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PATIENTS WITH CHRONIC PANCREATITIS ARE AT INCREASED RISK FOR OSTEOPOROSIS

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Repeated relapses of acute pancreatitis may result in chronic pancreatitis [1, 2]. There is considerable concern that the incidence of chronic pancreatitis will rise because of an increase in admissions with acute pancreatitis. Our group documented a 54% increase in accident and emergency admissions with alcohol-induced acute pancreatitis over a 7-year period, particularly among younger patients and among females [3].

Chronic pancreatitis is a progressive inflammatory disease in which the pancreas may become increasingly fibrous and strictured, affecting both endocrine and exocrine function. Endocrine deterioration may result ultimately in diabetes, although this is a late feature of the disease. An earlier manifestation is a progressive exocrine decline, causing nutrient malabsorption, as the production of digestive enzymes is decreased.

Maldigestion and malabsorption of carbohydrate, protein, and fat may occur, although the latter is more common and more clinically evident. Fat malabsorption may result in the malabsorption of fat-soluble vitamins; including vitamin D. Vitamin

D is important for bone health, as it is required for the optimal absorption of dietary calcium [4]. These nutrient losses, coupled with poor diet, alcoholism, and heavy smoking render the chronic pancreatitis patient at an increased risk of bone mineral density (BMD) loss, and ultimately osteopathy (osteoporosis and osteopenia).

In 2008, Dujsikova and colleagues [5] found that 5% of chronic pancreatitis patients had osteoporosis, whereas 39% in total had osteopathy including osteopenia, osteoporosis, and osteomalacia. Haaber et al [6] showed that patients with advanced steatorrhea had greater bone loss than those with preservation of exocrine function. Joshi and colleagues [7] showed a high prevalence of low BMD in young Indian patients with tropical calcific pancreatitis. However, there has been a dearth of studies on this topic from Europe and North America.

The economic burden of osteoporosis is significant. The total cost of osteoporotic fractures in England and Wales in 1994 was £742 million [8]. Crucially, osteoporosis is largely a preventable disease with appropriate screening and treatment. For this reason, there are published consensus guidelines for bone protection in other malabsorptive conditions including celiac disease [9] and inflammatory bowel disease [10], which recommend the routine assessment of BMD and the routine supplementation of calcium and vitamin D. In contrast, no bone health guidelines exist for chronic pancreatitis. Moreover, the incidence of osteopathy in a Western European chronic pancreatitis population remains unclear.

The aim of this prospective study was to determine the prevalence of osteoporosis and osteopenia in a Western European chronic pancreatitis population and to determine predictive factors.

MATERIALS AND METHODS

This study was prospective and cross sectional in design. Patients presenting to the Centre for Pancreatico-Biliary Disease, Adelaide and Meath Hospital, incorporating the National Children's Hospital (AMNCH) with a diagnosis of chronic pancreatitis were identified from a prospectively held database and invited to attend for assessment. Eighty-four patients with chronic pancreatitis were identified as eligible for the study between January 2007 and January 2011. Diagnosis of chronic

pancreatitis was made based on standard clinical and radiological data. Patients were classified by disease severity and assigned to one of 3 grades according to the Cambridge classification [11].

Recruitment

Sixty-two (74%) of the eligible patients participated in the study. Twenty-two patients were not included because of repeated failures to attend the clinic or declining to participate.

None of the patients had a history of other malabsorptive condition such as celiac disease or inflammatory bowel disease.

As a control arm, 66 healthy men and women were recruited and matched for age, sex, and socioeconomic status. Controls were not paid and were recruited from staff within our institution, and from local industry and transport facilities. The study was advertised using leaflets and posters and by email to the human resources departments. Controls with malabsorptive conditions or history of gastrointestinal resection were excluded. Ethical approval was granted by the AMNCH/St. James Hospital Joint Ethics Committee. Written informed consent was obtained for all patients and controls.

