MATHEMATICAL MODELING OF PHOSPHATE REBOUND DURING WEEKLY CYCLE OF HEMODIALYSIS SESSIONS

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OBJECTIVES

An abnormal level of serum phosphate, often elevated in patients on hemodialysis (HD), is a considerable clinical problem. Control mechanisms of phosphate level and specific distribution of phosphate within the body make the phosphate kinetics in patients on HD difficult to model mathematically. The pseudo one-compartment model (POC, Agar et al, 2011) can effectively describe the initial rapid decrease in serum phosphate followed by a period of relatively constant concentration. However, the model is unable to reflect the increase in serum phosphate (rebound) occurring before the end of HD session that is observed in some patients. Our aim was to extend the POC model by the time delay that captures the time lag in the response of the serum phosphate control loops, and to compare the extended model (dPOC, Fig. 1) with the clinical data and the results from the POC model.

METHODS

Cohort of 25 patients was examined during 3 consecutive 4-hour HD sessions of 1-week dialysis treatment cycle. Phosphate concentration was measured in serum samples every hour and in dialysate samples every 0.5 hour. Volumes of total and extracellular body water were measured by Body Composition Monitor. According to the POC model phosphate is removed during HD from a central distribution compartment (accessible for dialysis) and at the same time phosphate is mobilized from an inner, large compartment (being the representative of other phosphate storages within the body. c.f. Fig. 1). We introduced the time delay in the term that describes the rate of mobilization as a function of plasma phosphate concentration, which in dPOC has the value that it had some fixed time ago (time lag).

RESULTS

The serum phosphate concentration at the end of HD session was on average higher than the concentration measured at 3 hour of dialysis, $p = 0.01$, and this phenomenon is described correctly only by dPOC model, Figs. 2-3. The average sum of squared relative errors was smaller (by 40%) for the dPOC than that obtained for POC model. The predicted volumes of the phosphate central distribution compartment (on average 79% and 62% of extracellular water volume, ECW = 16 ± 3 L, for dPOC and POC models, respectively) were different ($p < 0.001$), whereas differences in the predicted values of phosphate inner clearance (K_M = 134 ± 106 and 117 ± 54 mL/min for POC and dPOC models, respectively, NS) and dialyzer clearance were statistically insignificant. The time delay was on average 40.3 ± 23.7 min (range 6 - 75.8 min). We observed a profound correlation between intradialytic rebound and the time lag (rho = 0.93, $p < 0.001$).

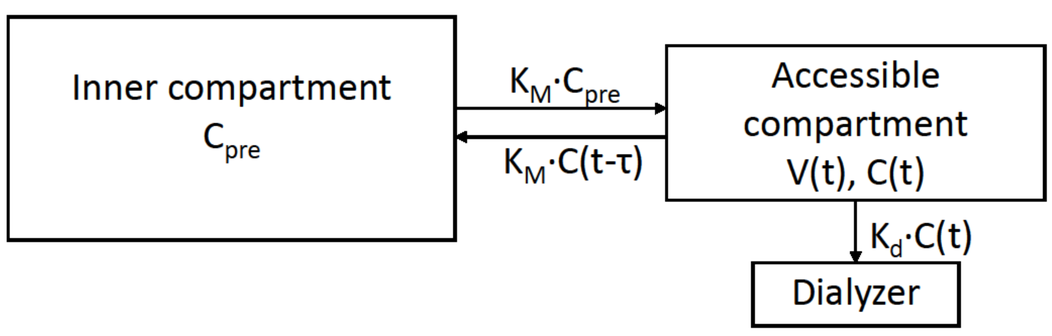

Fig. 1. Schematic representation of the pseudo one-compartment model with delay (dPOC), where C_{pre} – predialysis phosphate concentration, K_M – phosphate inner clearance, C(t) – phosphate concentration and V(t) – volume of accessible compartment, K_d – dialyzer clearance and τ – time delay.

