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Metabolic Alkalosis in Pregnant Patient Due to Citrate Load with Plasma Exchange

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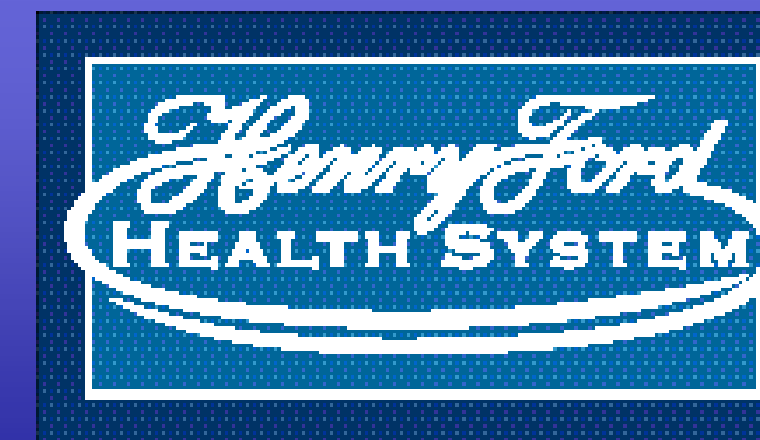
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SEVERE METABOLIC ALKALOSIS IN PREGNANT PATIENT DUE TO CITRATE LOAD WITH PLASMA EXCHANGE

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INTRODUCTION

Sodium citrate has been widely utilized as an anticoagulant in plasmapheresis. Diffuse alveolar hemorrhage (DAH) can be rapidly fatal and responds to therapeutic plasma exchange in 90% of patients. Metabolic alkalosis has been described in patients receiving PP. We report a rare case of iatrogenic citrate toxicity leading to profound metabolic alkalosis in a pregnant patient with normal kidney function.

CASE DESCRIPTION

A 22-year-old 16-week pregnant female admitted to intensive care unit with fever, rash and body aches. She was intubated due to respiratory failure secondary to DAH. Autoimmune workup and skin biopsy revealed a new diagnosis of systemic lupus erythematosus. Steroids and daily PP were initiated. On admission, patient's height was 150 cm and she weighed 71 kg.

Laboratory workup showed stable serum creatinine levels between 0.21 mg/dl and 0.51 mg/dl.

Patient received six daily PP treatments. Three days after initiation of PP, she was noted to have an increase in serum bicarbonate (HCO_3^-) level from 23 to 42 mmol/L. Patient also developed acute pancreatitis with lipase elevation to 1688 IU/l.

ABG showed pH of 7.55, pCO_2 46.2 mmHg. Peak pH was 7.62, following which she received one dose of acetazolamide.

Iatrogenic effective bicarbonate load with plasma exchange led to elevation of HCO_3^- to a critical level. Her HCO_3^- levels returned to baseline upon completion of apheresis.

