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An unrecognised case of metabolic acidosis following neobladder augmentation cystoplasty

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ABSTRACT

INTRODUCTION: We present a case where there was a delay in the diagnosis of severe metabolic acidosis in a patient with an orthotopic neobladder. There are a growing number of patients with orthotopic neobladders and a wider range of clinicians are encountering these patients. A delay in the diagnosis can lead to significant morbidity but if identified early it can be easily treated.
PRESENTATION OF CASE: A 59-year old patient with a recent neobladder augmentation cystoplasty was admitted under the medical team with a metabolic acidosis which was incorrectly presumed to be secondary to urosepsis. His condition rapidly deteriorated until a surgical review identified hyperchloremic metabolic acidosis secondary to neobladder augmentation. The patient required admission to the intensive care unit where he was treated with intravenous alkalising therapy which produced rapid metabolic improvement. Following a full recovery, he underwent neo-bladder excision and ileal conduit formation.
DISCUSSION: Hyperchloraemic metabolic acidosis develops due to the bowel segment absorbing urinary constituents including ammonium, hydrogen ions and chloride in exchange for sodium and bicarbonate. It can be diagnosed by careful interpretation of the arterial blood gas and calculation of the anion gap. This hyperchloraemic metabolic acidosis can be corrected with alkalizing agents combined with catheterisation.
CONCLUSION: Hyperchloremic metabolic acidosis is a well-established complication of urinary diversion. Patient with orthotopic neobladder with high residual urine and large capacity are at even higher risk of metabolic acidosis. This information should be clearly documented in the post-operative discharge documentation to ensure early recognition by non-specialists.

1. Introduction

Orthotopic neobladders act as internal urinary reservoirs connected to the native urethral sphincter and are constructed using segments of the terminal ileum or colon. There are a growing number of patients with orthotopic neobladders and a wider range of clinicians are encountering these patients. When bowel segments are used to store or divert urine, a hyperchloremic metabolic acidosis is a recognised complication but is sufficiently rare that it may not be recognised. A delay in the diagnosis can lead to significant morbidity but if identified early it can be easily treated.

We present a case where there was a delay in the diagnosis of severe metabolic acidosis in a patient with an ileal neobladder. The case highlights the acid-base disturbance which can arise following urinary diversion and emphasises the importance early recognition and referral the original surgical team.

2. Case presentation

A 59-year-old man underwent a radical cystoprostatecomy and ileal neobladder (Studer Pouch) reconstruction for G2pT2 bladder cancer. A 60 cm distal ileal segment was isolated approximately 25 cm proximal to the ileocaecal valve. The proximal 10 cm of ileum was left intact to form the afferent limb and the remainder was opened along the antimesenteric border. The free edges were sutured to create the neobladder and a standard ureteroileal anastomosis was performed.

Post-operative recovery was complicated by persistent urinary incontinence and urodynamics at 9 months showed a small capacity neobladder with high pressures (maximum functional capacity 270 ml, pressure at maximum capacity of 35 cm H_20). A neobladder augmentation cystoplasty was undertaken at 12 months which increased pouch capacity to approximately 550 ml.

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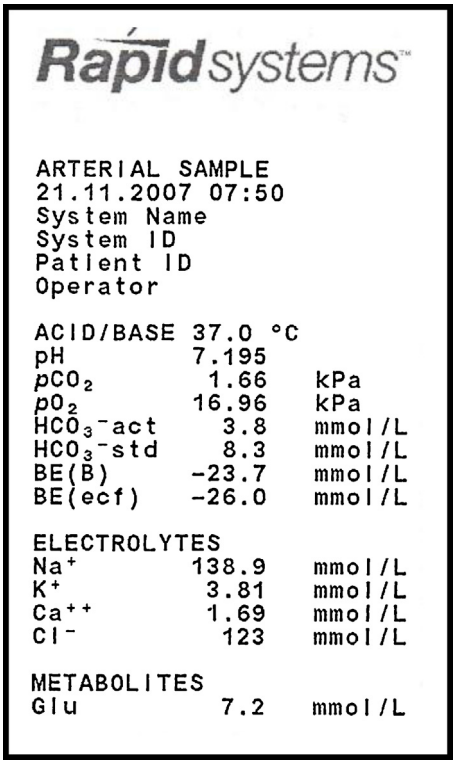
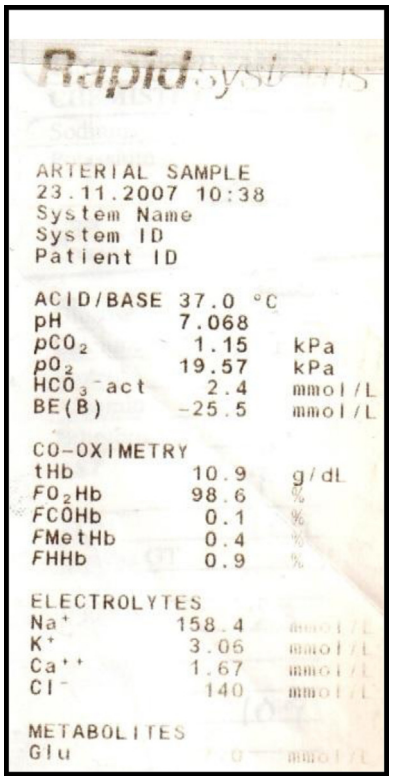


