Case Report

Infections; all in one

Abstract
To see and diagnose a patient with sepsis and septic shock is our daily work in the ICU. However, the case presented here showed multiple types of infections (bacterial, mycobacterial and fungal infections) each of them is a rare entity and is life threatening infection. This case demonstrates 2 rare forms of infections both in one diabetic patient with a background of leprosy. It also highlights the importance of properly and intensively treating a case of prolonged ketoacidosis (DKA).

Keywords: leprosy, emphysematous pyelonephritis, mucormycosis, diabetic ketoacidosis

Introduction
W.A.A is a 27 years old house wife presented with a diffuse dull aching abdominal pain, fever and mild dysuria. The condition was present for 2 weeks prior to her presentation at our medical ICU in Kasr AlAiny H Cairo University. She is known to have leprosy 3y ago diagnosed by skin smear as multi-bacillary lepromatous leprosy and received multidrug therapy for one year and then continued on prednisone 5mg/d. This case demonstrates 2 rare forms of infections both in one diabetic patient with a background of leprosy. It also highlights the importance of properly and intensively treating a case of prolonged ketoacidosis (DKA).

Case
W.A.A is a 27 years old house wife presented with a diffuse dull aching abdominal pain, fever and mild dysuria. The condition was present for 2 weeks prior to her presentation at our medical ICU in Kasr AlAiny H Cairo University. She is known to have leprosy 3y ago diagnosed by skin smear as multi-bacillary lepromatous leprosy and received multidrug therapy for one year and then continued on prednisone 5mg/d. She developed diabetes mellitus later (steroid induced). She claimed no family history of diabetes mellitus.

At admission; the patient was conscious, her temperature was 38.8ºC. She had normal stable blood pressure readings. Her pulse was regular at a rate of 120bpm. RR was 30/min. (acidotic breathing). ABG showed metabolic acidosis and she was diagnosed as having DKA. This explains much of her symptoms that were present for the previous 2w.1

Preliminary lab tests
a. Random blood glucose: 490mg/dL
b. ABG: PH: 7.29
c. PCO2: 13mmHg
d. PO2: 73mmHg
e. SO2: 92%
f. HCO3: 6.3mEq/L
g. Acetone in urine: ++

Skin manifestations of leprosy were present in the form of: marked loss of hair, maculo-papular rash over face, loss of outer one third of eye brow, erythematous and hypopigmented macules over the skin of the thenar and hyposthenia eminences of both hands. There were also: glove and stock hypoesthesia and thickened ulnar nerve. Treatment was started with insulin infusion, parenteral fluids and close monitoring of urine output and central venous pressure. Empirical antibiotics (cephalosporins) were given on the account of fever, mild dysuria and over 100 pus cells/mm3 in urine. 48h later; there was no improvement of her blood glucose level, pH, or urine acetone. Abdominal pain and fever continued. Moreover, she became oliguric and her creatinine started to rise2 (Table 1).

Table 1 Showing lab results

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>ESR</td>
<td>130</td>
</tr>
<tr>
<td>CRP</td>
<td>339</td>
</tr>
<tr>
<td>Urea</td>
<td>215mg/dl</td>
</tr>
<tr>
<td>Creatinine</td>
<td>4.9mg/dl</td>
</tr>
<tr>
<td>Hemoglobin</td>
<td>7g/dL</td>
</tr>
<tr>
<td>TLC</td>
<td>12.5</td>
</tr>
<tr>
<td>Platelets</td>
<td>44</td>
</tr>
<tr>
<td>Bilirubin</td>
<td>2.97mg/dL</td>
</tr>
<tr>
<td>AST</td>
<td>80 IU/I (N up to 38)</td>
</tr>
<tr>
<td>ALT</td>
<td>60 IU/I (N up to 40)</td>
</tr>
<tr>
<td>Albumin</td>
<td>2.2g/dL</td>
</tr>
<tr>
<td>Total protein</td>
<td>5.5g/dL</td>
</tr>
</tbody>
</table>

