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Influence of Smoking on Deep Vein Thrombosis after Myocardial Infarction

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Summary

As part of a study of the factors affecting the risk of deep vein thrombosis after myocardial infarction a surprising and unexplained finding was that non-smokers had a significantly higher incidence of thrombosis than cigarette smokers.

Introduction

Deep vein thrombosis, as detected by the radioactive fibrinogen test (Kakkar *et al.*, 1970), occurs in about 35% of patients after myocardial infarction (Murray *et al.*, 1970). The incidence is higher in older patients and those with varicose veins (Maurer *et al.*, 1971) and in the more severely ill (Nicolaides *et al.*, 1971). We report here the results of a prospective study of the factors affecting the risk of leg vein thrombosis after myocardial infarction.

Patients and Methods

The patients studied were those entering the coronary care unit of Queen Mary's Hospital, Roehampton, with a clinical diagnosis of myocardial infarction. About half constituted the control groups of two studies of prophylactic heparin therapy (Handley *et al.*, 1972; Handley, 1972), the remainder being consecutive admissions to the unit. The patients received an intravenous injection of ^{125}I-fibrinogen within 18 hours of admission to hospital, and a scintillation counter was used to examine their legs for the presence of deep vein thrombosis on alternate days for at least two weeks, using the technique described by Kakkar *et al.* (1970). Anticoagulants were not given unless there was ^{125}I-fibrinogen evidence of deep vein thrombosis.

Each patient was questioned about previous episodes of venous thromboembolism and examined for the presence of varicose veins. The severity of the myocardial infarct was assessed by means of the Peel index (Peel *et al.*, 1962). Smoking habits were recorded using the following definitions: (*a*) regular smoking—one or more cigarettes a day for at least the past year, with continued smoking up to the month before admission; (*b*) previous smoking—one or more cigarettes a day for at least a year in the past but none for at least a month before admission; (*c*) non-smoking—less than one cigarette a day for any year in the past.

Results

A total of 160 patients were studied. Of these, 45 were excluded because the clinical diagnosis of myocardial infarction was not confirmed by electrocardiographic or serum enzyme changes. Eight patients died within the two-week follow-up period. One had evidence of deep vein thrombosis at the time and necropsy confirmed the cause of death as massive pulmonary embolus; this patient was a non-smoker. A further seven patients were withdrawn from the study before the end of two weeks either because they were given anticoagulants by the clinician in charge of the case or because they were discharged from hospital; none had any evidence of deep vein thrombosis at the time. The remaining 100 patients all completed two weeks of follow-up, at which time the study was concluded. Comparison of the characteristics of the patients who were withdrawn with those of the patients who were studied showed no significant differences (table I).

Patients aged 70 years and over had a significantly higher incidence of deep vein thrombosis than those between 50 and 69 years of age ($P < 0.001$) (table II). The incidence in men (37%) was similar to that in women (33%).

TABLE I—*Comparison of Patients Included in Study with Those Withdrawn for Reasons Given*

	No. of Patients	Mean Age (Years)	Males: Females	Smokers: Non-smokers
Death	8	64·8	5: 3	5: 3
Anticoagulated, or discharged	7	56·0	6: 1	5: 2
Total withdrawals	15	60·6	11: 4 (2·8: 1)	10: 5 (2: 1)
Group studied	100	61·6	73:27 (2·7: 1)	61:37* (1·6: 1)

*Incomplete clinical information on two patients.

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TABLE II—*Relation between Age of Patient and Incidence of Deep Vein Thrombosis (D.V.T.) after Myocardial Infarction*

	Age (Years)			
	<50	50-59	60-69	≥70
No. of patients	8	31	40	21
D.V.T.	1	8	12	15
Incidence	12·5%	25·8%	30·0%	71·4%
		28·2%		

Clinical information was incomplete for two patients, and these are therefore excluded from further analysis. Deep vein thrombosis was significantly more common in patients with varicose veins (64%) than in those without (32%) ($P < 0.025$) (table III). Deep vein thrombosis also occurred more often in patients with a history of previous venous thromboembolism (60%) than in those without (35%), but with the small numbers involved the difference was not significant. The mean Peel index was 9·3 for those who developed deep vein thrombosis and 8·7 for those who did not. The difference is again not significant.

TABLE III—*Incidence of Deep Vein Thrombosis (D.V.T.) after Myocardial Infarction related to Various Risk Factors*

	No. of Patients	Previous Thromboembolism		Varicose Veins		Cigarette Smoking	
		Yes	No	Yes	No	Yes	No
No. of patients	98	5	93	14	84	61	37
D.V.T.	36	3	33	9	27	17	19
Incidence	37%	60%	35%	64%	32%	28%	51%
P value		N.S.		<0·025		<0·025	

A total of 61 patients gave a history of regular cigarette smoking within the month before admission, and most of these continued to smoke up to the day of their infarction. The remaining 37 patients were all non-smokers and none had smoked in the past. Of the 61 regular cigarette smokers 17 (28%) subsequently developed leg vein thrombosis compared with 19 (51%) of the 37 non-smokers. This difference is statistically significant ($P < 0.025$). No account was taken of pipe or cigar smoking. The number of cigarettes smoked did not materially affect the result, heavy smokers (more than 10 a day) having an incidence of thrombosis of 26% and light smokers (up to 10 a day) having an incidence of 33%. This difference is not significant ($P > 0.8$). The proportion of patients who were obese was similar in the two groups. Nineteen of the 61 smokers (31%) and 11 of the 37 non-smokers (30%) were 10% or more above the desirable weight for their height (Documenta Geigy, 1970).

The results for 60 patients aged between 50 and 69 years who had no varicose veins and gave no history of previous thromboembolism are given in table IV. These patients represent a group in whom the other known high-risk factors have been removed. Of the 22 non-smokers 9 (41%) developed deep vein thrombosis compared with 7 (18%) of the 38 smokers. This difference is significant ($P = 0.056$).

TABLE IV— *Incidence of Deep Vein Thrombosis (D.V.T.) after Myocardial Infarction in Patients aged 50-69 Years without Varicose Veins or History of Previous Thromboembolism according to Smoking History*

	Smokers	Non-smokers
No. of patients	38	22
D.V.T.	7	9
Incidence	18·4%	41%*

*$\chi = 3.72$; $P = 0.056$.

Discussion

Our results confirm the finding by others of an increased risk of venous thrombosis after myocardial infarction in patients over the age of 70 years and in those with varicose veins (Maurer *et al.*, 1971). Patients with a history of previous thromboembolism have an increased risk of postoperative deep vein thrombosis (Kakkar *et al.*, 1970), and this also proved to be the case for our patients after myocardial infarction, though statistical significance was not reached. We could not, however, confirm the finding of Nicolaides *et al.* (1971) that the more seriously ill patient is at greater risk, always assuming that a higher Peel index indicates a greater severity.

Hume *et al.* (1970) suggested that smoking may be a factor in the aetiology of venous thrombosis, but Maurer *et al.* (1971) specifically stated that cigarette smoking did not seem to affect the frequency of thrombosis after myocardial infarction. The lower incidence of thrombosis among smokers found in the present study is both surprising and difficult to explain. No patient was allowed cigarettes after admission to hospital, so the answer cannot lie with any acute effect of smoking. Possibly the sudden withdrawal of cigarettes has some effect on the vascular or haematological systems discouraging thrombosis. Alternatively the aetiology of myocardial infarction may differ in smokers as compared with non-smokers, the latter having some constitutional or acquired factor which also makes them more liable to develop venous thrombosis.

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