Changes in acid-base metabolism after orthotopic bladder substitution: ileal neobladder compared with ileal conduit

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Introduction

- The use of intestinal segments for orthotopic bladder replacement after radical cystectomy has become common. Several investigators have reported the frequent incidence of normal anion gap metabolic acidosis and electrolyte disturbance in those patients. The main cause of the metabolic acidosis is absorption of ammonium through the intestinal mucosa, but secretion of bicarbonate play minimal role in the acid load.
- Several factors appear to exert an important influence on the severity of the metabolic acidosis seen, including the type of urinary diversion and the portion of intestine used. But, metabolic acidosis is much less likely with ureteoileostomy, since rapid drainage of urine into an ileostomy bag means that contact time between the urine and intestine is normally too short for significant changes in urinary composition to occur.
- To analyze the patterns of metabolic acidosis and hypokalemia in patients following the construction of an ileal neobladder compared with ileal conduit method
- To search for the risk factors affecting metabolic acidosis.

Methods

- The medical records of 67 patients who underwent radical cystectomy and urinary diversions for invasive bladder cancer were analyzed.
- Patients with any illness such as severe pulmonary disorder or taking any medication that could lead to metabolic acidosis were excluded.
- Acid-base balance, serum electrolytes, renal function and effects of renal function on acid-base metabolism were compared in patients with ileal neobladder and ileal conduit.
- Metabolic acidosis was defined as a disorder associated with a low pH (<7.35) and a low bicarbonate concentration (<22 mEq/L). The normal plasma albumin-corrected anion gap (AG) had been considered to range between 7 and 13 mEq/L. The plasma albumin-corrected AG was calculated from the following formula:

Albumin-corrected AG = Na - (Cl + HCO3) + 2.5 (4.0 - serum albumin)

Results

- The urinary diversions were performed using ileal neobladder for 41 patients or ileal conduit for 26 patients (Fig. 1). No significant differences were observed in their baseline characteristics and preoperative variables examined between the ileal neobladder and ileal conduit groups, except for age (Table 1).
- Metabolic acidosis was detected in 16 patients (39.0%) with ileal neobladder and in 6 patients (23.1%) with ileal conduit. Metabolic acidosis with normal anion gap occurred more often in ileal neobladder group (9 patients, 22.0%) than ileal conduit group (1 patient, 3.8%), however the differences were not statistically significant (Fig. 2).
- The close association between the serum creatinine level and total CO2 ($p<0.01$, $r=-0.249$) were demonstrated (Fig. 3). Acute kidney injury and all-cause mortality were frequent in patients with metabolic acidosis, especially ileal neobladder group (Table 2).
- The risk of development of metabolic acidosis by multivariate analysis was associated with acute kidney injury, under the adjustment with other factors (Table 3).
- Postoperative hypokalemia in both groups showed no differences in statistically significance (Fig. 4).

Table 1. Baseline characteristics and pre-operative blood chemistry profile of patients

	Ileal neobladder, n = 41	Ileal conduit, n = 26	*p* value
Age (years)	62.3 ± 8.6	68.3 ± 7.3	0.004
Males	34 (82.9%)	18 (69.2%)	0.190
Diabetes mellitus	8 (19.5%)	1 (3.8%)	0.138
Clinical stage ≤ T1c ≥ T2a	 12 (38.7%) 19 (61.3%)	 2 (11.8%) 15 (88.2%)	0.095
Metastatic cancer	0	4 (20.0%)	0.019
Robot-associated surgery	13 (31.7%)	6 (23.1%)	0.445
Serum creatinine (mg/dL)	1.03 ± 0.25	1.11 ± 0.41	0.400
eGFR (by MDRD equation)	78.1 ± 16.1	73.0 ± 25.2	0.365
Serum potassium (mEq/L)	4.2 ± 0.4	3.9 ± 0.5	0.025
pH	7.43 ± 0.04	7.44 ± 0.04	0.313
$PaCO_2$ (mmHg)	37.1 ± 4.6	35.9 ± 4.0	0.345
$HCO3^-$ (mEq/L)	25.7 ± 2.8	25.1 ± 2.3	0.375
Base excess (μmol/L)	1.63 ± 2.53	1.20 ± 2.25	0.511
Anion gap	11.2 ± 3.0	11.7 ± 2.3	0.450

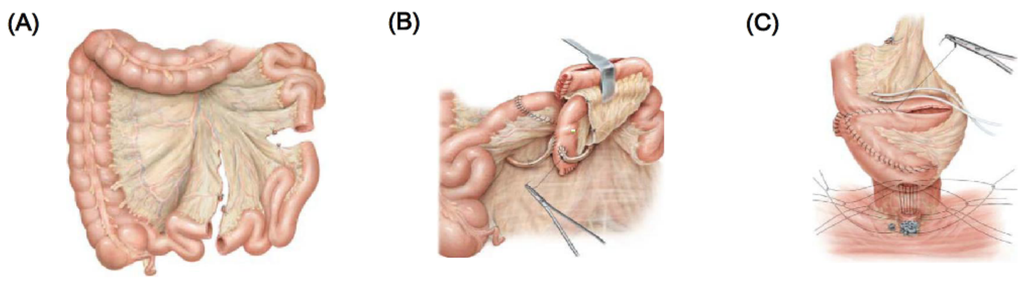

Fig. 1. Orthopedic neobladder operation technique. (A) An ileal segment is isolated about 25 cm from ileocecal valve. (B) End-to-side anastomosis of ureter to the unopened part of the tubular segment. (C) Anastomosis of the neobladder to the urethra.

