

Title: Normocalcemic Hyperparathyroidism and Structural Bone Compromise

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Abstract

Purpose: Normocalcemic hyperparathyroidism (NCHPTH) is characterized by elevated serum parathyroid hormone (PTH) levels in the presence of normal serum calcium levels, with the exclusion of secondary causes. NCHPTH remains underdiagnosed and is often associated with structural compromise of bone, namely osteoporosis and fragility fractures. The purpose of this study was to perform a prospective observational cohort study investigating NCHPTH in our Metabolic Bone Disease Service (MBDS). **Methods:** Between January 2022 and June 2024, 815 patients were referred to our MBDS with an ICD-10 osteoporosis diagnosis code. Each patient underwent dual-energy X-ray absorptiometry, and when indicated, assays for serum calcium, ionized calcium, 25-hydroxyvitamin D, and PTH. **Results:** NCHPTH was identified in 25 patients (3%) or approximating a patient a month. Skeletal involvement was frequent in the NCHPTH cohort, with 72% presenting with a history of fragility fractures. Mean PTH levels were 130.8 pg/mL, while serum calcium and 25-hydroxyvitamin D levels remained within the normal range. We compared the prevalence of NCHPTH in our MBDS to other referral populations, finding an increased prevalence in our service, likely due to the high frequency of skeletal compromise in this population. **Conclusion:** These results indicate that NCHPTH is not merely a biochemical phenomenon but is rather frequently associated with skeletal pathology. We conclude that reliance on serum calcium alone likely leads to underdiagnosis of NCHPTH, whereas routine PTH testing can facilitate earlier detection, allow for preventative interventions, and enhance secondary fracture prevention efforts.

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1 **Background:** Normocalcemic hyperparathyroidism (NCHPTH) is characterized by elevated
2 serum parathyroid hormone (PTH) levels in the presence of normal serum calcium levels, with the
3 exclusion of secondary causes. NCHPTH remains underdiagnosed and is often associated with
4 structural compromise of bone, namely osteoporosis and fragility fractures. The purpose of this
5 study was to perform a prospective observational cohort study investigating NCHPTH in our
6 Metabolic Bone Disease Service (MBDS).

7 **Methods:** Between January 2022 and June 2024, 815 patients were referred to our MBDS with an
8 ICD-10 osteoporosis diagnosis code. Each patient underwent dual-energy X-ray absorptiometry,
9 and when indicated, assays for serum calcium, ionized calcium, 25-hydroxyvitamin D, and PTH.

10 **Results:** NCHPTH was identified in 25 patients (3%) or approximating a patient a month. Skeletal
11 involvement was frequent in the NCHPTH cohort, with 72% presenting with a history of fragility
12 fractures. Mean PTH levels were 130.8 pg/mL, while serum calcium and 25-hydroxyvitamin D
13 levels remained within the normal range. We compared the prevalence of NCHPTH in our MBDS
14 to other referral populations, finding an increased prevalence in our service, likely due to the high
15 frequency of skeletal compromise in this population.

16 **Conclusion:** These results indicate that NCHPTH is not merely a biochemical phenomenon but is
17 rather frequently associated with skeletal pathology. We conclude that reliance on serum calcium
18 alone likely leads to underdiagnosis of NCHPTH, whereas routine PTH testing can facilitate earlier
19 detection, allow for preventative interventions, and enhance secondary fracture prevention efforts.

20 **Keywords:** Osteoporosis, orthopedics, fracture prevention, parathyroid-related disorders, bone
21 metabolism.

22 **Introduction:** A variant of primary hyperparathyroidism, normocalcemic hyperparathyroidism
23 (NCHPTH) is defined by high PTH levels and normal serum calcium levels in the absence of

24 causes of secondary hyperparathyroidism such as vitamin D deficiency, intestinal malabsorption,
25 chronic kidney disease, hypercalciuria, and medications that interfere with calcium
26 homeostasis.[1–3] There is uncertainty as to whether NCHPTH is a unique disease unto itself, a
27 *forme fruste* of primary hyperparathyroidism, or a diagnosis of exclusion of secondary
28 hyperparathyroidism. A number of studies have shown progression over time to hypercalcemia in
29 some patients, whereas other studies have not.[4,5] Regardless, NCHPTH is often associated with
30 structural damage to bone consisting of decreased bone density, hyper-resorption, increased
31 porosity and often culminating in fractures, as presenting clinical findings.[6–9]