Pan cultures were taken. Dermatology consultation was requested to exclude a leprotic reaction as the cause of fever. Type two leprotic reaction was diagnosed and it was recommended to give the patient: 40mg prednisone, rifampicin and dapson.2,3 Again; there was no improvement of her abdominal pain, fever, blood sugar or urine acetone. At that time, the patient became hypotensive, serum creatinine escalated, as well as AST, ALT and bilirubin. The diagnosis of septic shock was assumed. Meanwhile and due to the persistent state of DKA and fever; we started to think of a hidden maybe life threatening infections. Detailed examination revealed blackish oral lesions over


© 2015 Sholkamy et al. This is an open access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and build upon your work non-commercially.

Amany Abdel Maqsod Sholkamy, Ragaay Felkry, Ayda Wasfy
Department of Internal Medicine, Cairo University, Egypt

Correspondence: Amany Abdel Maqsod Sholkamy, Department of Internal Medicine, Faculty of medicine, Cairo University, kasr alainy, Egypt, Tel 02 01223567784, Email amanymaqsod@kasralainy.edu.eg

Received: November 01, 2015 | Published: November 23, 2015

Abstract
To see and diagnose a patient with sepsis and septic shock is our daily work in the ICU. However, the case presented here showed multiple types of infections (bacterial, mycobacterial and fungal infections) each of them is a rare entity and is life threatening infection. This case demonstrates 2 rare forms of infections both in one diabetic patient with a background of leprosy. It also highlights the importance of properly and intensively treating a case of prolonged ketoacidosis (DKA).

Keywords: leprosy, emphysematous pyelonephritis, mucormycosis, diabetic ketoacidosis

Introduction
W.A.A is a 27 years old house wife presented with a diffuse dull aching abdominal pain, fever and mild dysuria. The condition was present for 2 weeks prior to her presentation at our medical ICU in Kasr AlAiny H Cairo University. She is known to have leprosy 3y ago diagnosed by skin smear as multi-bacillary lepromatous leprosy and received multidrug therapy for one year and then continued on prednisone 5mg/d. This case demonstrates 2 rare forms of infections both in one diabetic patient with a background of leprosy. It also highlights the importance of properly and intensively treating a case of prolonged ketoacidosis (DKA).

Case
W.A.A is a 27 years old house wife presented with a diffuse dull aching abdominal pain, fever and mild dysuria. The condition was present for 2 weeks prior to her presentation at our medical ICU in Kasr AlAiny H Cairo University. She is known to have leprosy 3y ago diagnosed by skin smear as multi-bacillary lepromatous leprosy and received multidrug therapy for one year and then continued on prednisone 5mg/d. This case demonstrates 2 rare forms of infections both in one diabetic patient with a background of leprosy. It also highlights the importance of properly and intensively treating a case of prolonged ketoacidosis (DKA).

Preliminary lab tests
a. Random blood glucose: 490mg/dL
b. ABG: PH: 7.29
c. PCO2: 13mmHg
d. PO2: 73mmHg
e. SO2: 92%
f. HCO3: 6.3mEq/L
g. Acetone in urine: ++

Skin manifestations of leprosy were present in the form of: marked loss of hair, maculo-papular rash over face, loss of outer one third of eye brow, erythematous and hypopigmented macules over the skin of the thenar and hyposthenia eminences of both hands. There were also: glove and stock hypoesthesia and thickened ulnar nerve. Treatment was started with insulin infusion, parenteral fluids and close monitoring of urine output and central venous pressure. Empirical antibiotics (cephalosporins) were given on the account of fever, mild dysuria and over 100 pus cells/mm3 in urine. 48h later; there was no improvement of her blood glucose level, pH, or urine acetone. Abdominal pain and fever continued. Moreover, she became oliguric and her creatinine started to rise2 (Table 1).