Assessment

Height and weight were measured, from which body mass index (BMI; kg/m^2) was constructed. Height was measured without shoes using a stadiometer (Holtain Ltd, Crymych, Dyfed, United Kingdom). Weight was measured with subjects wearing light clothes and without shoes using a calibrated Seca 708 weighing scale (Seca, United Kingdom). All measurements were done by a single investigator (S.D.).

Participants were classed by socioeconomic status as follows: completed primary level or secondary level education, or completed third level or higher. Smoking status was recorded at interview for all participants and classed as current, former, or never smokers. For former or current smokers, a mean pack year was calculated. One pack year corresponded to 20 cigarettes per day for 1 year. Alcohol intake was quantified in units of alcohol (1 pint equivalent to 2 units, 1 standard glass

of wine equivalent to 1.5 units, and 1 standard measure of spirits equivalent to 1.5 units).

Of the 62 patients, 46 agreed to and provided a stool sample for the measurement of fecal elastase-1 to assess pancreatic exocrine function. Fecal elastase-1 was measured using an enzyme- linked immunosorbant assay (ScheBo Pancreatic Elastase-1 Stool Test, Germany) and was expressed as microgram per gram (µg/g) stool. Normal pancreatic exocrine function is more than 200 µg/g, mild pancreatic exocrine insufficiency is 100 to 200 µg/g, and severe pancreatic exocrine insufficiency is less than 100 µg/g.

C-reactive protein was measured by immunoturbidimetric assay, and parathyroid hormone (PTH) level was measured by solid-phase, 2-site chemiluminescent enzyme-labeled immunometric assay. Serum 25-OH D level was measured by liquid chromatography-tandem mass spectrometry. The Heaney [12] classification of less than 80 mmol/L was used to determine insufficient vitamin D levels, whereas less than 25 mmol/L was classified as deficiency. Patients were asked about current vitamin D supplement usage, and these were grouped as a "any vitamin D supplement" (vitamin D as part of a multivitamin) or "vitamin D-specific supplement" (vitamin D taken as a high dose supplement, with or without calcium).

All participants were offered a dual-energy x-ray absorptiometry (DXA) scan, which was performed in the AMNCH Radiology Department using a Lunar Prodigy Advance densitometer, software version 11.40 (GE Healthcare, GE Medical Systems, Belgium). Dual-energy x-ray absorptiometry was reported by a Consultant Radiologist who was blinded to the underlying clinical condition. Results were expressed as T-scores compared to values of young females. Diagnosis of osteoporosis or osteopenia was determined by the Consultant Radiologist using the lowest T-score at any area. According to the World Health Organization classification [13], T-score of between —1.0 and —2.5 standard deviations (SD) were classed as osteopenia, whereas T-scores below —2.5 were classed as osteoporosis. Dual-energy x-ray absorptiometry values, which are compared to females of approximately 30 years, are representative of "peak bone mass'.' T-scores

for the total hip were also reported, as well as T-scores at the femoral neck (because of the morbidity associated with femoral neck fracture). The right femoral neck was chosen arbitrarily. T-scores were reported rather than z scores; however, the inclusion of an age-matched control group allowed us to compare the chronic pancreatitis values to nonchronic pancreatitis age-matched controls.

Statistics

Bivariate analyses were performed evaluating group characteristics. Student t test was used to compare parametric data, and χ^2 test for categorical data. Continuous data were divided into tertiles for comparison where appropriate. One-way analysis of variance (ANOVA) was performed to compare group means, and post hoc (Tukey) analysis was completed where appropriate. For multivariable analysis, backward, stepwise linear regression was done to develop a model for the prediction of lowest T-score in patients. This involved starting with all possible predictive variables and testing them one by one for statistical significance and deleting those that are not significant from the model. For all analyses, P values were two-sided, and a $P<0.05$ was considered statistically significant. All statistical analyses were performed using Minitab, version 15.1 (State College, PA).

RESULTS

Group Characteristics

Characteristics of the study groups are shown in Table 1. Patients and controls were statistically similar ($P>0.05$ for sex, age, and education level). Alcohol was the cause of disease in 38.7% of patients. Just over 6% of both patients and controls were never drinkers.