Rapid systems

ARTERIAL SAMPLE
21.11.2007 07:50
System Name
System ID
Patient ID
Operator

ACID/BASE	37.0 °C	
pH	7.195	
pCO_2	1.66	kPa
pO_2	16.96	kPa
HCO_3^-act	3.8	mmol/L
HCO_3^-std	8.3	mmol/L
BE(B)	−23.7	mmol/L
BE(ecf)	−26.0	mmol/L
ELECTROLYTES		
Na^+	138.9	mmol/L
K^+	3.81	mmol/L
Ca^{++}	1.69	mmol/L
Cl^-	123	mmol/L
METABOLITES		
Glu	7.2	mmol/L

Fig. 1. ABG on admission.



ARTERIAL SAMPLE
23.11.2007 10:38
System Name
System ID
Patient ID

ACID/BASE	37.0 °C	
pH	7.068	
pCO_2	1.15	kPa
pO_2	19.57	kPa
HCO_3^-act	2.4	mmol/L
BE(B)	−25.5	mmol/L
CO-OXIMETRY		
tHb	10.9	g/dL
FO_2Hb	98.6	%
FCOHb	0.1	%
FMetHb	0.4	%
FHHb	0.9	%
ELECTROLYTES		
Na^+	158.4	
K^+	3.05	
Ca^{++}	1.67	
Cl^-	140	
METABOLITES		
Glu		

Fig. 2. Repeat ABG.

The patient presented to the emergency department 8 weeks post operatively with dyspnea at rest and right loin pain. His past medical history was significant for polycystic kidney disease and he was being treated for a urinary tract infection with oral antibiotics. On examination the patient was cachectic with dry mucous membranes. He was tachypaenic (26 breathes per minute), tachycardic (108 beats per minute) and hypotensive (92/64 mmHg). Cardiorespiratory and abdominal examinations were unremarkable and the midline laparotomy incision was well-healed with no signs of infection.

Blood tests demonstrated a leucocytosis ($14.1 \times 103/\mu l$) and mildly raisedC-Reactive Protein (23.3 mg/dl). Biochemistry revealed a mild hypokalaemia (3.3 mmol/L), an elevated creatinine (304 μmol/L from abaseline 98 μmol/L and an elevated urea (33.1 mmol/L). All other electrolytes and full blood count including C-Reactive Protein were within normal limits. Arterial blood gas sampling demonstrated an uncompensated metabolic acidosis with a normal anion gap of 12.1 mEq/l (Fig. 1). Urinalysis revealed microscopic haematuria (100×103 ml) and sterilepyuria (20 leukocytes/mm3). The ECG showed a sinus tachycardia andthe chest X-ray was normal.

The initial diagnosis in the emergency department was thought to besevere urosepsis with acute kidney injury. He was admitted to a general medical ward and treated with broad-spectrum antibiotics, urinary catheter and crystalloid fluid resuscitation. Over 48 h the patient became confused, lethargic and in increasing respiratory distress. A repeat arterial blood gas showed a worsening hyperchloremic metabolic acidosis (Fig. 2). His vital signs continued to deteriorate and he became unresponsive with a Glasgow Coma Score of 8/15 (E2V1M5). The family were informed that there had been a poor response to treatment for urosepsis and that there was a high risk of mortality.

At this point the original surgical team who performed the neobladder formation was consulted. The hyperchloremic metabolic acidosis was identified as being a reversible metabolic complication from neobladder augmentation. The patient was transferred to the intensive care unit where he responded to sodium bicarbonate 8.4% infusion at 200 ml/h. After 4 h he regained consciousness and repeat ABGs showed an improving metabolic acidosis. He remained on the intensive care unit for a further 3 days and additional sodium bicarbonate 1.26% infusions were required to maintain his acid-base balance. The patient was discharged with oral sodium bicarbonate and after a period of recuperation he underwent neo-bladder excision and ileal conduit formation. He had no further episodes of metabolic acidosis and has completed 4 years follow-up without complications.

