Basal and Stimulated Calcitonin and Procalcitonin by Various Assays in Patients With and Without Medullary Thyroid Cancer

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BACKGROUND: Calcitonin (CT) is a sensitive marker for evaluation of medullary thyroid cancer (MTC). However, CT measurement can vary with assay- and nonassay-dependent factors, and procalcitonin (PCT) measurement has been proposed for evaluating questionable increases in CT.

METHODS: We tested 2 fully automated CT assays (Immulite [IL] and Liaison [LIA]) and one nonautomated CT assay (Medipan, immunoradiometric nonautomated assay [IRMA]) and compared these results with PCT (Brahms, Kryptor). We evaluated preanalytical conditions and CT cross-reactivity in sera of 437 patients with clinical conditions associated with hypercalcitoninemia. Additionally, we determined the true “nil” CT concentration in 60 thyroidectomized patients and defined CT cutoff concentrations for pentagastrin stimulation testing in 13 chronic kidney disease (CKD) patients and 10 MTC patients.

RESULTS: Markedly decreased CT concentrations were found after storage of sera for >2 h at room temperature and >6 h at 4 °C. Cutoff concentrations for basal and stimulated CT were disease and assay dependent. Proton pump inhibitor therapy was the most frequent reason for increased CT. PCT concentrations were higher in patients with MTC than in CKD patients without infections (P < 0.001). Whereas IL and LIA demonstrated comparable analytical quality, the IRMA gave increased CT concentrations in nil sera and showed cross-reactivity with PCT in patients with concomitant bacterial infection.

CONCLUSIONS: IL, LIA, and IRMA detected increased CT concentrations in non-MTC patients and discriminated MTC from CKD patients in pentagastrin tests. PCT assessment may be helpful in the diagnostic work-up of increased CT concentrations in questionable clinical circumstances.

Calcitonin (CT) is an extremely sensitive marker for both the initial diagnosis and follow-up of patients with medullary thyroid cancer (MTC). Sandwich immunoassays are applied when measuring CT in the clinical laboratory. These assays use monoclonal antibodies for the recognition of intact and mature CT. Recently, most laboratories have moved from radio-labeled systems to fully automated chemiluminescence or fluorescence assays. Because the Nichols Institute Diagnostics test system, previously considered to be the “gold standard” for CT measurement, is no longer available, there is a need to evaluate the analytical and diagnostic capability of alternative fully automated CT assays and compare them with traditional manual methods. Since the interpretation of CT values in human serum may be complicated by various preanalytical and analytical factors or by increased CT concentrations in patients presenting no clinical evidence for MTC, such evaluation has to be done carefully.

In our study, we tested 2 chemiluminescent immunometric fully automated assays and one nonautomated radioimmunometric assay for analytical quality.
and potential pitfalls generally not evaluated by the manufacturer.

Our first aim was to investigate the impact of preanalytical factors on CT concentrations, including sample stability at various storage temperatures and storage times as well as assay interferences from hemoglobin and lipids. Second, we compared the frequency of increased CT concentrations in the 3 assays in clinical conditions known to be associated with hypercalcitoninemia, such as chronic kidney disease, Hashimoto’s thyroiditis, and proton pump inhibitor therapy. Third, we determined the true “nil” CT concentrations for the different assays by using sera from patients, who had been thyroidectomized for differentiated thyroid (i.e., nonmedullary) cancer and were disease free on follow-up. Fourth, we performed pentagastrin (PG) stimulation testing in patients with increased CT concentrations and non-MTC disease (chronic kidney disease) and as well as in MTC patients to define stimulated CT cut-off values for the 3 assays. Finally, we tested whether procalcitonin (PCT), a well-known biomarker for severe infection and sepsis, could be an additional diagnostic marker for the diagnosis of MTC in our patients.

Materials and Methods

IMMUNOASSAYS

Three commercially available CT assays were evaluated: (a) the chemiluminescent immunometric fully automated assay Immulite (IL) 2000 (Siemens, cut-point 95th percentiles: 8.4 ng/L for males; 5.0 ng/L for females, analytical sensitivity 2.0 ng/L); (b) the chemiluminescent immunometric fully automated assay Liaison (LIA) (DiaSorin, cut-point 95th percentiles: 18.9 ng/L for males; 5.5 ng/L for females, analytical sensitivity 1 ng/L); and (c) an immunoradiometric nonautomated assay (IRMA) (SELco, Medipan; cut-point 95th percentiles: 15.0 ng/L for males; 10.0 ng/L for females; analytical sensitivity 1.6 ng/L). All 3 assays were calibrated against the 2nd International WHO calibrator 89/620.

