48 (48.5)	7 (33.3)	0.206
Smoker, n (%)	52 (52.5)	9 (42.9)	0.421
Ex-smoker, n (%)	4 (4)	1 (4.8)	1.00
Dyslipidaemia, n (%)	54 (54.5)	15 (71.4)	0.155
Family history of ischaemic heart disease, n (%)	17 (17.2)	2 (9.5)	0.521
Previous acute coronary symdrome, n (%)	19 (19.2)	3 (14.3)	0.762
Absence of pre-infarction angina, n (%)	56 (56.6)	15 (71.4)	0.208

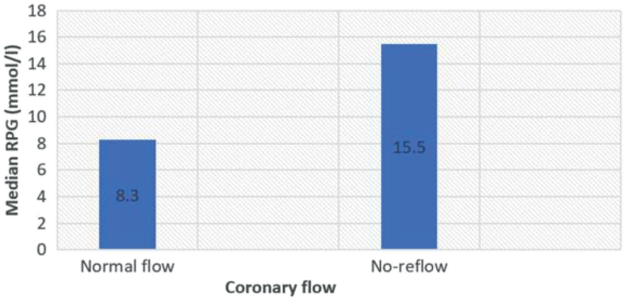

Fig 1. The admission random plasma glucose level in the studied groups.

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Conclusion

Admission hyperglycemia, rather than diabetes, is associated with a higher incidence of no-reflow post-PPCI. The control of admission hyperglycaemia can help to reduce the peri-procedural complications of PPCI. ■

References

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