Rapid PTH Assay by Simple Modification of Nichols Intact PTH-Parathyroid Hormone Assay Kit

To the Editor:

In recent years, there has been an interest in producing a rapid, intraoperative assessment of parathyroid hormone (PTH) in serum to evaluate the efficacy of parathyroid surgery [1-3]. Here, we report a simple modification of the intact PTH-parathyroid hormone overnight assay from Nichols Institute Diagnostics (San Juan Capistrano, CA) that is usable by laboratories having Abbott (Abbott Park, IL) Quantum Technology and an Abbott Dynamic Incubator. Our modification provides a result for the operating surgeon within 35 min; the 2SD limit of detection (95% confidence) is 20 ng/L (0.322 pmol/L; n = 20), as determined by EP Evaluator Release 3 (David G. Rhoades Associates, Kennet Square, PA).

Nussbaum et al. [1] reported a modification of the same assay used with internal jugular vein samples and incubation at 37 °C for 15 min with no rotation; their lower limit of detection was 25 ng/L (0.4025 pmol/L).

Our study was performed in Abbott wide-well enzyme immunoassay (EIA) plates with antecubital venous specimens taken before the operation and 8 min after removal of the last parathyroid tissue. Serum, EDTA plasma, and whole blood taken from the same subject were evaluated. We chose to assay serum because it yielded higher PTH results than EDTA plasma or whole blood in our method. The PTH beads from the kit were placed in a 20-place wide-well plate. The kit calibrators, controls, and both of the patient’s specimens (pre- and postexcorision) were all set up in duplicate except for the two highest-concentration calibrators, which we used in singlicate to allow the entire assay to fit on one plate. The kit isotope was added, and the plate was covered and placed in an Abbott Dynamic Incubator for 10 min at 45 °C in a rotating mode. The liquid was then aspirated and the beads were transferred to test tubes by utilizing the Abbott transfer system for EIA quantum technology. After washing the beads twice and removing the liquid by aspiration, we placed the tubes containing the beads in a gamma counter and counted the radioactivity for 1 min.

We performed intraoperative PTH assays on eight patients undergoing surgery for parathyroid adenoma or hyperplasia. The results were reviewed retrospectively and the study was classified as exempt by our Institution Research Review Board. The specimens analyzed at surgery were always retested with the unmodified Nichols Institute Diagnostics overnight assay. The correlation between the two methods was excellent (r = 0.9986) as determined by the EP Evaluator. The standard error of estimate was 23.053 by the Deming method. In the eight patients we studied, serum PTH decreased at least 50% in the 8-h postexclusion specimen. Other authors report that a decrease of >50% in the postexclusion specimen is considered to be predictive of a postoperative return to normal calcium concentrations [2].

References


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Biochemical Markers of Tubular Function in Patients Receiving Continuous Carboplatin Infusion

To the Editor:

Several studies have evaluated the nephrotoxicity of carboplatin [cis-diammine-1,1-cyclobutatedicarboxylate-platinum(II); an analog of cisplatin] infused in a single dose, but little information is available about its nephrotoxicity when patients undergo continuous prolonged infusion [1, 2]. Moreover, platinum compounds can exert their nephrotoxic effects not only on glomerular but also on tubular function, inducing tubular necrosis. We therefore decided to study renal performance by assessing both glomerular function, expressed as creatinine clearance, and tubular function, assaying the urinary enzymes N-acetylglucosaminidase (NAG) and γ-glutamyltransferase (GGT) as markers of tubular damage.

Enrolled in the study with their informed written consent were 21 hospitalized patients (17 males and 4 females) with head and neck tumors, all of whom were undergoing concomitant radiochemotherapy with carboplatin. The carboplatin was infused over 24 h with a volumetric infusion pump at the daily dose of 30 mg/m² for 14 consecutive days (total dose, 420 mg/m²).

All subjects had normal liver and renal function, as defined by serum activities of GGT and aspartate and alanine transaminases, blood urea nitrogen (mean ± SD 6.78 ± 0.3 mmol/L), and creatinine (79.5 ± 8.8 mmol/L). No one underwent forced hydration. Second morning midstream urine samples were collected 24 h before and just after the start of carboplatin infusion and subsequently on days 3, 5, 8, 12, and 14.

We studied the changes of basal values of creatinine and enzymatic activities and considered significant only changes >30%, taking into account both the day of the maximal increase and the trend. We observed three different kinds of response (Table 1):

Group A—5 of 21 (24%) patients presented no significant variations of tubular enzymes or of urinary creatinine. All these patients showed a low clinical toxicity (grade 1), estimated according to WHO criteria [3].

Group B—6 of 21 (28%) patients presented an early increase of tubular enzymes by day 3 of treatment without a concomitant significant variation of urinary creatinine. Five of these patients showed a grade 1 myelotoxicity; one patient had a grade 3; only one showed severe myelotoxicity (grade 4). Values for tubular enzymes and urinary creatinine for all patients in this group returned to normal within the time course of the therapy.

Group C—10 of 21 (48%) patients presented a late increase of GGT and NAG.