3. Discussion

Urinary diversions can be separated into noncontinent cutaneous diversions, continent cutaneous diversions, and orthotopic neobladders. Orthotopic neobladders act as internal urinary reservoirs connected to the native urethral sphincter and are constructed using segments of the terminal ileum or colon. When bowel segments are incorporated into the urinary tract a range of metabolic effects are encountered. The severity of the metabolic complications depends on the type of bowel segment used, the surface area exposed and the contact time between urine and bowel mucosa.

Neobladders have increased risk of hyperchloraemic metabolic acidosis as urine is in contact with bowel mucosa for prolonged periods of time [1]. In the early post-operative period a mild metabolic acidosis can be identified in up to 70% of patients [2]. Most cases are subclinical but patients with impaired renal function have higher risk of developing a persistent metabolic acidosis due to an impaired compensatory mechanism to metabolic challenges [3]. Severe metabolic acidosis requiring re-admission is less common and is reported as occurring in approximately 1% of patients [4]. In the literature there is a single previously reported case where admission to the intensive care unit was required due to the severity of the metabolic acidosis [5].

A hyperchloraemic metabolic acidosis develops due to the bowel segment absorbing urinary constituents including ammonium, hydrogen ions and chloride in exchange for sodium and bicarbonate [6]. It can be diagnosed by careful interpretation of the arterial blood gas. A metabolic acidosis is characterised by a decrease in arterial pH (<7.35) due to a reduction in serum bicarbonate concentration (<22 mmol/L). There are many causes of metabolic acidosis and this case emphasizes the importance of calculating the anion gap:

$$\text{Aniongap} = \text{Na} - (\text{Cl} + \text{HCO}_3)$$

In the present case there was a normal anion gap which could have helped exclude the initial diagnosis of severe urosepsis. A normal anion gap has a narrow differential diagnosis which includes only severe diarrhoea, Type 2 renal tubular acidosis and urinary diversion complications. Sepsis results in a raised anion gap secondary to lactic acidosis although a wide range of other conditions associated with increased acid production cause a raised anion gap.

A further learning point is the importance of involving the primary surgical team early in the care of patients who have undergone a recent operation. Discussion with the surgical team at this point could have established the metabolic acidosis was a post-operative complication and allowed early treatment to be commenced. It is also the responsibility of the surgical team to provide clear and detailed information regarding potential complications on discharge documents. This should ensure early recognition by non-specialist teams.

If diagnosed early hyperchloraemic metabolic acidosis can be easily corrected with oral alkalizing agents [1]. Sodium bicarbonate 2–6 gm daily is effective at restoring acid–base balance and should be considered even if the patient is asymptomatic as long term metabolic acidosis can cause bone demineralisation and osteomalacia [7]. In severe symptomatic acidosis or a pH below 7.2 administration of intravenous sodium bicarbonate is required combined with catheterization is to reduce the contact of urine with the bowel mucosa.

In severe case, neobladder excision and formation of an ileostomy should be considered. In the present case, a hyperchloraemic metabolic acidosis only developed following augmentation of the neobladder. There were no metabolic complications with the original neobladder which had a smaller capacity. The addition of an extra bowel segment increased the surface area of the neobladder and prolonged the contact time between urine and bowel mucosa allowing extensive metabolic exchange. We recommend extreme caution and careful deliberation when deciding to augment a neobladder due to the risk of metabolic complications. Instead, conversion to an ileal conduit should be the diversion of choice in patients not tolerating a neobladder. This allows continuous drainage of urine and reduces the metabolic complications.

4. Conclusion

This cases highlights the risks of hyperchloraemic metabolic acidosis in patients with orthotopic neobladders particularly with a large capacity following augmentation cystoplasty. The delay in the diagnosis led to significant morbidity but if it had been identified early it could have been easily treated with oral sodium bicarbonate. Learning points include the importance of clear post-operative discharge documentation to ensure such complications are recognised early by non-specialists. The case emphasises the importance of involving primary surgical team early in the care of patients who have undergone a major recent operation.

Conflicts of interest

None.

Funding

None.

Consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

Author contribution

D Eldred-Evans: Writing.
F Khan: Reviewed initial draft.
J Abbaraju: Review final draft.
S Sriprasad: Supervising project.

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