PCT was measured by using the PCT Kryptor assay (B.R.A.H.M.S; functional assay sensitivity 0.06 μg/L). To enable a comparison between PCT and the CT methods, 2 potential cut-points for pathological PCT values had to be considered: concentrations between 0.25 μg/L and 0.5 μg/L indicate a potential bacterial infection; concentrations >0.5 μg/L point to a high probability of severe bacterial infection.

In our hands, interassay CVs were <3.4% at 209 ng/L and <5.5% at 9.9 ng/L for the IL, 5.1% at 497 ng/L and 10.1% at 25 ng/L for the LIA, 3.7% at 98.9 ng/L and 8.9% at 12.9 ng/L for the IRMA assay, and 3.5% at 11 μg/L and 4.7% at 0.26 μg/L for the PCT assay.

PREANALYTICAL EXPERIMENTS

CT concentrations of pooled surplus serum from randomly selected blood donors spiked with recombinant CT (Sigma) were compared with endogenous CT concentrations from MTC patients. To enable the performance of a sufficient number of preanalytical tests, a pool of surplus serum from 6 patients with MTC (CT concentrations between 809 and 17 470 ng/L) was diluted 1:20 with pooled sera of healthy blood donors.

For both sample groups, the influence of preanalytical factors on the endogenous or recombinant CT concentration was tested for CT concentrations within the reference interval, in moderately increased (25–100 ng/L) and in highly increased (160–600 ng/L) CT concentrations.

SERUM SAMPLES

Blood samples were centrifuged within 1 h, and serum was aliquoted and stored at −20 to −40 °C until analysis.

BASAL CT AND PCT CONCENTRATIONS

CT and PCT concentrations were measured in sera of 437 patients who were free of clinical and diagnostic signs of MTC but were prone to have hypercalcitoninemia due to non-MTC disease or medications and included the following: (a) patients with Hashimoto’s thyroiditis (n = 98); (b) 50 patients without and 44 patients with proton pump inhibitor (PPI) therapy (20–40 mg lansoprazol daily, n = 31; 10–40 mg omeprazol daily, n = 9; 40 mg pantoprazol daily, n = 3; 45 mg lansoprazol daily, n = 1) (these patients had been hospitalized for bacterial infection (pneumonia, lower urinary tract infection, cholangitis, or diabetic foot syndrome; creatinine concentrations were within the reference interval in all patients); and (c) patients with chronic kidney disease (CKD) (stage 2–4: n = 123; 32 with PPI), under dialysis (CKD stage 5: n = 63; 27 with PPI) and after kidney transplantation (CKD stage 2–5: n = 59, 21 with PPI). CKD stages were defined according to the National Kidney Foundation Kidney Disease Outcomes Quality Initiative (www.kidney.org).

PATIENTS WITH NIL CT CONCENTRATIONS

We compared CT results of the above-mentioned groups with findings of patients expected to have no detectable CT concentrations, since they had been thyroidectomized for differentiated thyroid cancer (n = 60) and were disease free on follow-up (negative thyroglobulin, negative diagnostic 131-I scan).
STIMULATED CT AND PCT CONCENTRATIONS
PG stimulation testing was performed in 13 patients with chronic kidney disease (stage 2–4) with increased basal CT concentrations and in 10 patients with known MTC and postoperative basal CT concentrations ranging between 18 and 1511 ng/L. For PG testing, patients were positioned in a supine position and an intravenous cannula was inserted 15 min before testing. At 0 min, a blood sample was drawn and 0.5 μg/kg body weight PG (Nova Laboratories) was injected. Blood samples were drawn at 2, 5, and 10 min after PG injection.

The study was approved by the ethics committee at the University of Leipzig. Informed consent was obtained from patients for sample analysis and for undergoing PG testing.

STATISTICAL ANALYSIS
Statistical analysis was carried out by using SPSS 11.5 (Statistical Package for the Social Sciences for Windows) and Statistica 7.0 (Statsoft). Because the data were nonnormally distributed, Spearman correlation analysis was performed. A P value of <0.05 was considered significant.

