

EMPHYSEMATOUS PYELONEPHRITIS (EPN) PRESENTING AS HYPEROSMOLAR HYPERGLYCAEMIC STATE (HHS) IN A POORLY CONTROLLED T2DM

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Abstract

Emphysematous pyelonephritis is a severe necrotizing infection of the renal parenchyma. It is commonly found as a complication of uncontrolled diabetes mellitus. EPN has a terrible prognosis unless promptly identified & treated aggressively. In this case a 40 years old woman with type 2 diabetes mellitus was admitted with multiple episodes of vomiting along with lower abdominal pain & burning micturition. She was non adherent to her insulin medication. The patient was subsequently diagnosed with HHS and later EPN. We managed the patient with IV fluids, Infusion insulin followed by S/C insulin, IV antibiotics, percutaneous drainage and nephrectomy. This case is important because emphysematous pyelonephritis presenting with hyperosmolar hyperglycaemic state as a complication of poorly controlled diabetes mellitus is rare.

INTRODUCTION

Emphysematous pyelonephritis is an aggressive necrotizing infection of renal parenchyma seen predominantly in diabetics. It is a urological emergency that carries high morbidity & mortality. It is usually caused by gram negative gas forming organisms, most commonly *Escherichia coli* (70%), *Proteus mirabilis*, *Klebsiella pneumoniae* and less commonly by anaerobes like *Clostridium septicum* and *Candida albicans*. Metanalysis shows only 5-10 % of EPNs are bilateral, with 52 % of cases affecting the left kidney. Male to female ratio is 1: 4-6 and an average age of presentation is 57 years. EPN is prevalent in patients with T2DM and in immunocompromised states. These immunocompromised states include chronic kidney disease, renal allograft, alcoholism, chronic steroid use or patients on chronic immunosuppressant medication. However almost 90% cases are found as a complication of uncontrolled T2DM or due to non-compliance to medication. EPN presenting as hyperosmolar hyperglycaemic state is not very common.

CASE PRESENTATION

My patient, Mrs XYZ, aged 40 years, female presented in our ER with chief complaints of

multiple episodes of vomiting along with lower abdominal pain for 1 day & burning micturition for last 7 days. She is a known case of T2DM (under Basal & Bolus Insulin) & Dyslipidaemia (under Rosuvastatin). She has a past history of hospital admission with urinary tract infection back in Nov 2023 with urine culture showed growth of *E. coli* managed conservatively with sensitive IV antibiotics. She has an alleged history of self-ingestion of oral antibiotics at home but no documents available. At the time of presentation, she is alert, conscious, cooperative. Her PR - 102/m, BP - 80/50, RR - 26/m, CBG - HI, ABG shows pH - 7.397, pCO₂ - 25.8, pO₂ - 110, HCO₃ - 15.9, Lactate 1.8. Urine dipstick shows Glucose ++ & Ketone body moderate. Her serum osmolarity was calculated 324. On P/A examination she has left renal angle tenderness. Other systems show no significant abnormalities. She was immediately shifted to ICU. She was given 2 bottles of NS (500ml) in bolus then IVF NS 4 hourly. Insulin infusion with Regular insulin started & titrated accordingly. Empirical antibiotic therapy with InjPiperacillin+Tazobactam was started. On next morning her blood reports shows Hb-8.4 gm/dl, TLC-10860/cumm, N91L6M2E1, ESR-47, CRP-24, Pro Calcitonin-25.6 ng/ml, FBS-536 mg/dl, Ur-72 mg/dl, Cr-1.4 mg/dl, Na-130 mEq/l, K-3.7 mEq/l, Mg-1.6mmol/l, Ca-8.3 mg/dl, PO₄-3.5 mg/dl,