TABLE 1. Demographic, Social, BMI, and Osteopathy Details for Chronic Pancreatitis Patients and Healthy Controls

	Chronic Pancreatitis, N = 62	Controls, N = 66	*P*
Sex, n (%)			
Female	17 (27.4)	18 (27.3)	0.985*
Male	45 (72.6)	48 (72.7)	
Age, mean (SD), y	47.9 (12.5)	47.74 (11)	0.922†
Education level completed, n (%)			
Primary or secondary	40 (64.5)	40 (60.6)	0.65*
Third level or higher	22 (35.5)	26 (39.4)	
Smoking			
Never, n (%)	16 (25.8)	26 (39.4)	
Former, n (%)	9 (14.5)	21 (32.3)	
Current, n (%)	37 (59.7)	19 (29.2)	
Mean pack years (SD)‡	26 (22.5)	16.5 (17.2)	0.028§
Alcohol			
Never drinker, n (%)	4 (6.4)	4 (6.1)	
Former drinker, n (%)	31 (50)	2 (3.0)	
Current drinker, n (%)	27 (43.6)	60 (90.9)	
Mean units per week (SD)‖	22.4 (31.3)	14.7 (10.6)	0.249
Units, range	1–112	1–59	
BMI, mean (SD), kg/m^2	25.6 (5)	28.0 (4.1)	0.003§
Prevalence of osteopathy, n (%)¶			
Osteoporosis	18 (34)	6 (10.2)	
Osteopenia	21 (39.6)	20 (33.9)	0.001§
Normal bone density	14 (26.4)	33 (55.9)	

*Pearson χ^2 test.
†Student *t* test.
‡Mean pack years among current or former smokers only.
§Statistically significant difference.
‖Mean alcohol intake (units per week) among current drinkers only.
¶As per WHO guidelines: T-score < 2.5, osteoporosis; T-score 1.0–2.49, osteopenia; and T-score > 1.0, normal bone density.

Twenty-seven percent (27.4%) of patients had severe disease, and 37.1% had mild disease by the Cambridge classification. The mean (SD) fecal elastase-1 in the mild, moderate, and severe groups were 472.5 (75.1), 280.4 (195.0), and 135.0 (161.5) µg/g, respectively, which differed significantly across all 3 groups ($F_{2,43}$=21.2, P<0.000).

Almost three quarters (74.2%) of patients reported being either current or former smokers compared to 60.6% of controls (Table 1). Sixty percent of patients smoked greater than 1 pack per day (20 cigarettes per day) compared to 37.5% of controls. Lower educated controls were heavier smokers. In the control arm, 61.5% of subjects who finished third level education or higher were "never" smokers, compared to only 25% of those who completed first or second level education only. This difference was not apparent among patients, with 27.5% of lower educated patients being never smokers compared to 18.2% of higher educated patients.

Osteopathy Prevalence

Fifty-three patients and 59 controls consented to a DXA scan. These groups were also well matched and statistically similar. Mean (SD) patient age was 48.7 (11.8) years, and mean (SD) control age was 48.05 (10.7) years (P=0.889). Regarding sex, 75.5% of patients and 72.4% of controls were male (chi-squared P=0.714). Regarding education level, 21% of patients finished third level or higher compared to 24% of controls (chi-squared P=0.655).

In the patient group, 34% had osteoporosis (73.6% total osteopathy including 39.6% osteopenia). In contrast, 55.9% of controls had normal bone density, and 44.1% had low bone density (10.2% osteoporosis and 33.9% osteopenia) (P=0.001; Table 2).

Bone Density and Smoking

For patients, those in the highest smoking tertile had significantly poorer T-scores at the lumbar vertebrae (ANOVA, P=0.006; Fig. 1) than those in the lower smoking tertiles. Post hoc (Tukey) analysis showed that the difference was significant between the lowest and highest smoking tertiles only. For the mean total hip, there was a significant difference between the T-scores of the highest and lowest smokers (ANOVA, P=0.03).

