venous heparin and warfarin, which was given orally for about six months. One patient (case 1) required radiotherapy for a recurrence of a localised lymphoma but thereafter remained free of a recurrence 30 months after starting chemotherapy. Three patients showed no recurrence of lymphoma 14, 18, and 21 months after starting chemotherapy, and the last patient (case 5) was in remission, having recently completed chemotherapy.

Comment

Patients with cancer have an increased risk of thromboses.[2] The mechanisms are obscure but tumours may release unknown substance(s) into the circulation that directly or indirectly activate the coagulation system. This risk may be enhanced by cytotoxic chemotherapy as shown in patients with breast cancer.[3] Of a series of 117 patients with Hodgkin's lymphoma, 10 developed venous thromboses, mostly during cytotoxic chemotherapy,[4] as did our five patients, suggesting a causal role for chemotherapy. Support for vascular toxicity induced by chemotherapy was suggested by an in vitro model, which showed endothelial damage by a variety of cytotoxic drugs.[5] Alternatively, the presence of masses from lymphomas in the lungs or mediastinum could impair venous return and induce stasis, thereby enhancing an underlying predisposition to thromboses.

Despite the uncertainty about the cause of thromboses and thromboemboli in our patients their early recognition and treatment can lead to long term survival when the underlying lymphomas are controlled by chemotherapy.

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Fish oil and plasma fibrinogen

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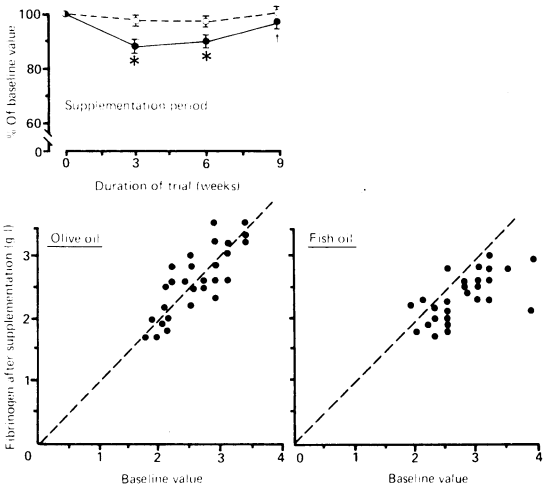
The low morbidity from ischaemic heart disease in people who eat large amounts of fish has been attributed to possible hypolipidaemic and antithrombotic effects of fish oils.[1] Intake of marine oils has been shown to lower plasma triglyceride and cholesterol concentrations, decrease thrombocyte aggregability, and increase the bleeding time.[1] As plasma fibrinogen is essential for the formation of thrombus and the risk of coronary disease is positively correlated with the plasma fibrinogen concentration,[2] we decided to see whether fish oils might possibly affect plasma fibrinogen concentrations.

Case report

Sixty four men aged 35-40 were randomly assigned to two equal groups. In a double blind trial one of the groups was given for six weeks 14 g fish oil concentrate daily (as 1 g capsules) containing 25·7% eicosapentaenoic acid (C20:5, n-3) and 20·5% docosahexaenoic acid (C22:6, n-3; Apothekernes Laboratorium AS, Norway). The other group was given 14 g olive oil daily as 1 g capsules, also for six weeks. Compliance was good, as judged from plasma fatty acid responses. Some men reported belching as a minor problem. There were five dropouts in the fish oil treated group and three in the olive oil treated group. Citrated plasma for measurement of fibrinogen concentrations was obtained before the trial (zero time) and after three, six, and nine weeks. Plasma fibrinogen value (as thrombin coagulation time[1]) was estimated by an automated method.

Mean plasma fibrinogen concentrations in the fish oil and olive oil treated groups at zero were 2·73 and 2·66 g/l. After three weeks in men taking the fish oil concentrate there was a significant decrease (13·2%; $p<0·05$) in plasma fibrinogen concentrations, but no further change was seen three weeks later (figure). Three weeks after stopping fish oil supplementation plasma fibrinogen concentrations had returned almost to initial values. In men taking olive oil capsules there was no significant change in plasma fibrinogen concentrations. To see whether the effect of the fish oil was related to initial plasma fibrinogen concentrations baseline fibrinogen values for each group were plotted against individual mean values—that is, at three and six weeks—after supplementation (figure). By analysis of covariance no such relation could be found.



Upper panel: Change in plasma fibrinogen concentrations in two groups of men taking dietary supplements of fish oil (●) or olive oil (○). (Points are means. Bars are SE.) Lower panels: Individual mean plasma fibrinogen concentrations—that is, at three and six weeks—after supplementation with olive oil and fish oil plotted against baseline values.
**Compared with zero time $p<0·05$ (paired samples t test).*
†Compared with three weeks $p<0·05$

There was a weak but significant positive correlation between smoking and plasma fibrinogen concentration ($r=0·260$; $p<0·01$). No difference in the effect of fish oil on plasma fibrinogen concentration between smokers and non-smokers could, however, be detected.