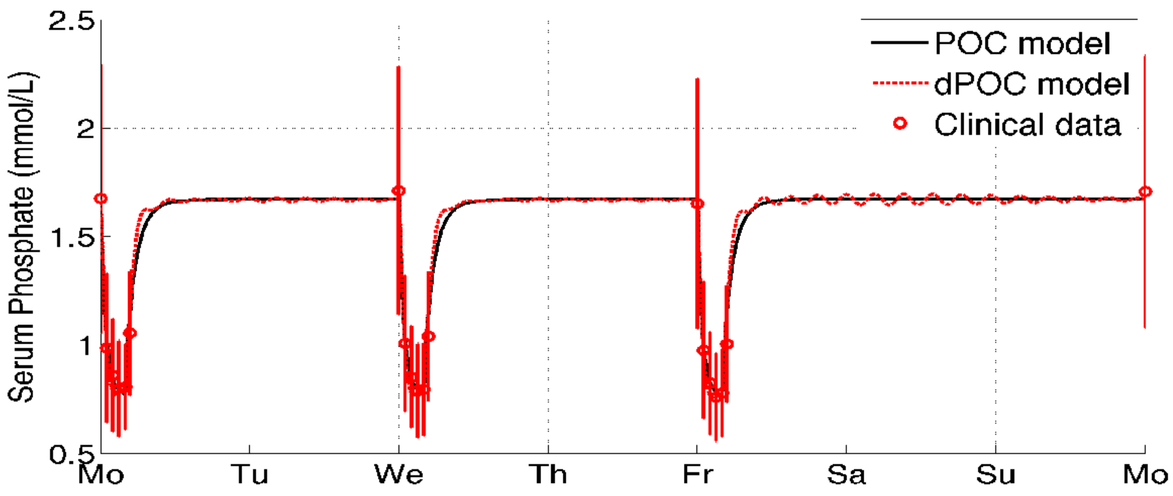

Fig. 2. The average model predicted curves according to the pseudo one compartment (POC, solid black line) and delayed pseudo one compartment (dPOC, dotted red line) models and corresponding experimental data (circles, mean ± SD) for the whole cycle of three consecutive hemodialysis sessions.

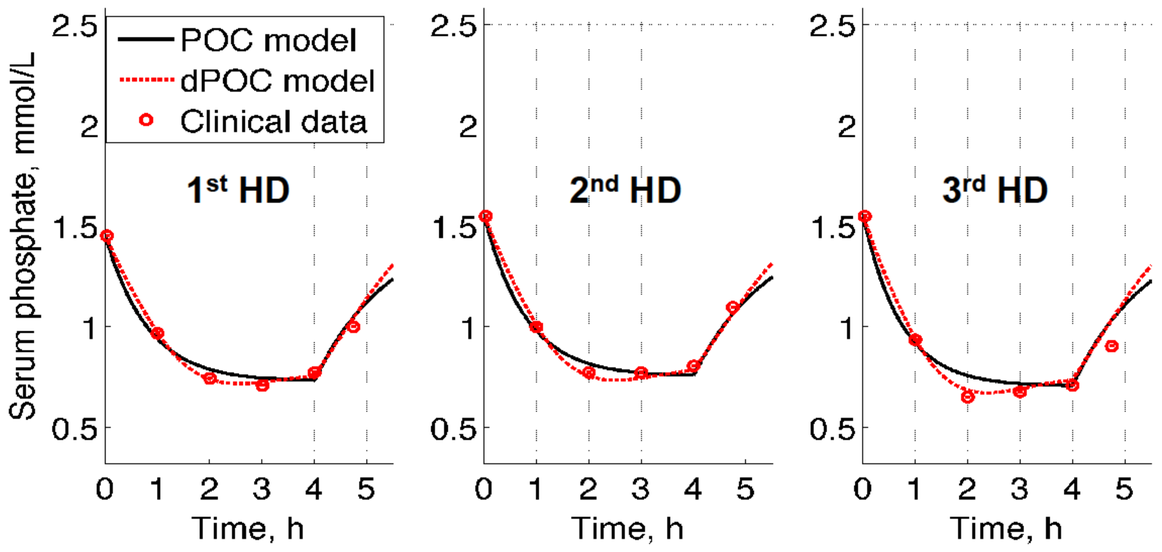

Fig. 3. Model predicted profiles for exemplary hemodialysis sessions with intradialytic serum phosphate rebound.

CONCLUSIONS

Extending the pseudo one-compartment model by the time delay in the activity of phosphate control loops provides significant qualitative improvement in the description of the kinetics of serum phosphate during HD. The intradialytic serum phosphate rebound is strongly associated with the speed of the phosphate feedback control system, i.e. the slower is the control response the larger is the intradialytic rebound.

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