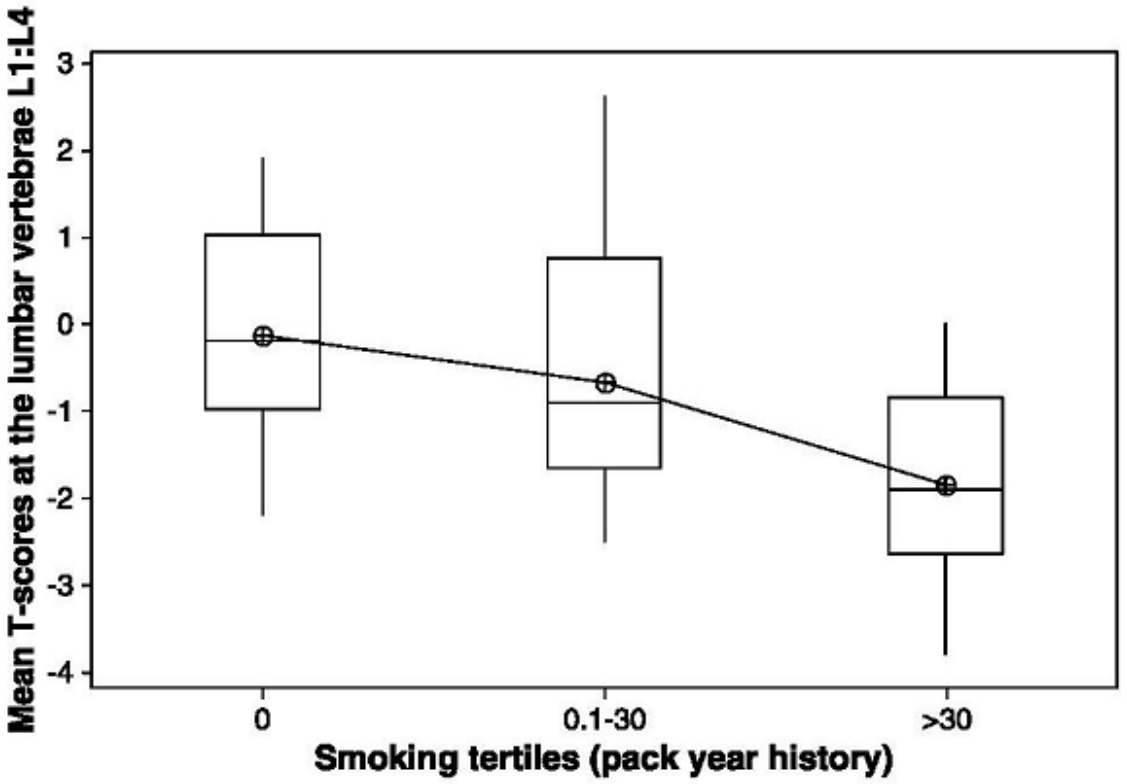

Fig. 1. T-scores at the lumbar vertebrae (L1:L4) for chronic pancreatitis patients by smoking tertiles. ANOVA, P = 0.006.

For controls, there was a significant difference in T-scores at the right femoral neck across smoking tertiles (ANOVA, P=0.005). The difference in T-scores of the total hip (ANOVA, P=0.064) and lumbar vertebrae (ANOVA, P=0.08) fell outside statistical significance.

Bone Density by Disease Severity, Age, and Sex

There was no relationship between severity of disease by the Cambridge classification and T-score at any area.

For patients, when comparing across age tertiles using ANOVA, there was an overall effect with the lowest age tertile having significantly higher T-score than both the middle and upper tertile at the right femoral neck (P=0.003; Fig. 2), mean total hip (P=0.037), but not at the lumbar vertebrae (L1:L4) (Table 2). Among controls, there was also an age effect seen at the right femoral neck (P=0.008) but not for the other areas. Among patients, there was no effect of sex for T-scores at any area. For controls, males had higher T-scores at the lumbar vertebrae and the right femoral

neck (Table 2).