32 This is a study of NCHPTH in a Metabolic Bone Disease Service (MBDS) that functions in large
33 part as a fracture liaison service. One of the clinical features of interest in this study is the presence
34 of structural bone disease, separating NCHPTH from what has been described as “just a
35 biochemical signature” in patients without bone or kidney complications.[5,10,11] Furthermore,
36 other studies investigating NCHPTH have reported a high prevalence fragility fractures, cortical
37 bone loss, and impaired trabecular microarchitecture in affected populations.[12,13] Several
38 reports have indicated that 50-57% of patients with NCHPTH had osteoporosis, and 46% had a
39 fracture in adulthood.[2,10,14] Fragility fractures have been reported to occur in 11-21% of
40 patients with NCHPTH.[15] Recent population-based data indicate that even modest and sustained
41 elevations of PTH levels may attenuate trabecular bone microarchitecture. Moreover, a major
42 study of data from the National Health and Nutrition Examination Survey (NHANES) affirmed
43 that isolated fasting PTH elevation impairs lower lumbar spine trabecular bone scores (TBS) and
44 that this may predispose to fracture.[6]

45 While prevalence rates have been reported to vary in the general population from 0.4-0.6%,[10,16–
46 18] those rates are higher in patients within referral centers, both MBDS and fracture liaison

47 services (FLS), with prevalence rates of 5.2-7.8% being reported[2,19]. This is likely due to a
48 concentration of the disease, given the patient population of such centers, and the detailed
49 methodology employed for diagnosis. Unlike in primary hyperparathyroidism, serum calcium
50 concentration cannot be relied upon as a surrogate marker to diagnose NCHPTH due to its
51 persistently normal levels. Accurate diagnosis therefore depends on documenting persistent PTH
52 elevation, with normal serum calcium, alongside strict exclusion of secondary causes.[20–23]
53 Therefore, it is particularly important that MBDS and FLS clinicians have a high index of suspicion
54 for NCHPTH so that appropriate diagnosis and treatment can be carried out.[7] The purpose of
55 this report is to characterize the prevalence and clinical features of NCHPTH in an MBDS. We
56 assess the utility of routine PTH measurements to screen for NCHPTH. We suspected that
57 NCHPTH occurs with some frequency in an MBDS, that it can be occult without recommended
58 laboratory studies, and that it is associated with structural compromise of bone, notably
59 osteoporosis and fractures as presenting clinical findings. The diagnosis of NCHPTH can be
60 missed by relying on serum calcium concentration as a surrogate marker simply because it is part
61 of the automated basic metabolic panel that is routinely ordered while measurement of PTH is
62 omitted in casual/routine screening.

63 **Methods:** After review, the [BLINDED] IRB waived jurisdiction. This is a prospective
64 observational cohort study in an MBDS of patients with osteoporosis. Between 01/01/2022 and
65 06/01/2024, 815 new patients were seen with an ICD-10 diagnosis code of osteoporosis. All
66 patients underwent bone density determination by dual-energy X-ray absorptiometry (DXA) scan
67 on a Hologic Horizon W QRB Series scanner. The initial diagnosis of osteoporosis was made by
68 a T-score of <-2.5 at spine, hip, and/or forearm. All patients were evaluated, where appropriate,
69 with a standard battery of laboratory tests, including PTH (Table 1). Indications for laboratory

70 testing were osteoporosis, atypical osteopenia, and/or atypical fractures or fracture mechanisms.
71 Eligibility criteria consisted of two sets of laboratory tests which measure serum PTH, serum and
72 ionized calcium, and 25-hydroxyvitamin D. The diagnostic criteria for NCHPTH in the MBDS
73 were elevated serum PTH, normal serum and ionized calcium, and normal 25-hydroxyvitamin D,
74 on duplicate examinations, and the absence of renal or gastrointestinal dysfunction.[2,3,5,6]
75 Exclusion criteria included suspected secondary hyperparathyroidism, other metabolic bone
76 disease, clinical suggestion of malabsorption (eg bariatric surgery, celiac disease, etc.), abnormal
77 age-adjusted eGFR, hypercalciuria >300mg/24hr, and medications that interfere with calcium
78 homeostasis, such as thiazide diuretics, lithium, and anticonvulsants.[1,2,5,7,10,21,24] To
79 minimize selection bias, all patients meeting eligibility criteria during the study period were
80 included. Laboratory tests were done at the [BLINDED]laboratories or in a community-based
81 laboratory. PTH concentration was determined by two-step immunoassay. Both serum and urine
82 calcium levels were determined colorimetrically with Arsenazo III dye. Ionized calcium levels
83 were measured by an ion specific electrode. 25-hydroxyvitamin D concentrations were determined
84 by a one-step immunoassay.

