A rare case of urosepsis in pregnancy


Durgabai Deshmukh Hospital and Research Centre, Hyderabad

Abstract

Urinary infections are common during pregnancy, affecting 10-15% of women and the infection is recurrent in up to 10% of women. The development of a renal abscess secondary to acute pyelonephritis is uncommon during pregnancy. Acute pyelonephritis complicates 1-2% of pregnancies. Severe manifestations like sepsis occur in neglected cases. Profound hypoglycemia is typically a late phenomenon in patients with severe bacterial septicemia which is a rare complication in urosepsis. A case of urosepsis with profound hypoglycemia in pregnancy which was almost fatal for the mother but recovered with prompt diagnosis and treatment is reported here.

Keywords: urosepsis; pyelonephritis; renal abscess; hypoglycemia

Introduction

Urosepsis is sepsis with a source localised to the urinary tract. The infection most commonly affects the right kidney (90%), it is usually unilateral and associated with high morbidity, which is why, early diagnosis and treatment are necessary [1-4]. While severe sepsis has a reported mortality rate of 20 to 42%, urosepsis maybe associated with high mortality rates in special patient groups [5]. Obstetric causes are chorioamnionitis, septic abortion, and surgical site infection. Important non obstetric causes are UTI and pyelonephritis, HIV, malaria and respiratory infection.

Case report

A 25 year-old primi gravida at 35 weeks 5 days period of gestation, presented to the hospital in an unconscious state, which was preceded by headache and vomiting of 4-5 episodes per day for the past 2 days. History of fever with chills of one week duration was present. She belonged to low socioeconomic strata. She was not a known epileptic nor diabetic.
On examination she was unconscious (GCS-4, E2V1M1) and responded only to painful stimuli. She was afebrile with mild pallor and icterus, pupils were sluggishly reacting to light, pedal edema of grade 1 was present and blood pressure was 140/90mm Hg, pulse -90 /min, SpO2 -89% on room air. CNS examination showed positive babinski’s sign bilaterally. There was no focal neurological deficit. No signs of meningeal irritation. Bilateral crepitations and rhonchi were present, cardiovascular system was found to be clinically normal.

Obstetric examination showed fundal height corresponding to 34 weeks of gestation with fetus in breech position with fetal heart being normal.

It was a case of primi with a fundal height of 34 weeks in an unconscious state with mild hypertension. The following conditions were considered viz eclampsia, cerebral sinus venous thrombosis (CSVT) and cerebrovascular accident. In view of deep unconsciousness a capillary glucose level was done.

Her capillary glucose showed undetectable glucose levels. At this point the following common causes of hypoglycemia such as acute fatty liver of pregnancy, fulminant hepatic failure due to viral hepatitis, unknown diabetic on possible hypoglycemic drugs and non-islet cell tumor were considered. She was immediately kept on 25% dextrose infusion and was shifted to intensive care unit. Further investigations were ordered.

Haemogram showed hemoglobin 8gm/dl, total leucocyte count 38,000/mm3, platelet count 2.5 lakhs/mm3. Peripheral smear showed marked neutrophilic leukocytosis with shift to left with evidence of hemolysis and toxic granulation. Random blood sugar-6mg/dl, blood urea-51mg/dl, S. creatinine-2.1mg/dl, urine albumin 2+ with abundant pus cells in urine sample. Liver biochemistry showed hyperbilirubinemia (total Bilirubin 2.9, direct 1.9 and indirect 1.0), with hypoalbuminemia and A/G ratio 1:1, with enzymes being normal. Prothrombin time (PT), international normalized ratio (INR) and activated partial thromboplastin time (APTT) were normal. Thyroid stimulating hormone (TSH) was normal.

Pro calcitonin was 9.42ng/ml (N-<0.1), and CRP was positive (1:4). Because of hypoglycemia, serum cortisol level was done which was 45.8 mcg/dl(normal). C-peptide level was done 2.87 ng/ml (N- 0.48-5.05) which was normal. Blood culture and urine culture also were sent. Serum electrolytes, electrocardiography (ECG) and 2D echo were normal. Ultrasound was done, which showed single live fetus of gestational age 35 weeks in breech presentation with no liquor.

Abdominal scan showed B/L enlarged kidneys with 2.5cm space occupying lesion in left kidney, showing renal abscess with ultrasound features of bilateral pyelonephritis (Figure 1). The cause of sepsis was most probably due to pyelonephritis.

Hypoglycemia was corrected with 25 % dextrose infusion. Her capillary glucose fluctuated between 25 mg% to 100 mg%. It took 4 days for blood glucose to stabilize. Her level of consciousness and general condition gradually improved (Table 1). Initial antibiotics started were cefuroxime auxetil, metrogyl and amikacin. I.V.