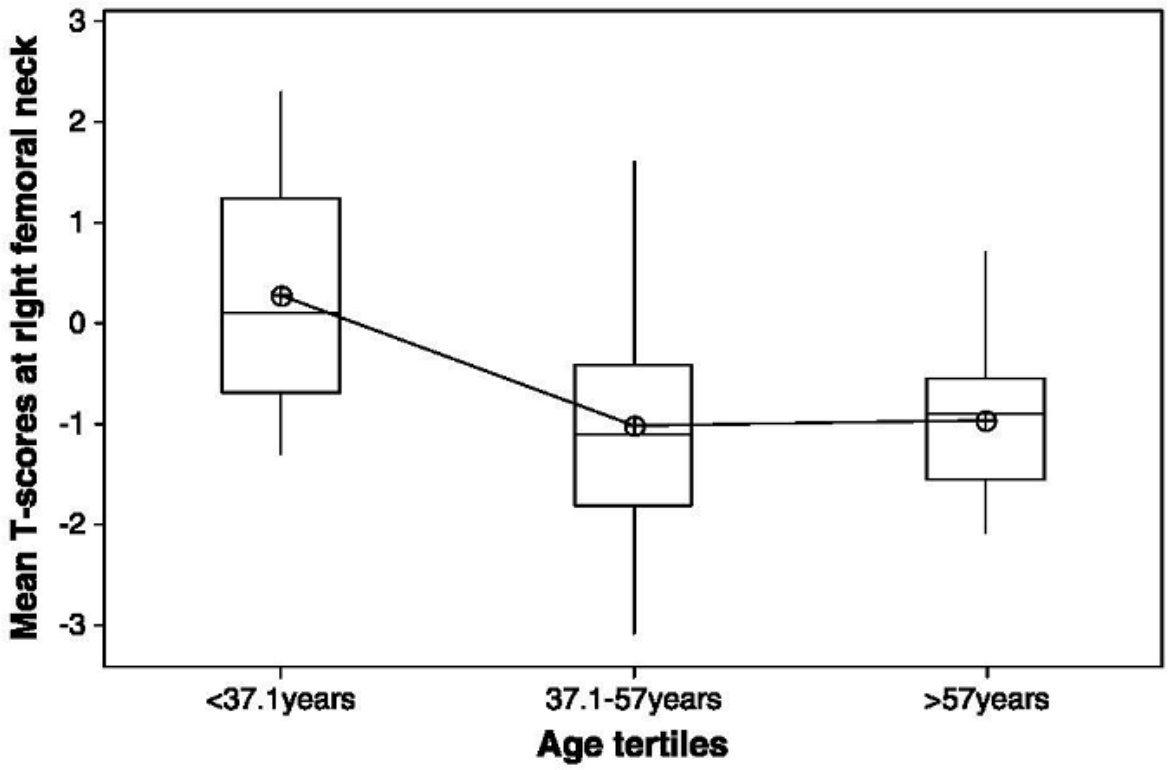

Fig. 2. T-scores at the right femoral neck for chronic pancreatitis patients across age tertiles. ANOVA, P = 0.005.

TABLE 2. Bone Density and T-Score Data for Patients and Controls

		Chronic Pancreatitis (N = 53 DXA Scans)									Matched Controls (N = 59 DXA Scans)								
		Any Area		Right Femoral Neck		Mean Total Hip		L1:L4 Lumbar Vertebrae			Any Area		Right Femoral Neck		Mean Total Hip		L1:L4 Lumbar Vertebrae		
		Lowest T-Score	SD	T-Score	BMD	T-Score	BMD	T-Score	BMD		Lowest T-Score	SD	T-Score	BMD	T-Score	BMD	T-Score	BMD	P*¶
All patients	N	−1.77	1.47	−0.754	0.945	−0.69	0.979	−0.815	1.037	N	−0.885	0.14	−0.104	1.032	0.071	1.076	0.034	1.215	0.005†
Sex																			
Male	39	−1.76	1.08	−0.925	0.944	−0.734	0.995	−0.783	1.024	43	−0.736	0.98	−0.002	1.068	0.255	0.123	0.310	1.259	<0.000†
Female	14	−1.81	2.36	−0.238	0.948	−0.562	0.933	−0.908	1.075	16	−1.280	1.19	−0.380	0.934	−0.433	0.947	−0.720	1.098	0.79
P‡				0.106		0.749		0.082					0.037†		0.1		0.017†		
Age, y§																			
First tertile	12	−0.636	0.22	0.278	1.089	0.173	1.095	0.109	1.223	14	−0.325	0.63	0.617	1.126	0.417	1.120	0.500	1.271	0.55
Second tertile	27	−2.081	1.17	−1.026	0.906	−0.967	0.946	−1.201	0.921	32	−0.981	1.21	−0.109	1.030	0.087	1.076	−0.084	1.202	0.002†
Third tertile	14	−2.23	1.83	−0.975	0.914	−0.500	0.996	−0.792	1.113	13	−1.233	0.88	−0.808	0.944	−0.317	1.029	−0.117	1.195	0.65
P‖				0.003†		0.037†		0.071					0.008†		0.252		0.453		