Results

INFLUENCE OF STORAGE TIME AND TEMPERATURE ON CT CONCENTRATIONS
We found that CT stability was not affected by up to four thawing and freezing cycles (data not shown). Next, we tested the stability of CT after sample storage at −40 °C and 4 °C for up to 7 days and at room temperature for 0–24 h. Results were expressed as percentage changes relative to basal concentrations of moderately increased and highly increased CT values. No influence on endogenous CT or recombinant CT concentrations was seen after 7 days of storage at −40 °C (data not shown). However, at 4–8 °C (refrigerator temperature), reduced endogenous CT concentrations were found in all samples stored ≥6 h, irrespective of the applied assay. After 12 h of storage, CT concentrations were reduced by 23% (see Table 1 in the Data Supplement that accompanies the online version of this article at http://www.clinchem.org/content/vol57/issue3). After 7 days, CT concentrations were reduced by 65% (IL), 33.5% (LIA), and 91.1% (IRMA). At room temperature, endogenous CT concentrations remained stable for only 2 h.

COMPARISON OF CT CONCENTRATIONS OF DIFFERENT KITS WITH PCT
CT concentrations by the IRMA were almost as high as those by LIA, but IL results were clearly lower (Fig. 1). Correlation coefficients (r) between the individual as-

![Fig. 1. Comparison of CT concentrations measured by LIA vs IL (A), IL vs IRMA (B), and LIA vs IRMA (C) for samples of patients with CKD (n = 13) and MTC (n = 10) before and during the PG test at 2, 5, and 10 min (n = 92). The regression equations for A, B, and C were as follows: LIA = 1.42 × IL + 8 ng/L; IL = 0.61 × IRMA − 9.8 ng/L; and LIA = 0.87 × IRMA − 1.1 ng/L, respectively.](image)
say results differed depending on CT concentrations: non-MTC subjects demonstrated distinctly lower correlation coefficients ($r$ range 0.52–0.76) than patients with MTC during PG testing ($r$ range 0.88–0.93) (Supplementary Table 2). Lower correlations were found between CT and PCT values ($r$ range 0.32–0.41) in all assays except the IRMA ($r$ range 0.45–0.75). To further explore this finding, we investigated the association between PCT and CT concentrations in patients who had been hospitalized with a suspected bacterial infection–related PCT increase ($n = 94$) but with no indication for MTC. As shown in Fig. 2, increased CT concentrations measured by the IRMA were found in patients with simultaneous increases of PCT concentrations ($r = 0.74, P < 0.001$). In contrast, no such association was observed for CT results obtained in with IL or LIA (Supplementary Figs. 1 and 2).

**HYPERCALCITONINEMIA DUE TO NON-MTC CONDITIONS MEASURED BY DIFFERENT CT ASSAYS AND COMPARISON WITH PROCALCITONIN**

In the group of Hashimoto’s thyroiditis patients (Table 1, $n = 98$), one identical sample was found to be highly increased in all three CT assays. On further assessment, the diagnosis of MTC was confirmed in this patient. From the remaining 97 samples, slightly increased CT concentrations were found in the range between 0% (IRMA, PCT), 1.0% (IL), and 3.1% (LIA).

In the cohort of patients with internal diseases and PPI therapy (Table 1, $n = 44$), CT increases were found in 6.8% of samples measured by IL, in 11.4% by LIA, and in 31.8% by IRMA. In comparison, 2.0% of patients without PPI treatment ($n = 50$) had increased CT concentrations by IL, whereas 10.0% of these patient samples showed increased CT concentrations by LIA and 26.0% by IRMA. Depending on the cut-point, PCT concentrations were increased between 6.8% and 13.6% in PPI users and 4.0% and 16.0% in PPI nonusers.

In patients with CKD stages 2–4 (Table 1, $n = 123$), increased CT concentrations were found in 13% by IL, 9.8% by LIA, and 24.4% by IRMA. In CKD stage 5 (dialysis group; $n = 63$), increased CT concentrations were found in 11.1% by IL, 1.6% by LIA, and 71.4% by IRMA. In patients with a kidney transplant ($n = 59$), increased CT concentrations were found in 23.7% by

![Fig. 2. Comparison of CT concentrations from IRMA (Medipan) with PCT concentrations measured by the Kryptor (B.R.A.H.M.S.) in patients with bacterial infections and treatment with and without PPI therapy ($n = 93$). Correlation analysis was performed by the Spearman method.](image-url)

**Table 1.** Number (percentage) of patients with CT concentrations above the sex-dependent cut-point (95th percentile reference interval limit) as a function of assay method and treatment with PPIs.

