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### Primary hyperoxaluria type 1 : clinical, genetic and biochemical studies

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## CHAPTER 02 Postponing urine acidification for 24 hours does not change the oxalate concentration

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### INTRODUCTION

*Oxalate is produced in excess in patients with primary hyperoxaluria and excreted into the urine. An accurate determination of urinary oxalate is indispensable for the diagnosis and therapeutic follow-up of patients with primary hyperoxaluria (1). For this purpose, urine collection is usually performed in acidified containers, to avoid false negative results due to potential precipitation of calcium oxalate. Although the effect of long-term urine storage at different conditions on oxalate concentration has been studied (2,3), no studies have addressed the need for acidification at voiding for the initial diagnosis of hyperoxaluria. Reliable results might also be obtained by urine acidification upon arrival in the laboratory. Such a procedure will abolish the risk of acid burns by accidental spilling or vapour inhalation of concentrated hydrochloric acid for patients or their caretakers.*

### METHODS

In the present study we investigated the effects of delayed acidification of urine upon its collection on the analysis of urinary oxalate. The study was conducted in four patients with primary hyperoxaluria type 1, of whom one received hemodialysis (PH1 dial), and in three control subjects, of whom one received peritoneal dialysis (Non-PH1 dial). All maintained a low oxalate diet starting three days prior to the first urine collection. The subjects collected morning urine samples after overnight fasting on four to eight non-consecutive days. Immediately after voiding, the urine was split into two aliquots. One-half was put in a dry container and the other half in a container containing 15 ml 25% hydrochloric acid and gently shaken. Both containers were taken to the laboratory within 4 hours after urine collection. The non-acidified urine was again divided into two portions. One was acidified to pH1 and the other was acidified to pH1 only after a storage period of 24 hours at 4°C.

Following acidification and prior to analysis, the urine samples were shaken at room temperature. Urinary oxalate concentration was determined by ion chromatography (Dionex-DX 300). Calcium, magne-

sium and citrate concentrations were measured at the same time (spectrophotometric, colorimetric) for calculation of the urine super saturation, in order to observe stability of ion concentrations over time (4).

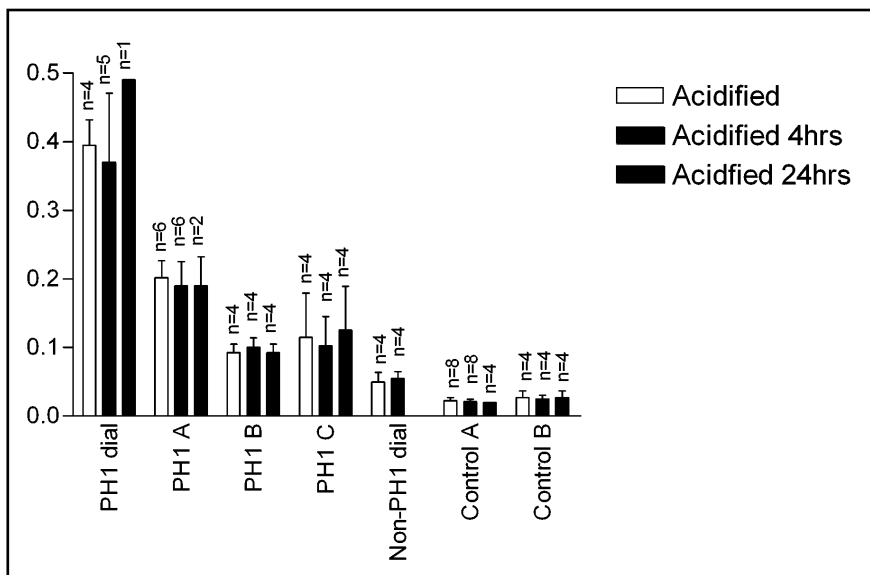
## RESULTS

The Wilcoxon signed-ranks test assessed differences in sample concentrations. Statistical significance was set at  $P < 0.05$  for all comparisons. The results showed that urinary oxalate concentrations, expressed as mmol/mmol creatinine, were not significantly different between the urine samples acidified immediately after voiding and the urine samples acidified after either 4 or 24 hours (Wilcoxon rank test,  $p = 0.609$  for 4 hours and  $p = 0.334$  for 24 hours, FIGURE 02.1). Urinary calcium concentration was below the upper range of reference value in all subjects. Calculated median urine super saturation was 16 (range (6-36) mmol/l for patients and 3 (range 2-5) mmol/l for controls.

## DISCUSSION

Postponed acidification of urine samples did not change the calculated super saturation. Apparently, there is a higher concentration of ions in the urine of patients with hyperoxaluria, which poses them at risk for urinary stone formation. However, this did not influence the determination of urinary oxalate, even if acidification was delayed for 24 hours. These results support postponed urine acidification at the laboratory.

**FIGURE 02.1** Urinary oxalate (mean  $\pm$  SD) in all subjects with immediate acidification, acidification 4 hours after collection and acidification 24 hours after collection.



No significant intra-individual day-to-day variation in urinary oxalate concentration, corrected for creatinine concentration, was measured in the urine of PH1 patients A and B, nor in the control subjects (non-PH1 dial, control A and B). This is in agreement with observations made by Zarembski *et al.* (5). However, a considerable intra-individual variation was observed for the urine samples of the PH1 patient on hemodialysis and in patient C (FIGURE 02.1). The variation in the PH1 patient on dialysis may be explained by oxalate mobilization from the extensive accumulation of oxalate in tissues, which may vary depending on the time intervals between the dialysis sessions. The variation of oxalate excretion in patient C may be the result of varying dietary intake. Nevertheless, the observed variations would not jeopardize the diagnosis of PH1, as even the lowest level of oxalate excretion in this subject was twice the upper limit of reference range. However, these variations may obscure the response to therapeutic interventions aimed at reducing oxalate production. More sensitive methods to detect changes in endogenous oxalate production are therefore warranted. In conclusion, we demonstrated that collected urine could be stored at 4°C in a home setting without acidification for a period up to at least 24 hours, without resulting in variations in oxalate concentration that would cumber a diagnostic screening of primary hyperoxaluria.

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