*Student *t* test comparing mean T-scores at the right femoral neck between patients and controls.

†Achieves statistical significance *P* = <0.05.

‡Student *t* test comparing mean T-score between the sexes at right femoral neck, total hip, and lumbar vertebrae for patients and controls.

§Patients: first tertile, <37.1 y; second tertile, 37.1–57 y; and third tertile, >57 y; controls: first tertile, <37.7 y; second tertile, 37.1–56 y; and third tertile, >56 y.

‖ANOVA comparing mean T-scores between the age tertiles at the right femoral neck, total hip, and lumbar vertebrae.

Predicting Low BMD

Using multivariable analysis, we developed a model for the prediction of lowest T-score in patients. In this model, heavy smoking history was predictive of a low T-score (P=0.002), each increase across the smoking tertiles was associated with a decrease in T-score of 0.775 on average. Body mass index was also predictive. A high BMI being predictive of a "higher" lowest T-score (P=0.003), with each increase across the categories of BMI having an increase in T-score of 0.584 on average. Age was also predictive, with every increase in age (years) having a decrease in T-score of 0.46 on average (Table 3). Vitamin D, disease severity, fecal elastase-1 levels, and education level were not significant in this model.

TABLE 3. Backward Stepwise Linear Regression Showing Factors Predictive of Lowest T-Score in Patients With Chronic Pancreatitis

	Coefficient	SE Coefficient	T	*P*
Constant	0.546	0.87	0.63	0.533
Smoking tertiles*	−0.775	0.24	−3.25	0.002
BMI categories†	0.584	0.19	3.08	0.003
Age, y	−0.046	0.014	−3.2	0.002

*Smoking tertiles are based on pack year history.

†BMI groups: underweight BMI $\leq$ 20 kg/m^2; normal weight BMI 20.1–24.9 kg/m^2; overweight BMI 25–29.9 kg/m^2; and obese or greater BMI $\geq$ 30 kg/m^2.

Vitamin D

Mean (SD) 25-OH D levels were 47.5 (21.6) in the patient group. There was no statistical difference in the 25-OH D levels of those who took a vitamin D-specific supplement or not (P=0.324), or between those who took "any" vitamin D-containing supplement (P=0.118). Serum 25-OH D levels of patients were weakly and negatively correlated with PTH levels (R=-0.314; P=0.021). The mean 25-OH D

levels of controls were 46.4 (20.4). Those who took any vitamin D-containing supplement had significantly higher vitamin D levels than those who did not (P=0.01). There was no difference in the 25-OH D levels between those who took vitamin D-specific supplements and those who did not (P=0.251). There was no significant correlation between vitamin D and PTH levels (P=0.514).

