

SHORT REPORTS

Tromsø Heart Study: vitamin D metabolism and myocardial infarction

The high mortality rates from myocardial infarction in northern Norway lead to the establishment of the Tromsø Heart Study (THS) in 1974.¹ An investigation by Lindén,² suggested that a high intake of vitamin D might be a precipitating cause of myocardial infarction in this area of Norway. We therefore studied data from the THS to determine whether vitamin D was a risk factor for myocardial infarction.

Patients, methods, and results

In 1974-8, 30 participants in the THS¹ developed myocardial infarction; 23 had been free from any known disease at the start of the study. For each of these 30 patients we selected two controls of the same age (within a year) who had had serum samples taken in the same month as the patient.

In 1978 deep-frozen serum samples from the THS were thawed and the concentration of 25-hydroxy vitamin D (25-OHD) measured, principally as described by Bouillon.³ Calcium and magnesium were analysed by atomic absorption spectrophotometry and albumin and phosphate by standard colorimetric procedures. Group specific component (Gc protein), which is the vitamin-D binding protein, was assayed by immunoprecipitation in a Gemaec fast analyser.⁴

The table shows mean values of some of the risk factors and of 25-OHD and Gc protein in the 23 patients free of disease at the start of the study and their controls. The observed differences were largely unchanged when all 30 patients and their 60 controls were compared.

Mean concentrations of 25-OHD and Gc protein measured in 1978 and risk factors recorded at the start of the Tromsø Heart Study in 23 men in whom myocardial infarction was diagnosed during the following four years and in 46 male controls matched according to age and month of blood sampling

	Patients		Controls		Significance† (P)
	Mean	SD	Mean	SD	
25-OHD (nmol/l)	59.0	24.1	63.4	27.2	
Gc protein (mg/l)	315	69.3	283*	82.9	
Cholesterol (mmol/l)	8.12	1.95	7.41	1.38	<0.05
Triglycerides (mmol/l)	2.13	1.06	1.38	0.78	<0.0025
Blood pressure (mm Hg):					
Systolic	133.8	19.7	128.8*	15.4	
Diastolic	82.7	11.2	80.6*	10.5	
Smokers (%)	91		83		

*n = 45.

†Unpaired Student's *t* test.

Conversion: SI to traditional units—Cholesterol: 1 mmol/l ≈ 38.6 mg/100 ml. Triglycerides: 1 mmol/l ≈ 88.5 mg/100 ml.

There were no significant differences between the patients and controls in weight, height, or haemoglobin concentration. 25-OHD concentrations¹ were slightly lower in the patients than in controls, though the difference was not significant. The most striking difference was the non-fasting triglyceride concentration, which was much higher in the patients.

Comment

Consumption of fish and fish oil, and hence of vitamin D, has been assumed to be substantially higher in northern Norway than in other regions. Our results do not support this view, as vitamin D concentrations in both patients and controls were comparable to those in other populations.⁵ We measured 25-OHD, the main circulating metabolite of vitamin D, and methods that measure the 25-hydroxylated metabolites of cholecalciferol and ergocalciferol are generally accepted to reflect the overall vitamin D status of the organism.⁵

Gc protein was higher among the patients than among the controls, though not significantly so. These higher Gc protein values tended to decrease even further the concentrations of free 25-OHD among the patients. Indeed, correcting 25-OHD concentration for Gc protein

concentration produced a statistically significant difference between the groups ($P=0.024$), with the patients having a lower concentration of free 25-OHD.

It has been postulated that vitamin D could influence the incidence of coronary heart disease by raising the serum cholesterol concentration. But at the concentrations found in our study there was no significant correlation between vitamin D status and the serum cholesterol concentration (linear regression, least squares method).

The hypertriglyceridaemia among the patients was our most striking finding, and our preliminary suggestion is that the difference between patients and controls was caused by dietary differences. We propose to study triglyceride values further.

The strength of the present study is its prospective nature. We believe that our data are, to a large extent, free from bias caused by seasonal or age-dependent variations or changes caused by a coronary attack. Our results give no reason to suggest that the vitamin D intake or the vitamin D status of the population of northern Norway account for their higher risk of myocardial infarction.

We thank Edith Holstad for technical help.

¹ Thelle, D S, *et al*, *Acta Medica Scandinavica*, 1976, **200**, 107.

² Lindén, V, *British Medical Journal*, 1974, **3**, 647.

³ Bouillon, R, van Kerkhove, P, and Demoor, P, *Clinical Chemistry*, 1976, **22**, 364.

⁴ Blom, M, and Hjørne, N, *Clinical Chemistry*, 1975, **21**, 195.

⁵ Favus, M J, *Medical Clinics of North America*, 1978, **62**, 1291.

(Accepted 21 May 1979)

University of Tromsø, 9001 Tromsø, Norway

TORSTEIN VIK, MD, pathologist, department of clinical chemistry
KENNETH TRY, MD, pathologist, department of clinical chemistry
DAG S THELLE, MD, physician, Institute for Community Medicine
OLAV H FØRDE, MD, physician, Institute for Community Medicine

Remission of a syndrome indistinguishable from motor neurone disease after resection of bronchial carcinoma

Motor neurone disease may occur in association with carcinoma.¹ The association may be coincidental, as malignant disease is common and motor neurone disease occurs in 2.45/100 000 population.² We report a case of bronchial carcinoma presenting as a syndrome resembling motor neurone disease. After pneumonectomy the syndrome remitted.

Case report

A 54-year-old man presented with a six-month history of progressive weakness of the legs and painful knees, tender swelling of the right breast, and haemoptysis. He smoked 40 cigarettes a day. Examination showed clubbing of the fingers and toes and right-sided gynaecomastia. There was slight wasting of the muscles of the shoulder girdle, extensor digitorum brevis, and gastrocnemius and severe wasting of the quadriceps. Tone was increased in the legs, and the quadriceps were fasciculating. Intrinsic hand muscles and finger extensors were weak. There was pronounced weakness of hip flexion, adduction, and abduction, but extension was normal. Dorsiflexion and plantar flexion of the ankles were weak. Brisk reflexes were elicited in the left arm and both knees and ankles. Abdominal reflexes were present and plantar responses were unequivocally extensor.

Chest radiography showed a mass at the left hilum extending into the upper lobe. Bone-scan and other radiological findings were normal apart from minimal degenerative changes in the cervical and lumbar spine. Fiberoptic bronchoscopy showed irregularity and widening of the subcarina between the lingula and left upper lobe. Biopsy yielded neoplastic cells. Cerebrospinal fluid was normal. The right lateral popliteal nerve had a motor conduction of velocity 48 m/s (normal). Electromyography with a central needle electrode in the right vastus medialis and rectus femoris showed no