On day 4, her general condition permitted to do a caesarian in view of a near term pregnancy with severe oligohydramnios and breech presentation. Caesarian section was done under general anesthesia. Surgery was uneventful and patient recovered well. A male baby of 1.6 kg weight, growth restricted, 36 weeker was delivered. Post-operatively her glucose levels were fluctuating. 25% dextrose infusion was given as and when required when she was in hypoglycemia. She was kept in hospital for one week and during this time her glucose levels were monitored 8th hourly. Urine culture and blood culture were positive for E.coli and her antibiotics were changed to clindamycin, and amikacin for a course of 10 days.
Table 1: Patient blood parameters gradually improved and urine output was adequate.

<table>
<thead>
<tr>
<th>Day</th>
<th>TLC</th>
<th>Platelets</th>
<th>Total bilirubin</th>
<th>Direct</th>
<th>indirect</th>
<th>S. alka ph</th>
<th>S. albumin</th>
<th>Creatinine</th>
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<td>1</td>
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<td>2 lac</td>
<td>mg%</td>
<td>1.0</td>
<td>1122</td>
<td>Gm%</td>
<td>mg%</td>
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</tr>
<tr>
<td>3</td>
<td>25000</td>
<td></td>
<td>2.9</td>
<td>1.9</td>
<td>0.1</td>
<td>2.6</td>
<td>2.1</td>
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</tr>
<tr>
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<td>20000</td>
<td></td>
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<td>1.6</td>
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<td>627</td>
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</tr>
<tr>
<td>7</td>
<td>18000</td>
<td>3 lac</td>
<td>0.8</td>
<td>0.3</td>
<td>4</td>
<td>1.76</td>
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</tr>
</tbody>
</table>

On 4th post-operative day CT scan abdomen was done to look for the renal abscess and to identify any other cause of hypoglycemia. CT showed features suggestive of B/L acute pyelonephritis with lobar nephronia (abscesses) largest being in right kidney (Figure 2), mild hepatomegaly with minimal right sided pleural effusion. A final diagnosis of urosepsis with hypoglycemia was made basing on the manifestations.

Repeat scan was done on 7th postoperative day which showed resolving abscesses. Her glucose levels had stabilized by 5th postoperative day. Both mother
and baby were discharged on 7th postoperative day in good condition. Follow up scan after one month showed normal sized kidneys with no abscesses.

Discussion

The urinary tract is very vulnerable to ascending infections in pregnancy. Pelvicalyceal dilatation, renal glycosuria and vesicoureteral reflux, predispose to ascending infection, apart from the immunological changes in the urothelium. Asymptomatic bacteriuria has to be treated in pregnancy. It is recommended to screen every pregnant women for asymptomatic bacteriuria. If untreated, 10 to 30% can develop acute pyelonephritis. Sepsis is an important cause of maternal mortality.

Liver is a major organ whose function can be deranged in sepsis. Liver also is the largest store house of macrophages and kupffer cell which are released into circulation to ward off the bacteremia [6]. Sepsis is associated with metabolic derangements most commonly hyperglycemia, which is managed with intensive insulin therapy.

However sepsis can also cause severe hypoglycemia. Hypoglycemia is a sign of overwhelming sepsis [7]. In patients dying with sepsis, liver steatosis and hepatitis is seen histologically [8]. Sepsis and inflammation can produce hypoglycaemia [9], although the mechanism is not well defined [10].

Impaired gluconeogenesis, increased NIMGU [non-insulin mediated glucose uptake] by macrophage rich tissues are postulated as causes of hypoglycemia. Acute inflammation mediated by lipopolysaccharide (LPS) can produce hypoglycemia. LPS suppresses fatty acid oxidation leading to elevated free fatty acid (FFA) and FFAs suppress gluconeogenic enzymes causing hypoglycaemia [6, 11]. Fischer et al, have reported that in nondoniabetics, hypoglycemic episodes were associated with sepsis [12].

In this patient neglected UTI resulted in pyelonephritis which then resulted in fulminant bacteraemic sepsis, she presented in a state of coma which was actually due to severe hypoglycaemia. As she was pregnant initially, pregnancy specific disorders such as eclampsia and stroke were thought of, subsequently when severe hypoglycaemia was detected, again pregnancy specific conditions such as acute fatty liver of pregnancy, fulminant liver failure due to hepatitis, which is quite common in our country and can be fatal in pregnancy, were thought of. However it was concluded finally that it was a case of severe urosepsis presenting as hypoglycaemic coma. Fortunately both mother and baby went home in good health, with prompt diagnosis and appropriate treatment.

Conclusion

In acutely septic patients, physicians should suspect, recognize and address derangements in glucose metabolism, especially early in the course of the illness and during active resuscitation. A methodical, combined, coordinated approach is required to diagnose and treat such cases, which results in favorable outcome.

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Conflicts interest

Authors declare no conflicts of interest.

References