DISCUSSION

High Osteoporosis Prevalence in Chronic Pancreatitis

A third of patients with chronic pancreatitis had osteoporosis, which was more than triple the rate in our matched control group, and almost 7 times what has been previously reported [5]. The prevalence of both osteoporosis and osteopenia seen in this chronic pancreatitis group is striking, especially given that the patients are mostly male, and largely in their 40s. When analyzing bone density for patients across age tertiles, there seemed to be no difference in T-scores for patients older than 37 years (the lowest tertile) — those in the middle and upper age tertiles had statistically similar T-scores. Compared to controls, T-scores for patients in the middle age tertile (37–50 years) were significantly lower. Therefore, it seems that in chronic pancreatitis, the protective effect of age only applies for the youngest patients. Those in middle age seem to have an accelerated risk of bone density loss. For patients with chronic pancreatitis, this age effect was significant at the total hip and right femoral neck, which represents significant morbidity and mortality in the event of a fracture. The risk of fracture increases by a factor of 1.4 to 2.6 for each SD decrease in bone density [10]. Therefore, the high osteopathy prevalence is likely to translate into future fractures as patients with premature osteoporosis have the additional risk factor of increasing age to augment their fracture risk considerably in the future. A study published in 2010 [14] reported that the risk of low-trauma (fragility) fracture in patients with chronic pancreatitis was comparable to other high-risk gastrointestinal conditions including celiac disease and postgastrectomy.

The British Society of Gastroenterology osteoporosis document [10] reports 18 cross-sectional studies of bone density in Crohn's disease documenting an osteoporosis prevalence of 0% to 57.6%. Of 8 such studies in celiac disease,

osteoporosis prevalence was 0% to 80%. Consequently, the British Society of Gastroenterology gave recommendations to prevent fractures in these groups. However, only 1 Crohn disease study [15] and 2 celiac disease studies [16, 17] reported osteoporosis prevalence greater or equal than the 34% seen in our study. Therefore, one could assume that the risk is greater in chronic pancreatitis than in Crohn disease or celiac disease, and thus, similar bone health guidelines are warranted in this disease.

The reasons for bone mineral loss in this group are multi-factorial and are likely to be driven by a complex array of factors. In this study, we also found a high prevalence of osteopathy among controls (44%), and thus, socioeconomic or issues specific to Ireland could be implicated. Vitamin D levels were low in both controls and patients, which might confer an increased risk of osteopathy in both groups. Potential drivers of bone loss in chronic pancreatitis include limited mobility, malabsorption of calcium and vitamin D, chronic inflammation, and poor dietary intake and/or displacement of diet by smoking or alcohol. In celiac disease and inflammatory bowel disease, normal regulatory control of bone metabolism may be adversely affected by proinflammatory cytokines released from inflamed intestines as well as from mature T-cells [18]. The effect of chronic inflammation on bone metabolism in chronic pancreatitis has not been characterized.

Smoking and Alcohol

Heavier smokers had the greatest loss of BMD in both the patient and control groups. However, for patients, the effect was significant at the lumbar vertebrae and total hip, whereas in controls, the effect was significant only at the right femoral neck. In controls, smoking was associated with education level: those who finished third level education or higher were two and a half times more likely to be never smokers than those who completed first or second level only. Therefore, for controls, a lower smoking history might be accompanied by other features of high socio-economic status including diet, physical activity, finance, and supplement usage — all of which could influence bone health to some degree. In contrast, among patients, there was no difference in smoking according to education level. Because those who smoked the most had the greatest bone mineral loss, smoking may be a driver of

bone mineral loss independent of socioeconomic status. A growing body of evidence shows that cigarette smoking is a risk factor for osteoporosis, although the nature and magnitude of the relationship remains uncertain. In a meta-analysis of 86 studies [19], smokers had a significantly reduced bone mass compared with nonsmokers (both former and never smokers) at all bone sites, but particularly at the hip. The effects were greater in men, in the elderly and were dose dependent. Whether smoking has a distinct toxic effect on bone microarchitecture, acts to displace bone-important nutrients, affects hormones or enzymes involved in bone regulation, or damages blood supply to the bone is open to debate. Importantly, smoking is a modifiable lifestyle factor, although it is not known if smoking cessation would prevent or reverse bone mineral loss in these patients. Notably, smoking is a risk factor for developing chronic pancreatitis [20, 21, 22], although frequently underrecognized [23].

