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Case Report

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# Cisplatin induced kidney injury and renal tubular acidosis in a cervical cancer patient: review of literature and case report

## Abstract

Cisplatin, a chemotherapy agent based on platinum, is regarded as one of the most effective medications for treating various cancers. A significant and dose-restricting side effect of cisplatin is nephrotoxicity, which was identified early in its clinical application. Various mechanisms have been identified that contribute to kidney dysfunction following cisplatin exposure, including toxicity to tubular epithelial cells, vasoconstriction in the renal microcirculation, and proinflammatory responses. Fanconi syndrome manifests as an impairment in the proximal tubular reabsorption of glucose, amino acids, uric acid, phosphate, and bicarbonate. It is recognized that most acquired cases of Fanconi syndrome are induced by medications, with many instances attributed to antineoplastic drugs, including cisplatin. In this report, we discuss a complex case involving multifactorial acute kidney injury that includes Fanconi syndrome induced by cisplatin.

**Case report:** A 43-year-old female suffered from advanced cervical cancer encroaching on the ureters bilaterally with bilateral obstructive uropathy, insertion of bilateral nephrostomy tubes. After her 1st session of chemotherapy with cisplatin and radiotherapy, the patient was admitted to the ICU due to septic shock and hypovolemia secondary to severe diarrhoea. Clinical symptoms of dehydration and septic shock were declared secondary to E. coli with Extended-Spectrum Beta-Lactamase (ESBL) which is an enzyme produced by some bacteria that makes them resistant to many common antibiotics, especially penicillin and cephalosporins leading to urosepsis. Fluid resuscitation and the use of positive inotropes were done. Serum creatinine rose to 8.2 mg/dl, Blood urea nitrogen (BUN) 89 mg/dl, HB 7 g/dl, Platelets 44000/ul. Acute Kidney injury was declared secondary to pre-renal hypovolemic, septic shock, and post-renal obstruction of the left nephrostomy tube. Her blood pH was 7.28, Serum bicarb 12, while her urine pH was 9 with albuminuria and no glucosuria. Type 2 renal tubular metabolic acidosis was declared, and in view of new-onset albuminuria, Urine Albumin to Creatinine Ratio of >150, acquired Fanconi syndrome secondary to cisplatin was declared. She was started on IV Sodium Bicarbonate correction, a high oral dose of 3 gm per day sodium bicarbonate, and serum bicarbonate was corrected. The patient's urine output improved after fluid resuscitation, and her serum creatinine improved dramatically from 8.2 to 3.2 mg/dl in four days and normalized to 0.9 mg/dl in 10 days. In conclusion, the acquired form of Fanconi syndrome is not uncommon and is under-diagnosed in various medical situations. Identification and early management dramatically improve renal survival.

**Keywords:** chemotherapy, nephrotoxicity, cisplatin, acute kidney injury

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## Introduction

Cisplatin is a platinum-based chemotherapy compound that is an effective therapy for many carcinomas and sarcomas, as well as lymphomas. Nephrotoxicity is its major adverse effect, and it is dose-related, causing apoptosis and necrosis of cells.[1] Multiple mechanisms are attributed to Cisplatin renal injuries and can involve multiple renal compartments, including blood vessels, glomeruli, and most commonly, the tubules.[1] Nephrotoxicity, if detected early, is generally reversible, but it can be permanent with late interference.[2]

A malfunction in the proximal tubular reabsorption of glucose, amino acids, uric acid, phosphate, and bicarbonate is the hallmark of Fanconi syndrome. Both inherited and acquired causes can cause it; the most frequent acquired instances were linked to antineoplastic drugs, including cisplatin. Because the tests now employed to follow patients on cisplatin lack sensitivity and appropriate timing, the prevalence of identifying Fanconi syndrome is underestimated.[3] Here, we describe a complicated case of multifactorial renal damage and the development of Fanconi syndrome caused by cisplatin.

## Case report

A 43-year-old woman experienced bilateral ureteric obstruction as a result of cervical cancer, which led to bilateral obstructive uropathy and the insertion of bilateral nephrostomy tubes. The patient collapsed during the first chemotherapy session with cisplatin and radiation. The patient was transferred to the critical care unit and was first diagnosed with hypovolemic shock due to acute diarrhoea. The patient was focused and aware. The patient's blood pressure was 84/50, her heart rate was 110, her temperature was 38, she had clinical signs of dehydration, and a positive urine culture for Escherichia coli may indicate sepsis. Fluid resuscitation and positive inotropes were used in initial management. Her initial investigations revealed a jump of serum creatinine from 1.2 mg/dl to 8.2 mg/dl, Blood urea nitrogen (BUN) 89, Haemoglobin 7 g/dl, and platelets 44. Ultrasound

of the abdomen showed bilateral hydroureteronephrosis, moderate on the right with grade 1 nephropathy, and mild on the left with grade 4 nephropathy. Pre-renal hypovolemia, septic shock, and post-renal obstruction of the left nephrostomy tube—which was replaced with a new one—all contributed to the multifactorial acute kidney injury. Her blood pH was 7.28, her serum bicarbonate (HCO3) was 12 mEq/l, her serum potassium was low at 3.4 mg/dl, and her urine had a high pH of 9 with a new-onset urine albumin/creatinine ratio >150 mg/gm, no glucosuria, consistent with type 2 renal tubular metabolic acidosis.