<table>
<thead>
<tr>
<th>Patient groups</th>
<th>n</th>
<th>IL &gt; cut-point</th>
<th>LIA &gt; cut-point</th>
<th>IRMA &gt; cut-point</th>
<th>PCT &gt; cut-point 0.25 µg/L</th>
<th>PCT &gt; cut-point 0.5 µg/L</th>
</tr>
</thead>
<tbody>
<tr>
<td>Thyroiditis</td>
<td>98</td>
<td>1 (1.0)</td>
<td>3 (3.1)</td>
<td>1 (0)</td>
<td>0 (0)</td>
<td>0 (0)</td>
</tr>
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<td>PPI</td>
<td>44</td>
<td>3 (6.8)</td>
<td>5 (11.4)</td>
<td>14 (31.8)</td>
<td>6 (13.6)</td>
<td>3 (6.8)</td>
</tr>
<tr>
<td>Non-PPI</td>
<td>50</td>
<td>1 (2.0)</td>
<td>5 (10.0)</td>
<td>13 (26.0)</td>
<td>8 (16.0)</td>
<td>2 (4.0)</td>
</tr>
<tr>
<td>CKDb</td>
<td>123</td>
<td>16 (13.0)</td>
<td>12 (9.8)</td>
<td>30 (24.4)</td>
<td>8 (6.5)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>Dialysis</td>
<td>63</td>
<td>7 (11.1)</td>
<td>1 (1.6)</td>
<td>45 (71.4)</td>
<td>20 (31.8)</td>
<td>10 (15.9)</td>
</tr>
<tr>
<td>Post-TX</td>
<td>59</td>
<td>14 (23.7)</td>
<td>11 (18.6)</td>
<td>14 (23.7)</td>
<td>n.m.</td>
<td>n.m.</td>
</tr>
<tr>
<td>Summary</td>
<td>436</td>
<td>42 (9.6)</td>
<td>37 (80.5)</td>
<td>117 (26.8)</td>
<td>42 (11.1)</td>
<td>15 (4.0)</td>
</tr>
</tbody>
</table>

*One sample of the thyroiditis group was excluded from the analysis, since the patient was diagnosed with MTC.

b CKD, CKD stage 2–4; Dialysis, CDK stage 5; post-TX, after renal transplantation; n.m., not measured because of an insufficient sample volume.

c Whole number of investigated samples, $n = 377$. 

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IL, 18.2% by LIA, and 23.7% by IRMA. More than 31.3% of CKD patients treated with PPI demonstrated increased CT or PCT concentrations. PCT concentrations were above the 0.25 μg/L cut-point in 6.5% of the patients.

Higher CKD stages were associated with increased CT concentrations by IRMA ($r = 0.19, P < 0.05$) and with a positive trend also for PCT concentrations ($r = 0.17, P = 0.06$). In contrast, no correlation was found between CKD stages and CT concentrations determined by IL and LIA.

Considering the data for men and women combined, our MTC-free patients ($n = 436$) demonstrated higher cut-points (95th percentile) for increased CT values compared to the reference values provided by the manufacturers. In women, we found CT cut-points (our data vs manufacturer’s data) of 4.3 vs 5.0 ng/L (IL), 6.5 vs 5.5 ng/L (LIA), and 42.0 vs 10.0 (IRMA); CT cut-points in men (our data vs manufacturer’s data) were 17.1 vs 8.4 ng/L (IL), 29.4 vs 18.9 ng/L (LIA), and 66.0 vs 15.0 ng/L (IRMA).

**CT CONCENTRATIONS OF THYROIDECTOMIZED PATIENTS**

To determine the true “nil” CT concentration, we investigated a cohort of patients ($n = 60$) who had undergone thyroidectomy for differentiated thyroid cancer and were considered cured. All samples measured with the IL showed results below the detection limit. In the LIA, 97% of the samples had CT values below the detection limit and 3% of the samples were within the reference interval. By contrast, 97% of the IRMA results were in the reference interval, suggesting cross-reactivity with molecules related to monomeric CT (data not shown).