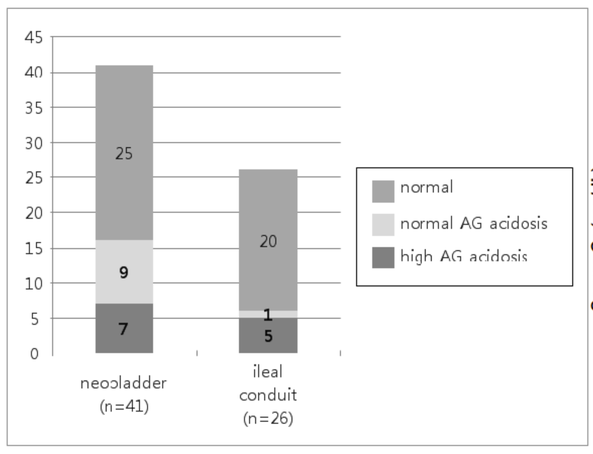

Fig. 2. Postoperative metabolic acidosis in patients with urinary diversion.

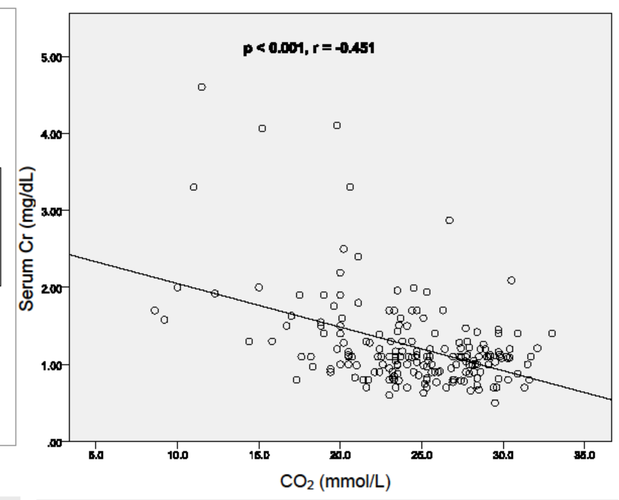

Fig. 3. Correlation of serum Cr and total CO_2 in patients with neobladder and ileal conduit.

Table 2. Renal function and clinical outcome according to metabolic status.

	Ileal neobladder Metabolic acidosis			Ileal conduit Metabolic acidosis			Total Metabolic acidosis		
	+	-	*p*	+	-	*p*	+	-	*p*
Serum Cr (mg/dL)	2.24±1.08	1.29±0.46	0.004	1.98±1.31	1.39±0.80	0.191	2.17±1.12	1.33±0.63	0.003
AKI	10 (62.5%)	2 (8.0%)	< 0.001	3 (50.0%)	3 (15.0%)	0.112	13 (59.1%)	5 (11.1%)	< 0.001
Death	5 (31.3%)	1 (4.0%)	0.026	0	4 (20.0%)	0.324	5 (22.7%)	5 (11.1)	0.210

Table 3. Multivariate analysis for predictive factors of post-operative metabolic acidosis

Variables	Hazard ratio (95% confidence interval)	*p* value
Unadjusted		
Age	1.00 (0.95-1.07)	0.907
Male gender	1.03 (0.30-3.49)	0.963
Diabetes mellitus	1.78 (0.43-7.41)	0.461
Ileal neobladder (vs. Ileal conduit)	0.47 (0.16-1.42)	0.176
Preoperative estimated GFR	0.98 (0.95-1.01)	0.111
Preoperative HCO_3^-	0.87 (0.68-1.10)	0.238
Acute kidney injury	11.56 (3.28-40.72)	< 0.001
Urinary tract infection	1.15 (0.41-3.21)	0.796
Urinary tract obstruction	2.29 (0.76-6.90)	0.139
Adjusted		
Age	1.00 (0.90-1.12)	0.968
Ileal neobladder (vs. Ileal conduit)	1.68 (0.34-8.36)	0.526
Preoperative estimated GFR	0.97 (0.93-1.01)	0.162
Preoperative HCO_3^-	0.78 (0.55-1.09)	0.148
Acute kidney injury	21.9 (3.9-123.9)	< 0.001

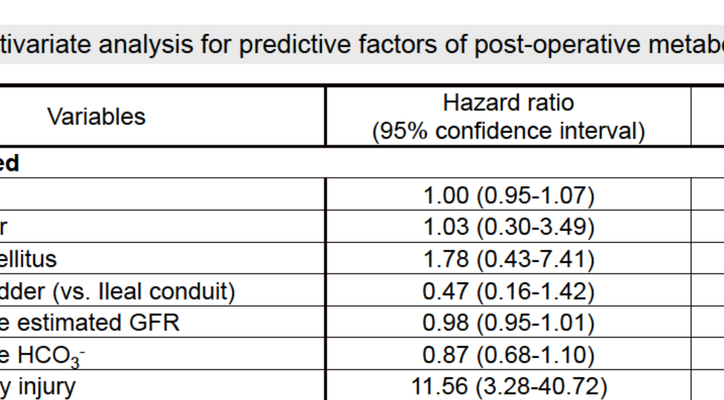

Fig. 4. Incidence of postoperative hypokalemia.

Conclusions

- Patients with ileal neobladder tended to develop metabolic acidosis with normal anion gap more frequently than those with ileal conduit, but there was no statistical significance.
- The degree of metabolic acidosis was closely associated with the renal function.

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