Comment

To our knowledge this is the first report that the plasma fibrinogen concentration may be reduced by dietary fish oils. Our results raise the question whether the suggested antithrombotic effect of fish oils[1] might be related to a lowering of plasma fibrinogen. The reported lack of a fibrinogen lowering effect of cod liver oil[4] may be related to the lower dose of n-3 fatty acids

provided by the amount of cod liver oil used (20 ml/day). Our findings confirm those of Kannel *et al* that smoking is associated with increased plasma fibrinogen concentrations.[2]

We have no data elucidating the mechanism of action of fish oils on plasma fibrinogen. Studies are in progress to investigate whether there might be direct or indirect effects of long chain n-3 fatty acids on hepatocyte fibrinogen production. In line with this, we speculate to what extent supplementation with long chain n-3 fatty acids may fine tune monocyte macrophages to modulate their hepatocyte stimulating activities, as it is well known that n-3 polyunsaturated fatty acids affect production of monocyte effector molecules—for example, platelet activating factors acether.[5]

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Obesity and postoperative complications of abdominal operation

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Although severely obese patients do present special problems at operation,[1,2] it is not clear whether moderate obesity significantly increases the incidence of postoperative complications and, if so, whether preoperative weight loss reduces the risk. We therefore carried out a prospective study to see whether moderate obesity was associated with increased postoperative morbidity.

Patients, methods, and results

Given that about 15% of patients are obese and that the incidence of a particular complication is about 25% in non-obese patients, a total of 500 patients would give an 80% chance of detecting an increase in incidence of 15% or more—that is, an incidence of 40% or more in obese patients. Over five years we recruited 473 patients (176 men and 297 women) of whom 31 men (18%) and 42 women (14%) were obese according to a prespecified definition of a Quetelet index (weight/height^2) of 27 or more for men and 30 or more for women. Apart from a few eligible patients who could not be included for various reasons, those recruited formed a consecutive series.

Of the 473 patients, 369 (78%) had cholescystectomy, 54 (11%) vagotomy, 21 (4%) gastrectomy, and 29 (6%) small or large bowel resections (initially, only patients having cholescystectomy were included.) Preoperatively, patients were weighed, their height was recorded and skinfold thickness measured with Holtain calipers at triceps, subscapular, and suprailiac sites. At operation the depth of subcutaneous fat was measured by the surgeon. Patients were then seen on the second, fourth, and sixth postoperative days and reviewed at an outpatient visit six weeks after operation. Sepsis of the wound and drain site was divided into three categories of severity: serous discharge, superficial infection (stitch abscess), and discharge of pus. Other possible complications included deep vein thrombosis, pulmonary embolism, chest and urinary infections, and unexplained fever.

Complete details were available for 469 of the 473 patients. The table shows that there was a significant excess of wound sepsis in the obese of both sexes, being possibly more noticeable in the women. The incidence of wound sepsis was 25% (98/396) in non-obese and 43% (31/73) in obese patients, an increase of 18% (95% confidence interval 5% to 30%) in obese patients. (In those who had a cholecystectomy the difference was confined to women.) Similar results were obtained with skinfold thicknesses. No differences with obesity were found in the incidence of deep vein thrombosis and pulmonary embolism, chest and urinary tract infections, unexplained fever, or other complications reported at the outpatient visit. Seven patients, one of whom was obese, died within six weeks after operation.

Incidence of wound sepsis in 469 patients. Values are numbers (percentages) of patients

	Men		Women		Total	
	Not obese (n=144)	Obese (n=31)	Not obese (n=252)	Obese (n=42)	Not obese (n=396	obese (n=73)
Well healed	98 (68)	19 (61)	200 (79)	23 (55)	298 (75)	42 (58)
Serous discharge	36 (25)	5 (16)	35 (14)	12 (29)	71 (18)	17 (23)
Stitch abscess	1 (1)	2 (7)	3 (1)	1 (2)	4 (1)	3 (4)
Pus discharge	9 (6)	5 (16)	14 (6)	6 (14)	23 (6)	11 (15)

Men: $\chi^2=9{\cdot}08$, df=3, p=0·028.
Women: $\chi^2=12{\cdot}14$, df=3, p=0·007.
Both sexes: $\chi^2=14{\cdot}87$, df=3, p=0·002.

Comment

We found an increased risk of postoperative morbidity in obese patients only for the occurrence of wound infection. If knowledge of a patient's obesity had resulted in a tendency to overreport complications in obese patients, a difference in complications other than wound sepsis would also be expected.

Despite the increase in wound infection in obese patients we conclude that the degree of increased morbidity associated with moderate obesity is so small that it would not be practicable to study the benefit of planned preoperative weight loss in moderately obese patients and that probably little is to be gained in attempting preoperative weight loss in this group of patients.

We thank colleagues at Northwick Park for permission to include their patients in the study.

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