Case Report

A Case of *Burkholderia cepacia* Sepsis in a postpartum patient with colonic perforation due to Ogilvie's syndrome: Case from a tertiary care hospital in Bangladesh

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Abstract:

Traditionally known as a nosocomial organism and preponderance to infect the immunocompromised patients or with preexisting structural damage in lung parenchyma[1], Burkholderia cepacia infection in an apparently immunocompetent host in ICU settings is not very commonly heard of. On the other hand, acute colonic pseudo-obstruction (Ogilvie's syndrome) is also a very rare surgical complication. Here we present a case of a postpartum lady who developed colonic perforation following caesarean section due to Ogilvie's syndrome and Burkholderia cepacia sepsis and eventually expired.

Keywords: *Burkholderia cepacia, Ogilvie's syndrome.*

Introduction:

Burkholderia cepacia complex, discovered by Walter Burkholder in 1949, refers to a group of catalase negative, non-lactose fermenting, aerobic gram negative rods[1,2]. In humans it is an opportunistic pathogen mainly seen in patients with cystic fibrosis or, immunocompromised states such as chronic granulomatous disease or, inpatients post lung transplantation[1-6]. Being resistant to multiple antimicrobials it is very notorious to treat. Ogilvie'ssyndrome,being a rare post operative complication, if not timely addressed and treated, can be lethal. This case report enumerates that these arevery rare combination and the unfortunate story of the deceased patient.

Case Report:

Mrs. X 23 years old 3rd gravidae from Shahrasti, Chadpur, had been initially admitted in a private hospital with full term pregnancy with laborpain. She delivered a healthy baby by caesarean section (CS) and the indication was previous 2 caesarean section (CS) and less fetal movement. Her initial postoperative period was uneventful, but from 6th postoperative day, she developed abdominal pain, constipation, vomiting and abdominal distension, which was provisionally treated conservatively for acute intestinal obstruction for 5 days and on 12th POD when she developed hypotension and desaturation,was shifted to Intensive care unit (ICU), Cumilla medical college hospital. On our initial impression, she was anaemic, febrile, hypotensive (BP was 80/60mmHg and did not respond to fluid challenge), hypoxic (initial SpO_2 was 84% with 6 L of oxygen), edematous and had labored breathing. She also had severe tenderness on abdomen palpation and board like rigidity over the whole abdomen. She was provisionally diagnosed as perforation of gas containing hollow viscus preceded by acute intestinal obstruction and septic shock and treated with supplemental oxygen, meropenem, metronidazole, noradrenaline, analgesics, nasogastric suction and other supportive measures. Her conditions did not at all improve and surgery consultation was taken the next day. She was clinically and radiologically confirmed as a case of perforation. Immediately she was rushed into emergency OT and laparotomy was done, there was a large perforation over the ascending colon and whole peritoneal cavity was flooded with fecal matter. Perforation was repaired, peritoneal toileting was done and temporary ileostomy was done. The patient was kept on invasive ventilation. Patients routine blood tests and plain X ray abdomen done on admission into ICU is attached below.

Table 1: Patient's Routine Lab Parameters

Parameters	Results	Normal value
Hb%	8.8 gm/dl	12-16gm/dl
WBC	19,300 / cumm	4.000-11,000/ cumm
Neutrophil	84%	40-75%
Lymphocyte	13%	20-45%
MCV	72.3fl	76-94fl

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MCHC	26.4g/dl	29-34g/dl
Platelet	5,50,000/ cumm	1,50,000-4,00,000/cumm
Amylase	223 U/L	<100U/L
Creatinine	2.7 mg/dl	0.7-1.2mg/dl
Urea	84mg/dl	15-40mg/dl
Electrolyte	Na: 123mmol/L	Na:135-145mmol/L
	K:3.5mol/L,	K:3.5-5mmol/L
	Cl:112mmol/L	Cl: 95-107mmol/L
	Calcium: 7.8 mg/dl	Calcium: 8.5-10.5mg/dl
	Magnesium: 0.7mmol/L	Magnesium: 0.75-1.0mmol/L
SGPT	130 IU/L	Upto 40U/L
RBS	9.8 mmol/L	<7mmol/L
CRP	86 U/L	Less than 6
D Dimer	2.85 g/dL	<0.5g/dL
RAT and RT- PCR for SARS-CoV-2	Negative	
Urine RME	Pus cell 10-15/HPF	
	Epithelial cell 3-5/HPF,	
	RBC 3-5/HPF	
	Protein ++	
Urine C/S	No growth	
Blood C/S (1st)	No growth	
On admission, 12th POD of CS		
ABG	pH 7.2, $PaCO_2$ 42 mmHg, $Pa0_2$ 58 mmHg, HCO_3 13	pH: 7.35-7.45, $PaCO_2$: 34-45mmHg, PaO_2: 90-113mmHg, HCO_3: 21-29mmol/L
Albumin	2.3 g/L	3.5-5g/dl