**PG STIMULATION IN PATIENTS WITH CKD VS PATIENTS WITH MTC**

Higher stimulated CT values were found by IL and LIA compared with the IRMA assay (Table 2). There was an overlap in basal CT concentrations between CKD and MTC for 2 patients by IL and three patients by LIA. Interestingly, no such overlap was seen with the IRMA and PCT assays.

Importantly, PG-stimulated CT concentrations in the three CT assays revealed no overlap between CKD and MTC patients. Although PG stimulation led to only marginal increases in PCT concentrations, a clear discrimination between CKD and MTC patients was also possible on the basis of stimulated PCT concentrations. Of note, median basal PCT concentrations of MTC patients were significantly higher than median concentration of patients with CKD (3.32 μg/L vs 0.076 μg/L, $P < 0.001$).

**Discussion**

Our preanalytical experiments demonstrated that CT was unstable in serum kept at room temperature, with hormone concentrations decreasing after 2 h in all three assays. Such instability is indeed suggested by the package inserts of some LIA and IRMA kits; however, published data on CT stability are scarce (6, 7). Accordingly, samples for CT measurement should be stored in the frozen state, although other investigators have reported a 10% to 30% decrease, even in frozen samples (6). In contrast, PCT concentrations appeared to have better stability than CT (6). Interestingly, we did not find major CT changes over at least four freeze-thaw cycles.

CT concentrations in basal and PG-stimulated serum samples showed considerable variation across the different assays, despite the use of the single calibrator WHO 89/620. Several major causes can be suggested to explain these discrepancies. First, the specificity of the CT antibodies across assays is diverse, leading to their different recognition of molecular isoforms of CT (5, 8–11). As a consequence, an assay-independent cut-point for CT of 10 or 20 ng/L, as proposed in the literature (12), appears to be questionable. Second, it is unclear if the recent nonradioactive CT methods detect only monomeric molecules, as was suggested to be the “state of the art” for IRMAs 10 years ago (4). Third, and important for clinical application, we found indirect, strong evidence for cross-reactivity of PCT and CT by the IRMA assay in samples with a clinical background of possible infection.

Analysis of CT concentrations in non-MTC patients again suggested analytical differences between the three assays and a disease-dependent clinical relevance of increased CT concentrations: slightly increased CT concentrations in patients with thyroiditis may be explained in part by the use of the 95th percentile as the upper limit of the reference interval. A somewhat higher incidence of increased CT concentrations was found in 94 hospitalized patients with a suspected bacterial infection–related PCT increase. The frequency of increased CT concentrations in these patients was related to the degree of cross-reactivity of the individual CT methods with this prohormone: IRMA > LIA > IL.

It is well known that the use of PPIs leads to chronically increased gastrin concentrations that may potentially stimulate C-cells. Our assay-dependent frequency of increased CT concentrations by PPI ranged from 2.0% to 26.0% (no PPI) and 6.8% to 31.8% (PPI). These data, supported by earlier findings (13) and our results of increased CT concentrations in 70% of CKD patients on PPI treatment, suggest that PPI treatment should be interrupted and CT analysis should be re-
peated in case of pathologically increased basal CT values.

In patients with CKD, we observed a considerably lower frequency of increased CT concentrations (IL 11% to 13%; LIA 2% to 10%) compared to findings of other investigators that described a frequency of up to 30% for both patients on and not on hemodialysis (14–19). In rare cases, MTC can cause such increases (20). The fact that the glomerular filtration rate (CKD stage) of our patients was not associated with CT concentrations measured by IL and LIA suggests that the increased CT was most likely caused by C-cell hyperplasia than by a delayed clearance of the hormone. Accordingly, our results of maximal increases in basal CT concentrations up to 59.5 ng/L (IL) or 85.5 ng/L (LIA) in CKD patients were not strongly indicative of either the diagnosis of MTC or a decreased renal clearance (21).

Furthermore, in a cohort of 60 thyroidectomized non-MTC patients, CT concentrations were, as expected, either below the detection limit or within the reference interval. The detectable CT concentrations for the LIA and the IRMA suggest some minor cross-reactivity with non–thyroid-related substances comparable to reported data of assays from Scantibody (8), Nichols (4), or CIS (22).

