About PTH measurement

Nefrología 2008; 28 **(4)** 461

To the editor: In an attempt to answer the question asked by C. de La Piedra and colleagues in their article «Differences depending on parathyroid peptides: What are we measuring?»,[1] I call your attention to the following considerations:

1. It should be verified that hospitals providing higher results express these as pg/mL, rather than pmol/L. It is not unusual that in multicenter studies some participant forgets conversion from his/her routine working units into the agreed units. Indeed, the striking differences between the three hospitals disappear if the series of highest results is divided by 3.43, the conversion factor to pg/mL.

2. The goodness of results reported by all three hospitals may be shown by the very close correspondence of ratio values between the methods: for all three hospitals, the Immulite/Elecsys ratio is 1.02 ± 0.11, and the Abbott/Immulite ratio 1.29 ± 0.01. Results are therefore reliable, at least «relatively».

3. The wide variability between methods (advocated as invalidating of K/DOQI references) is not such in practice, because 90% of hospitals use the Immulite (from DIPESA) and the Elecsys or Modular (from ROCHE) systems, and as we have just seen, the results of both are «exchangeable».

4. It is a fact known to all laboratory technicians that differences in PTH do not only stem from the existence of various circulating parathyroid peptides and different methods to measure them, but also from a characteristic physical property of such peptides, namely their extreme lability to time and temperature.[2] The only «real» values are those reported during surgical procedures, when rapid measurements are made under no temperature changes. The situation is quite different when serum is not separated until 1 hour after blood collection, is kept in test tubes in racks for 2 additional hours at room temperature, subsequently frozen for one week, and finally thawed and let to reach room temperature for 1 or 2 additional hours: PTH levels are up to 30% lower than those found with a rapid measurement.[3] This may be the reason for the different results in the hospital with the lowest values as compared to the other two hospitals.

It would thus be desirable that nephrologists become interested not only on the methods used to measure PTH, but also on some pre-test and sample traceability issues having an impact on the quality of results, such as whether or not the processed sample was frozen, and whether the laboratory working system allows for delays (times longer than 40 min) in the serum separation (serum is the sample of choice for measuring PTH), freezing, and thawing phases. We technicians are fully aware of the need for rapid serum separation, freezing, and testing, but not all laboratories have automated sample management systems for prioritizing, for instance, PTH samples and minimizing duration of processing steps. However, this will soon occur, and will result in the desired decrease in differences between PTH measurements.

1. De la Piedra C, Fernández E, González Casaus Mª L, González Parra E. Diferencias en la función de los péptidos paratiroideos ¿Qué estamos midiendo? *Nefrología* 2008; 28 (2): 123-128.
2. Indridason OS. Causes of lability in PTH values. *Semin Dial* 2000; 13 (5): 337.
3. Martín-Gil FJ, San-Miguel Hernández A, Iglesias-García R, Martín-Rodríguez L, Del Río M, Fernández-Senovilla JC. Comparación de resultados de PTH intacta por los sistemas Architect i2000 de Abbott, Immulite 2000 de Siemens y Modular E170 de Roche. *XV Reunión de la Sociedad Andaluza de Análisis Clínicos.* Mojácar, 6-8 de marzo de 2008.

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About PTH measurement. Reply to Dr. F. J. Martín Gil

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To the editor: In reply to the letter by Dr. F. J. Martín Gil

The publication (1) alluded to by Dr. F. J. Martín Gil (2) was an editorial, and this is why the materials and methods may possibly not have been explained in sufficient detail.

The study on the figures provided by each PTH measurement was performed as follows: whole blood was drawn from 150 patients on hemodialysis. Tubes were subsequently centrifuged and aliquoted, after which they were all stored at -80ºC. In all centers, aliquots were thawed just before analysis. The time from sample thawing to testing could not therefore have influenced the results obtained.

On the other hand, we can guarantee that differences in results were not due to inadequate unit conversion.

As stated by Dr. Martín Gil, serum for PTH measurement is a delicate, easily degraded sample. This is an important point to be considered by the laboratory sample reception section.

However, the different levels provided by the different tests are a real fact, and are due to the different antibodies used and the different test calibration. Because of this, a normal value may be converted into a pathological value, and vice versa.

This is why the Spanish Society of Nephrology wanted to call the attention of clinicians to this possibility. Results of this study have been summarized in a small card providing formulas for converting a PTH value obtained by a

given procedure to the value that would be obtained by the conventional Nichols test.

The group that performed this study intends to prepare a longer publication on this subject in the near future.

We thank Dr. Martín Gil for his letter, that allowed us for a more detailed explanation about some issues that were possibly not sufficiently clear in the editorial.

1. De la Piedra C, Fernández E, González Casaus Mª L, González Parra E. Diferencias en la función de los péptidos paratiroideos ¿Qué estamos midiendo? *Nefrología* 2008; 28 (2): 123-128.