85 Patients with elevated PTH and normal serum calcium concentration without secondary causes,
86 were suspected of having NCHPTH. When needed, vitamin D supplementation was used to bring
87 the serum 25-hydroxyvitamin D to about 40 ng/mL. Because PTH levels are susceptible to
88 fluctuation due to dietary calcium intake, medication, hydration levels at time of testing, and assay
89 variability, the protocol minimized these effects to provide an accurate PTH level by conducting
90 two PTH tests per patient spread apart over 3 months and ensured vitamin D levels were within
91 the normal range prior to diagnosis. Primary hyperparathyroidism was identified by elevated serum
92 calcium and PTH and those patients were referred to endocrine surgery for further evaluation and

93 treatment. In our community, upper limits of normal serum PTH vary by laboratory (65–91 pg/mL).
94 For comparison purposes, PTH levels are expressed as both absolute values and as a ratio of
95 observed patient values (Pt) to the upper limit of normal for their respective laboratory (Lab)
96 indicating normalized values (Pt/Lab) (Table 2). In this context, elevated PTH Pt/Lab would be
97 >1.0. Patients with NCHPTH often underwent PTH ultrasound or ^{99m}Tc Sestamibi PTH scan
98 (Figure 1). If surgery was indicated, these scans were used to assist with operative planning. It is
99 important to note that imaging plays no role in the diagnosis of NCHPTH or the indication for
100 surgery. Treatment, including osteoporosis medications, were withheld until the final diagnosis.

101 **Statistical Analysis and Study Design:** This is an observational study, and statistics are largely
102 descriptive. Mean, median, standard deviation, range, and confidence interval are reported for the
103 continuous variables while frequency and percentage are reported for the categorical variables and
104 compared by chi-square analysis. The sample size was based upon the primary dependent variable
105 in the study, serum PTH concentration and was estimated with intent to detect a difference of 2
106 standard deviations in PTH between the NCHPTH and normal cohorts. This effect size is a
107 conservative estimate based on pilot data, which found a mean difference of 86, a pooled standard
108 deviation of 35.6, and a Cohen's d of 2.4. Given these parameters, we found that a sample size of
109 n = 24 per group maintained a power of at least 90% for the comparisons we intend to examine.
110 Statistical significance was taken as p<0.05.

111 **Results:** The base cohort of 815 osteoporotic patients consisted of 676 females (83%) and 139
112 males (17%) of mean age 71 (range:14-95 years). Of the 815 patients, 26 were excluded due to
113 secondary hyperparathyroidism or due to failure to complete follow-up laboratory tests. Twenty-
114 five patients (3%), or approximately one patient per month, of our base osteoporotic cohort were
115 identified as having NCHPTH by the clinical and biochemical criteria above. Of these patients, 23

116 (92%) were female and 2 (8%) were male, with a mean age of 73 years (range: 58-89) consistent
117 with the general distribution of patients in our MBDS. Other symptoms associated with primary
118 hyperparathyroidism, such as stone formation, were not observed in this group. Of the 25 patients,
119 10 (40%) underwent parathyroidectomy.

120 NCHPTH patients presented to our MBDS with a history of structural bone compromise, including
121 fractures and osteoporosis. 18/25 (72%) patients presented with fractures and 7/25 patients (28%)
122 had a history of multiple fractures over time. The fracture diagnoses are presented in Table 3. No
123 characteristic fracture patterns were identified in the population. The observed fractures were all
124 fragility fractures and the mean age of patients who fractured was 71.8 years. By comparison, in a
125 random osteoporotic cohort matched for age and sex, the fracture incidence was 80%. The point
126 is not that NCHPTH creates a greater incidence of fracture than does osteoporosis, but that it is
127 not protective of fractures and needs to be taken seriously as a condition that has a high incidence
128 of material failure of bone, both metabolic and structural. NCHPTH is not simply a biochemical
129 finding with no consequence, but rather a condition with substantial structural morbidity and
130 functional disability, mandating accurate diagnosis and appropriate treatment intervention. The
131 bone densities of the NCHPTH population are presented in Table 4. Mean bone densities were
132 osteoporotic in the femoral neck and forearm, and osteopenic in the lumbar spine and total hip.
133 4/25 (16%) patients had pre-existing lumbar spine surgery with implants, and no spinal DXA
134 scores were available.