Cisplatin-induced acquired Fanconi syndrome was identified. Serum bicarbonate was corrected with improving serum creatinine as part of therapy, which also included an intravenous correction of sodium bicarbonate and a high oral dose of 3 gm daily (Figure 1). Following fluid resuscitation, the patient's urine production increased to 1 ml/kg/hr, and her serum creatinine significantly reduced from 8.2 mg/dL to 3.2 in four days before normalizing to 0.9 mg/dl in fourteen days.

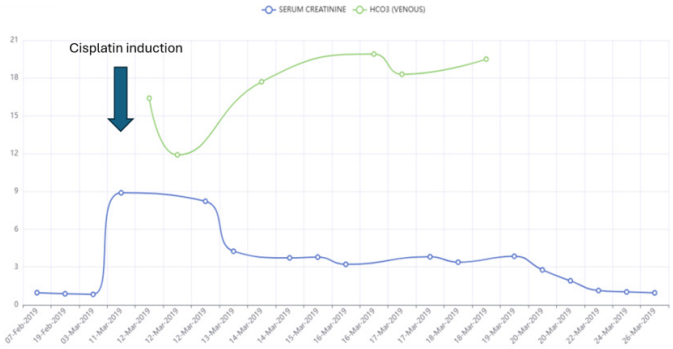


**Figure 1** Monitoring of serum creatinine and bicarbonate post-cisplatin induction and during the recovery phase. Showing the basal level of serum creatinine before the episode of obstructive uropathy, on the day of cisplatin induction, and during the recovery phase.

## Discussion

Our case is one of the challenging case scenarios of complex renal injuries, including pre-renal injury secondary to dehydration, post-renal acute obstructive kidney injury, as the patient had bilateral hydronephrosis. Metabolic acidosis can be secondary to acute kidney injury, either pre-renal or post-renal; however, in such cases, urinary pH will be either within 6-7. In our case, the finding of urinary pH 9 plus proteinuria directed the diagnosis toward renal injury secondary to cisplatin.

Active hydration with saline and simultaneous administration of mannitol before, during, and after cisplatin treatment significantly reduces cisplatin nephrotoxicity, and this strategy has been accepted as the standard of care for reducing the associated side effects.[4] The detailed mechanism surrounding the benefits of salt is uncertain, but volume expansion with saline or hypertonic saline may increase the rate of cisplatin excretion. In addition, salt provides a high concentration of chloride ions that competitively prevent the dissociation of the chloride ions from the cisplatin molecule, thereby reducing the formation of the reactive form of cisplatin.[5] A recent study demonstrated that saline does not reduce the cellular accumulation of cisplatin but instead activates a stress response within the cell that modifies sensitivity to cisplatin. The osmotic stress decreases the accessibility of cisplatin to DNA, induces proximal tubular cell resistance to the apoptotic pathway, and changes the metabolic activation of nephrotoxins. However, this approach may interfere with the antineoplastic activity of cisplatin by blocking tumoricidal effects.

In our approach, we preferred to use sodium bicarbonate for hydration and not sodium chloride (saline) to avoid the chloride overload as it interferes with the anti-tumour activity of cisplatin and aggravates the metabolic acidosis secondary to acquired Fanconi syndrome.

## Conclusion

Fanconi syndrome is a rare congenital tubular disorder; however, the acquired form is not uncommon and is underdiagnosed in various medical situations. Identification and early management of acquired Fanconi syndrome dramatically improves renal survival.

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## Conflicts of interest

The authors declare that there are no conflicts of interest.

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## References

1. Pabla N, Dong Z. Cisplatin nephrotoxicity: mechanisms and renoprotective strategies. *Kidney Int.* 2008;73(9):994–1007.
2. Perazella MA. Onco-nephrology: renal toxicities of chemotherapeutic agents. *Clin J Am Soc Nephrol.* 2012;7(10):1713–1721.
3. Oh GS, Kim HJ, Shen A, et al. Cisplatin-induced kidney dysfunction and perspectives on improving treatment strategies. *Electrolyte Blood Press.* 2014;12(2):55–65.
4. Cornelison TL, Reed E. Nephrotoxicity and hydration management for cisplatin, carboplatin, and ormaplatin. *Gynecol Oncol.* 1993;50(2):147–158.
5. Hanigan MH, Deng M, Zhang L, et al. Stress response inhibits the nephrotoxicity of cisplatin. *Am J Physiol Renal Physiol.* 2005;288(1):F125–F132.