HbA1c-14.1 %, LFT - low albumin(3.3gm/dl), Lipid profile - high Triglyceride(330mg/dl) & high LDL(77mg/dl)& Blood ketone - nil. Her Urine RE/ME shows Glucose 1+, Pus cell- 8-10/hpf, RBC-Numerous/hpf and no ketone body. Insulin infusion was titrated according to her CBG level. USG KUB done that shows a small reniform like kidney of 45 mm size in the left renal fossa. Left kidney was contracted without any mass, calculus or hydronephrosis. Later, on that night she developed severe pain abdomen. Her abdomen was distended with sluggish IPS. A continuous ryle's tube suction was given that shows bilious secretion. Surgery opinion was taken. They suspected sepsis induced paralytic ileus. Her urine output decreases over time. On next morning her blood report shows Hb-7.4 gm/dl, TLC-7460/cumm, N92L6M1E1, ESR-32, Ur-113 mg/dl, Cr-2.0 mg/dl, Na-128 mEq/l, K-3.0 mEq/l. She developed Acute Kidney Injury of pre renal type. Her hydration was increased as suggested by nephrologist. In the meantime, she was shifted from infusion insulin to sub cutaneous insulin regimen (basal + bolus). NCCT Abdomen was performed that shows gas in the left renal parenchyma suggestive of EPN. Her urine culture does not show any growth of bacteria. However, her blood culture shows growth of Escherichia coli sensitive to InjMeropenem&InjTigecycline. We escalated the antibiotic coverage to InjMeropenem&InjTigecycline from InjPiperacillin+Tazobactam. Later the patient was referred out to a higher center for further management. They performed Percutaneous nephrostomy first to drain the pus & by some time to decrease the blood septemia. Later radical nephrectomy was done to save the patient. She was managed conservatively in the ICU, then HDU and then in general ward with IV antibiotics & other supportive therapy.



Figure 1

DISCUSSION

In 1898, Kelly & McCullum reported the first case of kidney infection with gas accumulation. In 1962 Schultz and Klorfein named this disorder as emphysematous pyelonephritis. The pathogenesis of EPN is still unclear. It is believed that multiple factors including diabetes, elevated glucose level in kidney tissue, impaired renal circulation, urinary tract obstruction, decreased host immune function

and the presence of gas producing microbial infections could cause EPN. EPN most commonly affects the diabetic patients and is currently believed that the increased susceptibility to develop EPN in diabetic patient is due to impaired renal tissue perfusion. Since patients with uncontrolled diabetes tends to have elevated blood sugar & glycosuria these organisms thrive greatly in this environment. In a favourable environment these organisms cause fermentation of glucose & lactate to carbon dioxide which develops a necrotizing infection in the perinephric fat of the kidney. The major gas content found by investigators in EPN include nitrogen (60%), hydrogen (15%), carbon dioxide (5%) and oxygen (8%). CT scan is considered as the best method to diagnose EPN. The most commonly accepted CT classification system for EPN was proposed by Huang and associates in 2000. These authors suggested 4 classes of EPN:

- Class 1, gas is located only in the collecting system
- Class 2, gas is located within the renal parenchyma but without extension to the extra renal space;
- Class 3A, gas or an abscess spread to the perinephric space;
- Class 3B, gas or an abscess spread to the pararenal space; and
- Class 4, bilateral EPN or solitary kidney with EPN.

Management includes fluid resuscitation, glycemic control, broad-spectrum antibiotics, and treatment of comorbid conditions. For moderate to severe EPN, invasive procedures such as percutaneous drainage, debridement of the infected area can be done. However, for severe cases simple, complex, or radical nephrectomy must be done. EPN can be complicated by several diseases including emphysematous pyelitis (gas in the collecting system), emphysematous cystitis (gas in the wall of the urinary bladder), and shock. According to the research, patients experiencing secondary shock following EPN have a mortality rate of 54%. Patients with hemodynamic shock presenting with altered mentation, acute kidney injury requiring dialysis, and nutritional deficiency have a very high mortality rate. Polymicrobial infections and delay in empiric antibiotics are associated with higher mortality. Additionally, in individuals with EPN, hyperosmolar hyperglycaemic state is a significant predictor of death.

CONCLUSION

EPN is a rare necrotizing infection of the renal parenchyma with a high mortality rate if not treated promptly and aggressively. Uncontrolled diabetic patients are at higher risk of EPN. Therefore vigorous investigations including labs, imaging, and consultation should be performed to come up with a definitive diagnosis to treat the patients

appropriately and on time. E. coli and other gas forming bacteria are responsible for developing EPN in uncontrolled diabetic patients. In this case, the patient was non-compliant with her insulin regimen. Eventually, the patient developed EPN on the background of uncontrolled T2DM. Our patient improved significantly with aggressive inpatient medical & surgical (percutaneous drainage& nephrectomy) management. It is necessary to educate every patient about controlling high blood sugar with proper medical management to avoid uncontrolled diabetes-related severe complications like HHS or EPN.

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