135 Biochemical data are presented in Table 5. Mean PTH levels were elevated (Pt/Lab = 1.60 pg/mL).
136 Mean serum calcium, ionized calcium, urine calcium levels, and 25 hydroxyvitamin D levels were
137 within normal limits. Renal function was normal in all included patients, with all patients having
138 normal creatinine and eGFR levels.

139 **Discussion:** Virtually all populations of NCHPTH have been described in metabolic bone referral
140 and FLS environments indicating not only a concentration of prevalence but also the special
141 attention of these environments to diagnose NCHPTH given an appropriate index of suspicion and
142 recommended diagnostic studies, particularly serum PTH.[16] ~~Several studies have shown that~~
143 ~~patients with fragility fractures evaluated outside of dedicated bone units are undertreated and do~~
144 ~~not receive secondary fracture prevention.[25,26]~~ MBDS and FLS provide a structured framework
145 for the evaluation and management of fragility fractures and, thereby, an understanding of their
146 underlying pathological causes, including osteoporosis.[25] Several studies have shown that an
147 FLS is an effective organizational approach to secondary fracture prevention.[17,18,26–30]
148 Recent evidence has indicated that NCHPTH is an uncommon phenomenon, particularly when
149 contemporary diagnostic stringency is applied. In the largest rigorously screened metabolic bone
150 referral cohort to date, 6,280 adults were referred for evaluation and only 11 individuals met the
151 case definition requiring ≥ 2 normal serum calcium values, repeated elevation of parathyroid
152 hormone (PTH) levels, and exclusion of secondary causes, corresponding to a prevalence of
153 0.18%.[20] Community-based surveys, which relied on a single calcium measurement and low
154 normal vitamin D levels ($< 20\text{ng/mL}$), have yielded higher prevalence rates of 0.2-0.7%, however
155 these estimates may overstate the prevalence due to the lack of strict diagnostic criteria.[21] In our
156 MBDS, we observed an NCHPTH prevalence of 3%, approximating 1 case/month in the 29-month
157 observation period, with the majority of patients being women, consistent with our broader MBDS
158 demographic. A compendium of data from several endocrine referral services ($n = 11,957$ patients)
159 reported an average prevalence of 1.38% (range 0.4-8.9%).[16] In our study, the NCHPTH patients
160 were drawn from a baseline comparison population that also had osteoporosis and fractures, and
161 our observed prevalence should be seen in that context. The variability in estimates of prevalence

162 reflect differences in diagnostic criteria, techniques, exclusion criteria, and definitions of normality
163 which make it difficult to establish consistency in clinical definition.[10,31] The prevalence of
164 structural bone failure in NCHPTH will also vary substantially depending upon the population of
165 patients being seen. MBDS referral populations, including ours, specifically consist of patients
166 with osteoporosis and/or fractures, rather than those presenting for screening, and NCHPTH is
167 found in the evaluation of osteoporosis.

168 Regardless of its precise definition, structural compromise of bone is **associated with** NCHPTH,
169 defined by either osteoporosis, fragility fractures, or both. Both magnitude and the frequency of
170 low bone density and the degree of bone loss were found to be greatest at the distal 1/3 radius
171 followed by the femoral neck reflecting a greater sensitivity of cortical bone sites to PTH-mediated

172 bone turnover. In our MBDS cohort, 18/25 (72%) of our patients experienced an osteoporotic
173 fracture at various sites. Reports from 5 bone centers of 109 patients with NCHPTH reported an
174 prevalence of osteoporosis of 44% (range 25-57%).[16] ~~One study from a bone referral center~~

175 ~~reported that 21/37 (57%) of patients with NCHPTH had osteoporosis in at least one site.[7] In our~~
176 ~~MBDS cohort, 18/25 (72%) of our patients experienced an osteoporotic fracture at various sites.~~

177 By comparison, ~~in 3 reports, not from an MBDS, of 55 multiple reports comprising 92~~ NCHPTH
178 patients showcased that 13-46% of them experienced fractures, 16.2% (range=13-21%)

179 ~~experienced fractures.[16] Another report, from a bone referral center, reported 17/37 (46%)~~
180 ~~NCHPTH patients experienced fractures.[7] As expected, the prevalence of fractures and~~

181 ~~NCHPTH is higher in an MBDS and FLS than in the general population, or even metabolic bone~~
182 ~~referral centers, emphasizing~~ These results emphasize that NCHPTH patients do exhibit

183 associated with structural bone failure,[7] providing the impetus for evaluation and therapy.
184 Several studies have shown that isolated PTH can have a negative effect on the structure of bone.

