DISTURBANCE IN THE HEMOSTATIC SYSTEM IN RHEUMATOID ARTHRITIS

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Abstract

Disturbance of fibrinolysis is common in rheumatoid arthritis (RA), and it may be associated with the increased cardiovascular risk observed in this population. We aimed to assess coagulation derangement and investigate whether abnormalities are influenced by demographic, inflammatory or metabolic factors in patients with RA. Levels of tissue plasminogen activator (tPA), plasminogen activator inhibitor (PAI-1), fibrinogen, prothrombin fragment 1 + 2 (PF1 + 2), thrombomodulin (TM), protein C and Von Willebrand factor (vWF) were compared between 141 RA patients and 50 healthy hospital controls. Within RA, coagulation factors were assessed alongside several demographic, inflammation and metabolic indicators. RA patients had higher levels of coagulation factors than controls. After correction for age and sex, having RA predicted increased tPA ($B = 0.15$, $P < 0.001$), PAI-1 ($B = 0.21$, $P < 0.001$), fibrinogen ($B = 0.86$, $P < 0.001$), PF1 + 2 ($B = 0.20$, $P < 0.001$), and TM ($B = 0.01$, $P = 0.03$) levels. CRP correlated positively with tPA ($P < 0.05$), fibrinogen ($P < 0.001$), TM ($P < 0.05$), PF1 + 2 ($P < 0.001$) and vWF ($P < 0.001$). Metabolic factors linked with coagulation factors were hypertriglyceridaemia (tPA, $P < 0.05$; PAI-1, $P < 0.05$; protein C, $P < 0.05$) and insulin resistance (tPA, $P < 0.01$; PAI-1, $P < 0.01$; vWF, $P < 0.05$). Imbalance of coagulation and fibrinolytic mechanisms is common in RA and associates with age, inflammation, and metabolic factors. Further studies may determine whether these abnormalities are the consequence of acute inflammation or markers of vascular dysfunction.

Key words: RA inflammation, associates, coagulation.

Objectives: To assess the outcome of ACS in rheumatoid arthritis compared with case matched controls in the context of underlying cardiac risk factors, clinical presentation, and subsequent management.

Methods: 40 patients with rheumatoid arthritis and ACS identified from coronary care admission registers between 1990 and 2000 were case matched as closely as possible for age, sex, classical cardiovascular risk factors, type and severity of ACS, and admission date (±3 months) with 40 controls. A standardised proforma was used for detailed case note review.

Results: Age, sex, other cardiovascular risk factors, and type and severity of presenting ACS were not significantly different between cases and controls. Recurrent cardiac events were commoner in rheumatoid arthritis (23/40, 57.5%) than controls (12/40, 30%) (p=0.013); there were 16/40 deaths in rheumatoid arthritis (40%) v 6/40 (15%) in controls (p=0.012). Recurrent events occurred earlier in rheumatoid arthritis (log rank survival, p=0.05). Presentation with chest pain occurred in all controls compared with 33/40 rheumatoid patients (82%) (p=0.006); collapse occurred in one control (2.5%) v 7/40 rheumatoid patients (17.5%) (p=0.025). Treatment during the ACS was not significantly different in the two groups.

Conclusions: Recurrent ischaemic events and death occur more often after ACS in rheumatoid arthritis. Atypical presentation is commoner in rheumatoid arthritis. There is an urgent need to develop identification and intervention strategies for ACS specific to this high risk group.

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