Disease Severity

Surprisingly, contrary to what was found in other studies [6, 24], loss of BMD did not seem to be associated with disease severity, with the highest losses seen in the moderately severe group. This is surprising given that the most marked pain, nutritional deficits and exocrine dysfunction, is seen when the disease is at its most severe. The fact that we did not identify a linear relationship between chronic pancreatitis severity and BMD indicates that BMD loss might be driven by factors additive to the disease process. This finding may also be indicative of problems with current severity stratification methods. The Cambridge classification used in this study provides a clinically utilizable system based on endoscopic retrograde pancreatographic findings. However, it does not address the issues of exocrine and endocrine insufficiency or the presence of extrapancreatic complications [25]. Therefore, along with other factors, this may account for the failure to find a relationship between bone density and disease severity.

Vitamin D and Calcium

Average serum 25-OH D (vitamin D) levels were in the insufficient range for both patients and controls, although surprisingly not different between the 2 groups.

One possible explanation for this may relate to higher supplementation levels among patients. In addition, controls are heavier on average than patients, thus vitamin D might be sequestered in adipose tissue, leading to lower bioavailability. For populations living at Northern latitudes such as Ireland, vitamin D deficiency and insufficiency is a public health problem because of the lack of vitamin D-producing rays for much of the year. The low levels might at least partially explain the high osteopathy levels seen in both patients and controls. Supplementation may help to increase vitamin D levels — our data showed that supplementation may increase levels, at least in healthy subjects. For patients with chronic pancreatitis, routine vitamin D supplementation is probably necessary. Only 10% of dietary calcium is absorbed in a vitamin D-deficient state, but in the vitamin D-sufficient patient, calcium intake must also be adequate to prevent loss of BMD. We suggest that calcium intakes similar to that suggested in celiac disease (1000 mg/d) is warranted in chronic pancreatitis. This study is limited by the fact that we did not measure dietary intake nor was it assessed by similar studies [5, 6]. It is likely that poor intakes of calcium are related to lower bone density.

Few studies have examined dietary intake in chronic pancreatitis. Vaona and colleagues [26] reported dietary intake of 40 chronic pancreatitis patients compared to controls; however, they did not report on intakes of calcium or vitamin D. Because dietary intake of calcium and vitamin D (coupled with nutrient malabsorption caused by pancreatic exocrine insufficiency) is a contributory factor in the development of osteopathy, ensuring adequate dietary intake of these nutrients should form part of the overall dietetic management of chronic pancreatitis.

CONCLUSIONS

Osteoporosis levels were strikingly high in this group of patients with chronic pancreatitis of various etiologies. Routine bone health assessments, including regular DXA scans, are warranted as part of the overall clinical and nutritional review in patients with chronic pancreatitis. In addition, dietary evaluation and routine calcium and vitamin D supplementation should form part of the usual treatment of patients with chronic pancreatitis. In conjunction with advice on alcohol avoidance, advice on

smoking cessation should be prioritized to protect BMD.

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Patients with chronic pancreatitis are at increased risk for osteoporosis

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Objectives. Patients with chronic pancreatitis may be at an increased risk of low bone density because of malabsorption of vitamin D and calcium, poor diet, pain, alcoholism, and smoking. We investigated the rates of osteoporosis in patients with chronic pancreatitis compared to matched controls.

Methods. The study was cross sectional in design. Sixty-two patients (mean age, 47.9 years; 72.6% male) and 66 matched controls were recruited. Dual-energy x-ray absorptiometry, smoking, and socioeconomic data were recorded.

Results. Thirty-four percent of patients had osteoporosis compared to 10.2% of controls. T-scores at the right femoral neck were lower in patients than controls (P=0.005). Patients in the highest smoking tertile had the poorest T-scores at the lumbar vertebrae and total hip. Patients in the youngest age tertile had the highest T-scores (P=0.003), but there was no sex difference.

Conclusions. Patient osteoporosis rates were triple that of controls, and almost 7 times what has been previously reported. Given the resource burden of osteoporosis, we suggest that routine bone density assessment is performed in patients with chronic pancreatitis.