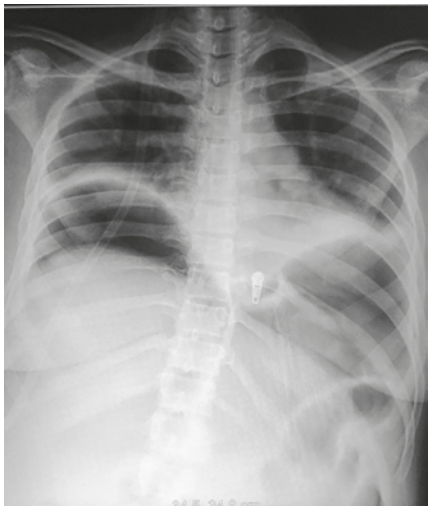

FIGURE 1: Patient's plain X-ray Abdomen showing free gas shadow beneath right dome of the diaphragm

Despite aggressive management, her condition was not improving and she developed progressive, non-reversible shock. Lateron, 3rd day following admission into ICU (15th POD of CS) culture from tracheal aspirate and 2nd blood culture and sensitivity (FAN method) was sent, and though tracheal aspirate culture came negative, prolonged blood culture revealed growth of *Burkholderia cepacia,* which was only sensitive to ceftazidime, meropenem and minocycline. Patient's Blood Culture sensitivity pattern has been attached below:

Table 2: Patient's Blood Culture sensitivity pattern

Antimicrobials	MIC	Interpretation
1. Ticarcillin/ Clavulanic acid	>128	Resistant
2. Piperacillin/ Clavulanic	>128	Resistant
3. Ceftazidime	4	Sensitive
4. Cefoperazone/ Sulbactam	16	Resistant
5. Cefepime	4	Resistant
6. Aztreonam	16	Resistant
7. Imipenem	>16	Resistant
8. Meropenem	4	Sensitive
9. Amikacin	>64	Resistant
10. Gentamicin	>16	Resistant
11. Ciprofloxacin	>4	Resistant
12. Levofloxacin	>8	Resistant
13. Minocycline	4	Sensitive
14. Tigecycline	>8	Resistant
15. Colistin	>16	Resistant
16. Trimethoprim/ Sulfamethoxazole	>320	Resistant

Ceftazidime was added on with the existing treatment and other supportive treatments were optimized.But unfortunately, she expired on the 9th day of ICU admission keeping all our efforts in vain.

Discussion:

Burkholderia cepacia is one of the species in *Burkholderia cepacia* complex. Being ubiquitous in nature, and unbelievable ability to form symbiotic relationship with amoeba and fungi has allowed it to thrive in a diverse range of environment[1,2,5,6]. It has been found to survive in diverse aqueous medicated solution such as intravenous fluid, irrigation fluid, antiseptic solutions, mouthwash,inhaler solutions, ultrasound gel and even respiratory therapy equipments.[3,4] Orthodoxly it is found in patients in cystic fibrosis and patients with chronic airway disease. It has been shown to infect hospitalized patients more commonly who are on haemodialysis, have recent abdominal surgery, underwent

bronchoscopic procedure, or having presence of central catheters.Being highly resistant to traditional multiple antimicrobials, the treatment is also very challenging and longer duration treatment is also required[2,3,4,5,6]. On the other hand,acute colonic pseudo-obstruction or, Ogilvie's syndrome is also a relatively uncommon phenomenon, characterized by colonic obstruction without an identifiable mechanical cause.Though associated with wide medical and surgical conditions, most commonly it has been reported after caesarean section[7].Though pathogenesis is unknown, and assumed to be multifactorial, butan unknown disturbance to autonomic innervation of distal colon, more precisely parasympathetic S2-S4, is being postulated[7,8]. Classically it presents between 2 to 12 days postoperatively. If not timely recognized, it can lead to colonic ischemia, perforation and life-threatening peritonitis and sepsis. Notably, 3-15% of patients develop colonic ischemia and perforation.Mortality is 15% in early appropriate treatment and 36 to 44% in perforated and ischemic colon[10,11] Our patients developed symptoms on her 6th day, initially treated conservatively, and unfortunately when clinically deteriorated was transferred to ICU. Caesarean section, spinal anaesthesia, opioidanalgesia, electrolyte imbalance may all have contributed to Ogilvie's syndrome for our patient. Though aggressive surgical intervention was done, but unfortunately it was too late. Moreover, the concomitant *Burkholderia cepacia* sepsis was the last nail in the coffin. Intravenous fluid, distilled water used in oxygen delivery devices, respiratory support devices, indwelling catheters all maybe the potential sources. Though our empirical antimicrobials were sensitive, and ceftazidime was added later on, but it was too late to respond. Early colonoscopic decompression and neostigmine perhaps would have saved the life.

Conclusion:

Ogilvie's syndrome, though rare but must be kept in differentials of acute intestinal obstruction following caesarean section. If not timely treated it can be fatal with a high mortality. *Burkholderia cepacia* sepsis is also quite uncommonin immunocompetent individuals and as resistant to polyantimicrobials, treatment is very very challenging. Proper sterilization should also be ensured to prevent *Burkholderia cepacia* outbreak in hospital settings. Our clinical acumen, could be lifesaving in these critical patients.

AUTHOR'S CONTRIBUTION:

CSP was involved in clinical diagnosis & management of the patient, literature review & manuscript writing. TK was involved in microbiological diagnosis. ZRM and BR were involved in management of the patient. NC was involved in manuscript writing and literature review.

CONFLICTS OF INTEREST:

Nothing to declare

CONSENT:

Taken from the family of the patient.

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