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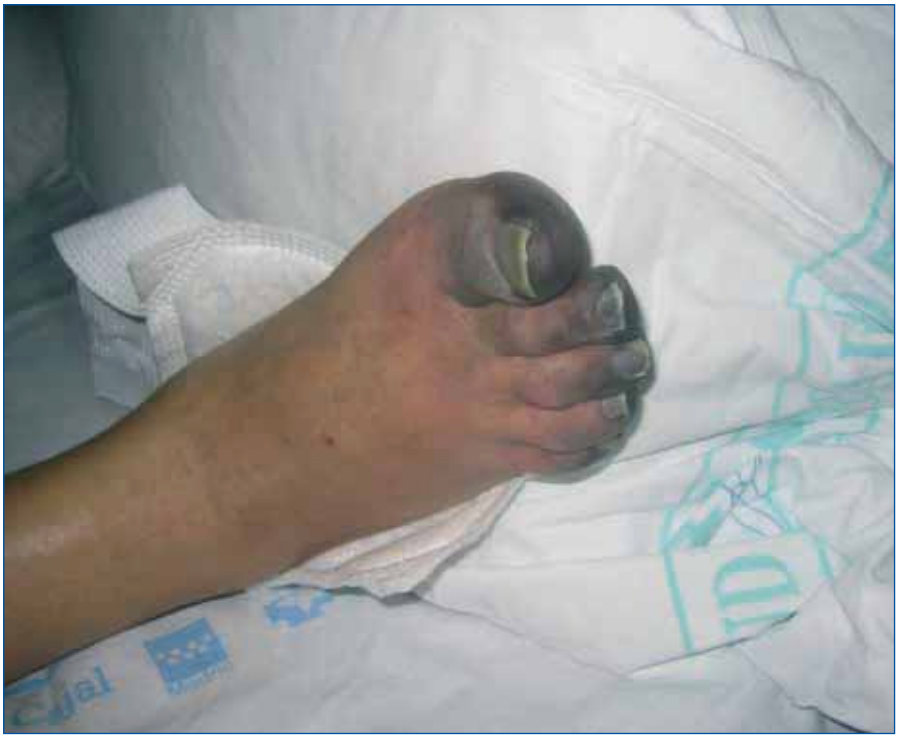

Figure 1. Distal cyanosis affecting toes resulting from microvascular thromboses.

Management of heparin-induced thrombocytopenia in a patient on hemodialysis complicated with thrombosis in the extracorporeal circuits

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To the editor: Heparin-induced thrombocytopenia (HIT) is a hypercoagulability state resulting from production of antibodies against a heparin-platelet factor 4 complex that occurs in 0.2%-1% of patients receiving heparin.[1] The condition should be considered when a greater than 40% decrease from baseline is detected in platelet count between 4 and 10 days since the start of heparinization.[1,2] Mortality from thrombosis is high (30%), and urgent action consisting of heparin discontinuation and administration of an antithrombotic, lepidurin in our setting, is therefore required.[3]

A 72-year-old male patient who experienced an acute myocardial infarction during coronary bypass surgery is reported here. The patient required insertion of an aortic counterpulsation balloon and administration of unfractionated heparin (UFH). Three days later, fever, hypotension, and renal failure requiring hemodialysis occurred. Four days later, circuit thrombosis, not prevented by prostacyclin addition, was detected. Platelet count decreased to 42,000(mL from a baseline value of 210,000/mL. On the following day, the patient showed cyanosis in the fingers of both feet (Figure 1), impossibility of hemodialysis persisted, and a right femoral thrombosis was detected by echo Doppler. HIT was suspected and confirmed by platelet aggregation tests and ELISA. Heparin was discontinuation

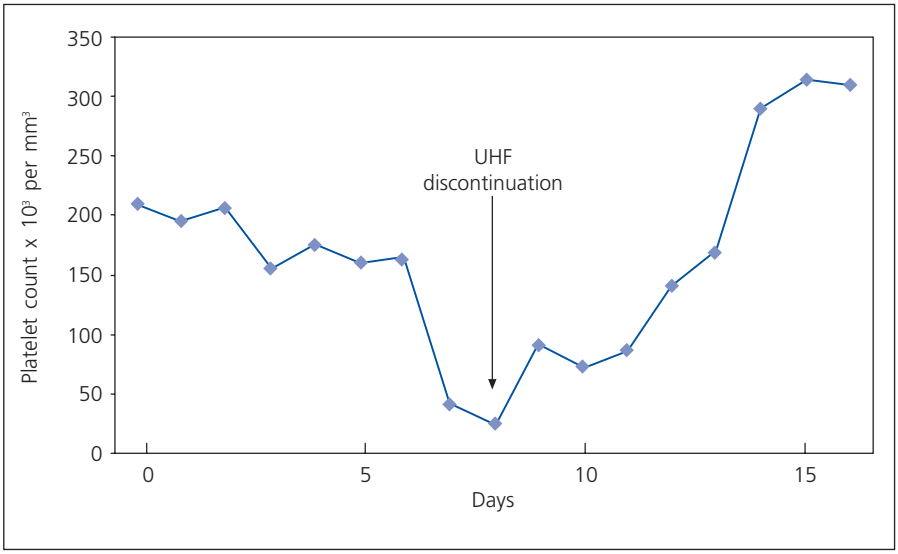

Figure 2. Evolution of the platelet count.