Using the PG test, a distinct method-dependent increase of the stimulated-to-basal CT ratio was observed. The cause for the approximately 2-fold stronger increase of CT values in the IL and LIA assays compared to the IRMA values cannot be clarified by this work, but the results clearly underlined the necessity to define assay-specific cutoff values for diagnostic PG testing. Assuming the highest CT or PCT concentration of the CKD patients as this cutoff for the differentiation between non-MTC hypercalcitoninemia and MTC, the diagnostic sensitivity for the correct diagnosis of MTC by basal CT measurement was 100% for the IRMA and PCT, and 90% and 80% for IL and LIA.

### Table 2. Basal and peak concentrations of CT and PCT in individual patients with CKD and MTC during PG testing.

<table>
<thead>
<tr>
<th>Patient ID</th>
<th>CT basal (LIA, ng/L)</th>
<th>CT basal (IL, ng/L)</th>
<th>CT basal (IRMA, ng/L)</th>
<th>PCT basal (µg/L)</th>
<th>PG-CT (LIA, ng/L)</th>
<th>PG-CT (IL, ng/L)</th>
<th>PG-CT (IRMA, ng/L)</th>
<th>PG-PCT (µg/L)</th>
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<tr>
<td>CKD1</td>
<td>52</td>
<td>28.1</td>
<td>39</td>
<td>0.074</td>
<td>107</td>
<td>84.2</td>
<td>85</td>
<td>0.077</td>
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<td>CKD2</td>
<td>7.3</td>
<td>&lt;2.0</td>
<td>9.6</td>
<td>0.071</td>
<td>60.7</td>
<td>12.9</td>
<td>21</td>
<td>0.071</td>
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<td>CKD3</td>
<td>10.9</td>
<td>&lt;2.0</td>
<td>6.5</td>
<td>0.030</td>
<td>41.8</td>
<td>17.3</td>
<td>14</td>
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<td>77</td>
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<td>2.7</td>
<td>2.93</td>
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<td>43.4</td>
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<td>10 100</td>
<td>7010</td>
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<td>155</td>
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<td>4150</td>
<td>2130</td>
<td>3810</td>
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<td>853</td>
<td>609</td>
<td>1504</td>
<td>4.16</td>
<td>10 300</td>
<td>7620</td>
<td>10 724</td>
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<td>0.54</td>
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<td>534</td>
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<td>701</td>
<td>701</td>
<td>1.39</td>
</tr>
<tr>
<td>MTC9</td>
<td>68</td>
<td>79</td>
<td>211</td>
<td>0.87</td>
<td>327</td>
<td>437</td>
<td>711</td>
<td>1.58</td>
</tr>
<tr>
<td>MTC10</td>
<td>18.2</td>
<td>14.7</td>
<td>74.8</td>
<td>0.49</td>
<td>223</td>
<td>243</td>
<td>462</td>
<td>1.48</td>
</tr>
</tbody>
</table>

*Maximal concentrations for the CKD patients are shown in bold; minimal concentrations and concentrations below the maximal levels in MTC patients are shown in bold and italics.*
Serum Calcitonin and PCT Measurements by Different Assays

respectively. After stimulation with PG, all four assays were capable of differentiating completely between both groups of patients.

The evaluation of the three different CT assays was accompanied by the parallel measurement of PCT concentrations in patient sera. Assuming that ambulatory patients with Hashimoto’s thyroiditis had no bacterial infection, PCT concentrations <0.25 ng/L appear to exclude MTC. The number of increased basal PCT concentrations in CKD patients was lower than the number of increased CT concentrations measured by IL, LIA, or IRMA. In comparison to patients with known MTC, basal PCT concentrations in CKD patients undergoing PG revealed an even better discriminative value than basal CT concentrations, irrespective of the CT assay used. However, PCT was not or was only marginally stimulated by PG, and the stimulation was more pronounced for patients with MTC. So far, there are only few clinical studies that have investigated the role of PCT in the diagnosis of MTC (6, 23–25 ). Our findings support the data of these studies and suggest that in patients with increased CT concentrations but no clinical suspicion of MTC, as in CKD patients, PCT could be a promising additional diagnostic marker to exclude MTC. Prospective studies with higher numbers of patients will be required to address this issue appropriately.

In conclusion, whereas IL and LIA appear to exhibit comparable analytical quality, the IRMA has the issue of increased CT concentrations in nil sera and raises some concerns because of a high degree of cross-reactivity with PCT. All three assays detect increased CT concentrations in non-MTC patients and discriminate MTC from CKD patients in PG tests. PCT assessment may improve and complete the diagnostic work-up of increased CT concentrations in questionable clinical circumstances.

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References


