Atherosclerotic lesions, characterized by hemorrhage, inflammation, fatty streaks, and plaque formation, are commonly seen in the aorta and the coronary arteries. In the coronary arteries, PLA2 plays a critical role in the development of atherosclerotic lesions, which can lead to adverse cardiovascular events. In this study, we used an in vitro model of human aortic smooth muscle cells to investigate the effects of PLA2 on the plaque formation process. Our results showed that PLA2 significantly increased the expression and activity of matrix metalloproteinases (MMPs), which are key enzymes involved in the degradation of extracellular matrix components. These findings suggest that PLA2 may contribute to the development of atherosclerotic lesions by inducing MMP expression and activity.
and echocardiographic data. Adequate recordings of coronary flow for the assessment of CFR were obtained from all patients. Interobserver and intraobserver variabilities for the measurement of Doppler velocity recordings were 5.0% and 3.9%, respectively. There were no correlations between CFR and body mass index, blood pressure, heart rate, left ventricular mass, fasting blood sugar, HbA1c, and lipid profile. On the other hand, CFR was inversely correlated with age ($r = -0.34$, $p < 0.05$) and serum ADMA concentration ($r = -0.57$, $p < 0.01$) (fig 1). Multiple regression analysis to assess independent associations of CFR (with age, sex, body mass index, blood pressure, heart rate, left ventricular mass, fasting blood sugar, HbA1c, lipid profile, and ADMA) showed that only serum ADMA concentration was independently associated with CFR ($\beta = -0.41$, $p < 0.05$).

**DISCUSSION**

The present study shows that in patients with NIDDM serum ADMA concentration and CFR are inversely correlated and circulating ADMA and CFR are independently inversely associated. Coronary endothelial vasodilator dysfunction is known to predict long term atherosclerotic disease progression and cardiovascular event rates. Coronary endothelial vasodilator dysfunction has also been reported in patients with NIDDM. This finding indicates that coronary microcirculation cannot be estimated from short term glycaemic control because temporal fasting blood sugar and HbA1c may not necessarily reflect CFR. Therefore, serum ADMA concentration may be related more sensitively than fasting blood sugar and HbA1c, to coronary microcirculation.

In summary, serum ADMA concentration correlates with CFR in patients with NIDDM, independently of temporary glycaemic control. Serum ADMA concentration may be a sensitive marker of coronary microcirculation in patients with NIDDM without evidence of coronary artery disease.

**REFERENCES**