ILLUSTRATIONS

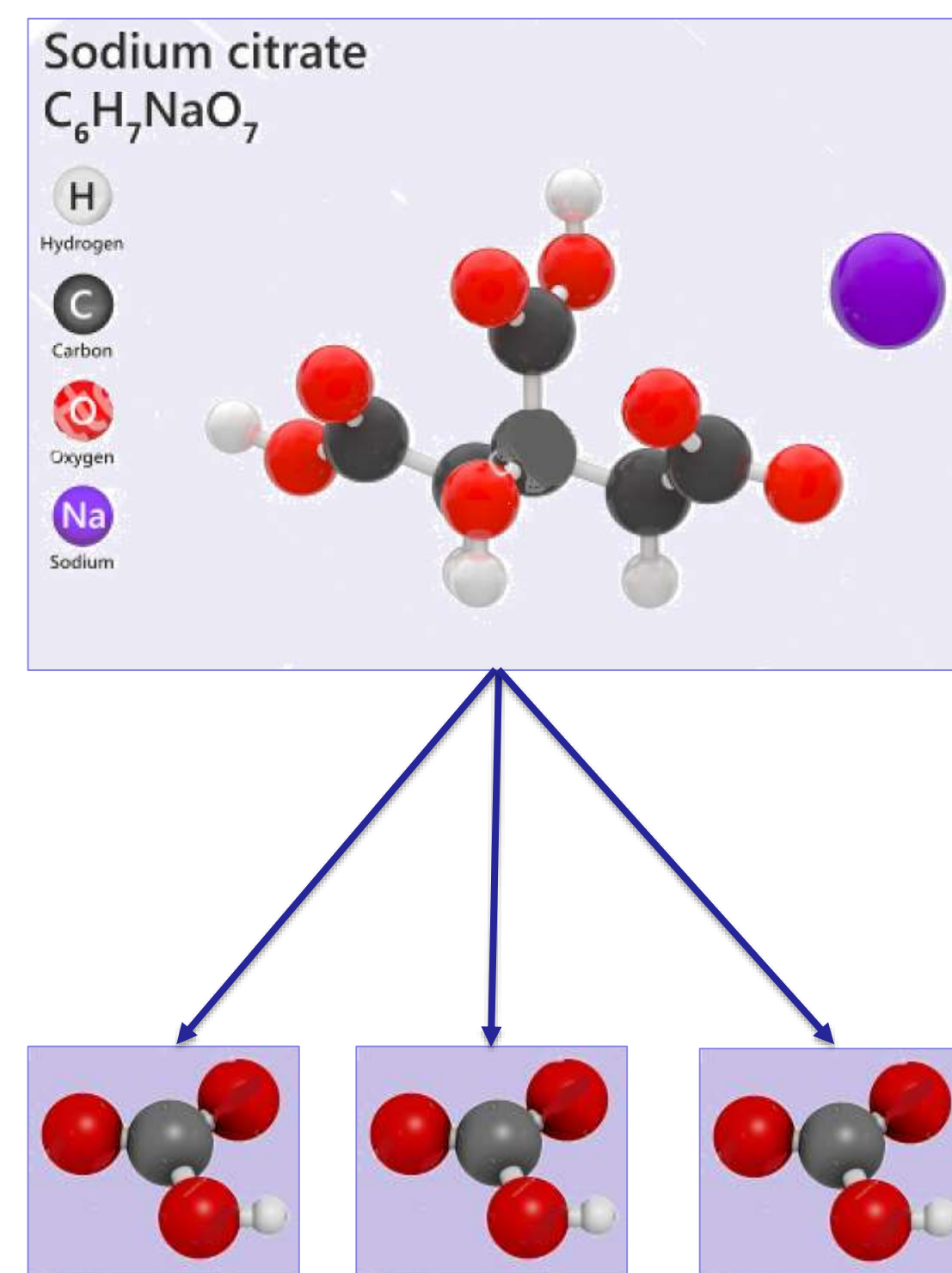


Figure 1. One citrate molecule conversion to three molecules of bicarbonate



Figure 2. Plasma exchange circuit

Day	1	2	3	4	5	6
Citrate load, ml	316	293	308	313	366	368
Serum HCO_3^- mmol/L	28	36	37	42	35	36
pH	7.51	7.49		7.55	7.62	7.52

Figure 3. Laboratory data

DISCUSSION

Volume of distribution of hydrophilic substances is increased by 40-50% in pregnancy.

PP offers a replacement of one plasma volume in 24 hours. Total citrate anticoagulant load was 294 mmol in the first four days.

Under normal conditions citrate is rapidly metabolized in the liver producing bicarbonate which is eliminated by urine.

One molecule of citrate can be converted to three molecules of bicarbonate, therefore total bicarbonate load was approximately 882 mmol.

The rapid increase in patient's HCO_3^- resulted from this bicarbonate load.

Parameters for bicarbonate distribution and maximal excretion have not been well described in pregnancy.

Our patient developed volume contraction in the settings of acute pancreatitis, which possibly affected bicarbonate excretion.

Citrate delivery needs to be protocolized and monitored closely to make it safe and effective.

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