185 One study of 3516 subjects derived from the 2005-2006 NHANES demonstrated a negative
186 association between PTH and TBS of -0.0034 (95% CI: -0.0050-0.0017) (p<0.0001.)[6] In this
187 study, serum calcium concentration averaged 9.5 mg/dl and 25-hydroxyvitamin D averaged 57
188 mg/dl indicating the isolated effect of PTH on TBS. TBS reflects bone microstructure and is related
189 to the mechanical strength of bone.[32] Low TBS has substantial predictive value for fracture risk
190 in prospective and cross-sectional studies.[6,33,34] Other studies, using qCT also demonstrated an
191 association between serum PTH levels and bone microarchitecture.[35,36]

192 Occult NCHPTH can be identified by utilization of recommended diagnostic laboratory guidelines.
193 For many years, stakeholder individuals and institutions have recommended a biochemical
194 evaluation of serum and urine of osteoporotic individuals and those with fragility fractures
195 including prominently, serum PTH.[14,37,38] While constituents of the laboratory evaluation may
196 vary somewhat among centers, between 2001 and 2020, the American Society for Bone and
197 Mineral Research, American Association of Orthopedic Surgeons, the American Orthopedic
198 Association, and many other societies have published consensus guidelines for secondary fracture
199 prevention.[26,39,40] Subsequent studies have pointed out that these are often not followed in the
200 initial work-up of osteoporosis with or without fractures.[16,41–44] The experience in our MBDS
201 confirms that patients who have been referred to us from the community have often started on
202 medication, usually bisphosphonates, without the recommended diagnostic evaluation. With
203 serum calcium being an unreliable marker of NCHPTH, it is important to consider NCHPTH and
204 to follow recommended laboratory testing guidelines as part of the initial workup, especially for
205 fragility fractures and osteoporosis, before starting any medical therapy. PTH testing should be
206 performed prior to instituting, or after withholding, medical therapy for osteoporosis as many of
207 these therapies secondarily elevate the PTH levels, thus precluding an accurate diagnosis. Without

208 appropriate biochemical testing, the prevalence of NCHPTH can be underestimated as a cause of
209 reduced bone density and fractures.[21,44]

210 This study has some limitations and some advantages. This was an observational study and
211 association but not causation can be determined. Following recommended laboratory guidelines
212 strengthens the associations. Secondly, referral bias could lead to higher detection rates of
213 NCHPTH due to the concentration of NCHPTH patients in the MBDS environment.

214 Because data is reported from an MBDS with a relative concentration of NCHPTH patients, the
215 prevalence of the condition is not comparable to that in the general population. It is, however,
216 consistent with other reports of referral populations and it points out the opportunity for clinicians
217 to identify NCHPTH patients in those environments. Our population, even in an MBDS, may be
218 too small to establish a definitive incidence or to describe stereotypical fracture patterns.
219 Additionally, the overall fracture rate of the base population was unavailable, therefore the fracture
220 rates of the base and NCHPTH populations could not be compared. However, we observed that all
221 of our NCHPTH population exhibited structural compromise of bone, which should be considered
222 a skeletal manifestation of NCHPTH and provides a justification for treatment intervention, which
223 is supported by the literature.[6]

224 Our observations have led to several conclusions. (1) NCHPTH remains a diagnostically elusive
225 condition that may be underrecognized in osteoporotic patients, or those with a history of fragility
226 fractures, particularly when depending upon serum calcium concentration as the sole screening
227 marker. (2) The clinical and radiological aspects of NCHPTH are difficult to quantify because of
228 the differing evaluative methods used to rule out secondary hyperparathyroidism and by the small
229 sample sizes in most studies leading to under-recognition of NCHPTH in osteoporotic patients, or
230 those with a history of fragility fractures.[1] (3) In our MBDS, we identified NCHPTH in 3% of

231 our osteoporotic patients, most of whom had a fracture history. Osteoporosis and fractures should
232 be considered as “presenting symptoms” that are skeletal manifestations of elevated PTH and
233 should be included in treatment decisions. (4) These findings support the recommendation that a
234 structured diagnostic approach, which incorporates guideline-driven serum PTH screenings, can
235 enable early detection of NCHPTH that would be otherwise missed and that coexisting structural
236 bone damage, either osteoporosis or fractures, indicates treatment interventions or detailed follow-
237 up. Future studies are warranted to investigate the long-term outcomes of NCHPTH-focused
238 interventions and to assess the cost-effectiveness and broader integrations of routine PTH
239 screenings in MBDS clinics.

