

Dialysis-Related Acidemia and Acidosis by Dialysate: The Forgotten Issue of CO₂ Overload from Dialysate

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Dear Editor,

During hemodialysis, the arterial line of the extracorporeal circuit allows for easy access to arterial blood sampling [1], but may return unexpected findings.

We would like to briefly comment on the unique acid-base pattern featured by high partial pressure of carbon dioxide (pCO₂) not associated with hypoxia. Hypercapnia causes a decrease in pH. At a glance, hypercapnic acidemia calls up respiratory acidosis, but hypercapnia from lung failure is closely associated with hypoxia. There is another circumstance of hypercapnia featured by normal oxygenation and lung function. This non-hypoxic hypercapnic acidemia may be called 'dialysis-related acidemia' [2–4].

During bicarbonate-dialysis there is a gain of CO₂ [5]. After mixing of the acid concentrate with the bicarbonate-containing solution, the pCO₂ in the dialysate ranges between 80 and 100 mm Hg [3, 6, 7], whereas arterial pCO₂ commonly is below 40 mm Hg (as a result of the ventilatory response to the metabolic acidosis in kidney failure). This leads to high CO₂ dialysance and thus to a sizable increase in pCO₂ in

blood returning to the patient [2–4, 8]. This hypercapnia lowers pH despite high bicarbonate concentration but without hypoxia, unless lung disease coexists. The added CO₂ normally is quickly removed by respiration and systemic pCO₂ remains essentially unaffected.

Having said that, 'dialysis-related acidemia' is the typical acid-base pattern of blood exiting the filter (venous line) in bicarbonate dialysis (fig. 1a).

If dialysis-related acidemia is found in the arterial line, then vascular access should be inspected for correct needle placement or fistula malfunction because recirculation occurs when blood exiting the filter is not flowing into systemic blood but is reentering the extracorporeal circuit [9]. A more serious problem can occur if dialysis-related acidemia of venous line is coupled with underlying pulmonary or cardiac disease. If the patient is unable to breath away the CO₂ added from the dialysate (fig. 1b), then hypercapnic acidosis will occur or be exacerbated [2, 4]. In such a case, the label 'acidosis by dialysate' should be used because the mixed acidosis found in the arte-

rial line is due to CO₂ load from dialysate overcoming CO₂ removal by the lung. With this type of mixed acidosis, hypoxia is also commonly present. An important clue would be a pre-dialysis pCO₂ above normal value. Such a disorder cannot be identified without measurement of pCO₂, pH, and bicarbonate in arterial blood. Shortness of breath may be unmasked by starting the dialysis session and may not be relieved by oxygen therapy. Decreased myocardial contractility and vasodilatation secondary to hypercapnia may be clinically evident. Most notably, the symptoms should disappear (slowly) by simply stopping dialysate flow [4, 10]. The usual acid-base pattern is mixed metabolic and respiratory acidosis with severe fall in pH and hypoxia. Recognition of 'dialysis-related acidemia' and 'acidosis by dialysate' as different entities should lead to increased awareness of these patterns.

Disclosure Statement

None declared.

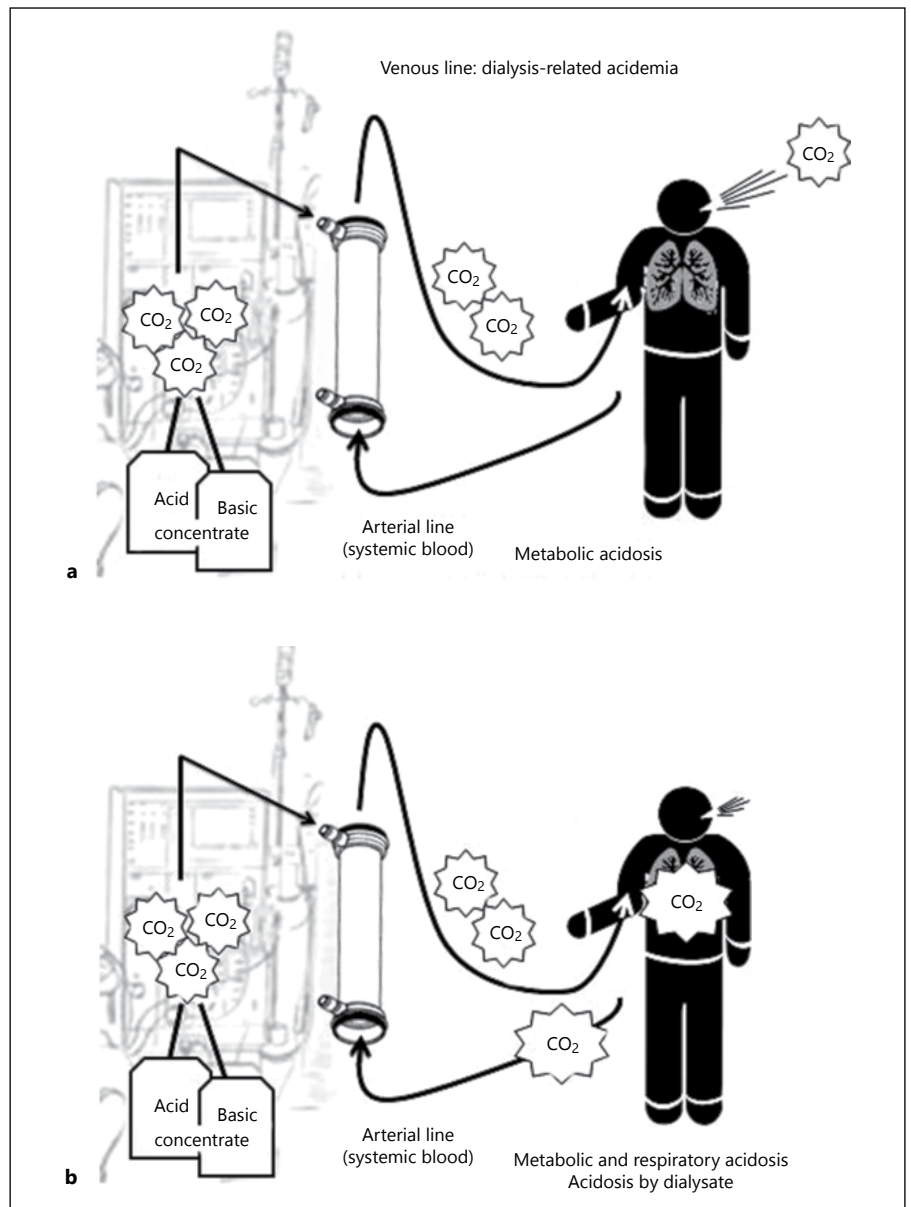


Fig. 1. a The course of CO₂ during bicarbonate dialysis. **b** Hypercapnic acidosis when the lung is unable to breath away the burden of CO₂ from the dialysate.

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