240

241

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247 T.C., J.M., R.A; Formal analysis and investigation: J.P., A.K., R.A.; Writing - original draft
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Table 1. Recommended Laboratory Tests for Osteoporotic Fractures[26,39]

Bone	Kidney	Serum
Bone-specific Alkaline Phosphatase, Calcium, 25 Hydroxyvitamin D, PTH, C- or N-telopeptide, Phosphorous, Magnesium	Creatinine, eGFR, Blood Urea Nitrogen	CBC, SPEP, ESR, Albumin

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Table 2. Mean [BLINDED]PTH Lab Values with a Selection of Comparison Studies. Results are Normalized (Pt/Lab)

Origin of Values	PTH Lab Range	Mean PTH	Pts/Lab
	65	120.5	1.83
	80	138.1	1.73
[BLINDED]* (pg/mL)	91	113	1.24
<i>Bilezikian et al, 2010</i> (pg/mL) [18]	65	93	1.43
<i>Palermo et al, 2020</i> (pg/mL) [2]	72	126.8	1.76
<i>Marques et al, 2011</i> (pg/mL) [20]	65	109.5	1.68
<i>Cusano et al, 2013</i> (pg/mL) [21]	66	77.5	1.17

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* In our community, PTH upper limits of normal vary by laboratory (65–91 pg/mL).

Table 3. Fracture Sites of the NCHPTH Population

Fracture Type			
<i>Hip (Intracapsular)</i>	2	<i>Distal Radius</i>	3
<i>Hip (Extracapsular/Subtrochanteric)</i>	5	<i>Foot and Ankle</i>	4
<i>Tibia/Fibula</i>	3	<i>Vertebral</i>	2
<i>Femoral diaphysis</i>	1	<i>Pelvis</i>	3
<i>Distal Femoral Condyle</i>	2	<i>Humerus</i>	4

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Table 4. DXA T-Scores and Descriptive Statistics Stratified by Anatomic Location

DXA	1/3 Forearm	Total hip	Femoral Neck	L1-L4
<i>Mean</i>	-3.4	-2.3	-2.7	-1.9
<i>Median</i>	-3.3	-2.1	-2.5	-2.0
<i>95% CI</i>	-4.0 – -2.8	-2.6 – -2.0	-2.9 – -2.3	-2.6 – -1.3
<i>Range</i>	-5.5 – 0.4	-4.0 – -1.1	-4.5 – -1.6	-5.1 – 1.3
<i>SD</i>	1.4	0.8	0.8	1.5
<i>Number of patients with a T-Score below -2.5</i>	17 (68%)	8 (32%)	13 (52%)	8 (32%)

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Table 5. Biochemical Data of the NCHPTH Population

CHEMISTRY	1st PTH (pg/mL)	2nd PTH (pg/mL)	1st Serum calcium (mg/dL)	2nd Serum Calcium (mg/dL)	Ionized Calcium (mg/dL)	Urine Calcium (mg/24h)	Creatinine (mg/dL)	25OHD (ng/mL)
<i>Mean</i>	124.3	137.4	9.6	9.5	4.9	110.8	0.76	50.2
<i>Median</i>	117	124	9.5	9.6	4.9	103	0.77	49.4
<i>95% CI</i>	108.1 – 140.5	119.6 – 155.2	9.4 – 9.8	9.4 – 9.7	4.7 – 4.9	87.6–133.9	0.7–0.8	43.9 – 56.5
<i>Range</i>	71.3 – 265	82 – 296	8.6 – 10.4	8.8 – 10.4	4.1 – 5.3	50-228	0.46-0.99	25.1 – 84.4
	41.2	45.4	0.5	0.5	0.3	45.8	0.14	16.1

Figure Legend:

Figure 1. (a) ^{99m}Tc Sestamibi scan of parathyroid adenoma. Scan shows increased ^{99m}Tc uptake in the inferior right parathyroid. (b) Sagittal-oriented ultrasound of the right thyroid lobe. Evident is a deep fibrocystic heterogeneous nodule suggestive of parathyroid adenoma (white arrow).

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