Table 1 Showing lab results

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>ESR</td>
<td>130</td>
</tr>
<tr>
<td>CRP</td>
<td>339</td>
</tr>
<tr>
<td>Urea</td>
<td>215mg/dl</td>
</tr>
<tr>
<td>Creatinine</td>
<td>4.9mg/dl</td>
</tr>
<tr>
<td>Hemoglobin</td>
<td>7g/dL</td>
</tr>
<tr>
<td>TLC</td>
<td>12.5</td>
</tr>
<tr>
<td>Platelets</td>
<td>44</td>
</tr>
<tr>
<td>Bilirubin</td>
<td>2.97mg/dL</td>
</tr>
<tr>
<td>AST</td>
<td>80 IU/I (N up to 38)</td>
</tr>
<tr>
<td>ALT</td>
<td>60 IU/I (N up to 40)</td>
</tr>
<tr>
<td>Albumin</td>
<td>2.2g/dL</td>
</tr>
<tr>
<td>Total protein</td>
<td>5.5g/dL</td>
</tr>
</tbody>
</table>

Pan cultures were taken. Dermatology consultation was requested to exclude a leprotic reaction as the cause of fever. Type two leprotic reaction was diagnosed and it was recommended to give the patient: 40mg prednisone, rifampicin and dapson.2,3 Again; there was no improvement of her abdominal pain, fever, blood sugar or urine acetone. At that time, the patient became hypotensive, serum creatinine escalated, as well as AST, ALT and bilirubin. The diagnosis of septic shock was assumed. Meanwhile and due to the persistent state of DKA and fever; we started to think of a hidden maybe life threatening infections. Detailed examination revealed blackish oral lesions over
Infections; all in one

Fever and high blood sugar and/or DKA non-responsive to treatment should raise the suspicion of severe life threatening infections in diabetics patients.

Life threatening infections:

i. Emphysematous pyelonephritis
ii. Emphysematous cholecystitis
iii. Necrotizing fasciitis
iv. Mucormycosis
v. Malignant otitis externa

Abdominal US revealed bilateral emphysematous pyelonephritis. The condition was confirmed by CT of the abdomen. So, this diabetic leprotic young patient had 2 types of infections each by itself is considered rare life threatening conditions; emphysematous pyelonephritis (type II involving the parenchyma but does not extend beyond the capsule) and mucormycosis involving the hard palate. As the diagnosis was made we were faced with deteriorating renal and liver functions and picture of septic shock and MOFs. The condition was complicated more and more. She became progressively dyspnic. Echocardiography revealed severe pericardial effusion. Pericardiocentesis was done; 800cc of hemorrhagic effusion was aspirated and was sent for culture and lab assessment.

Hemorrhagic pericardial effusion is commonly due to tuberculosis, or malignancy. Lab results of the aspirate revealed: negative ADA and PCR for TB. No malignant cells on cytological examination. Culture and sensitivity showed no growth. The condition was attributed to hypoalbuminemia and thrombocytopenia (platelet count was 44,000) causing hemorrhagic pericardial effusion. Thrombocytopenia is a common finding in cases of pyelonephritis, the etiology of which is not exactly identified.

Problem list:

i. Uncontrolled diabetes mellitus
ii. Leprosy
iii. Emphysematous pyelonephritis
iv. Mucormycosis
v. Hemorrhagic Pericardial effusion
vi. Septic shock

Steroids were stopped. IV fluids, vasopressors and hemodialysis were started. IV broad spectrum antibiotics, tight control of diabetes and IV fungizone were given. The renal functions started to improve together with her general condition. No surgical or percutaneous intervention was needed. Two weeks later she was discharged home after clinical improvement for follow up. This case demonstrates a constellation of several types of infections all in one patient; bacterial, mycobacterial and fungal infections. Two of them are considered rare infections and each is a life threatening infection. The patient started to deteriorate, septic shock and MOF eventually ensued and we were about close to losing this young house wife. However, intensive care monitoring, proper antimicrobial and the multidisciplinary care were all sharing to save her life.

Conclusion

It is to be mentioned that these types of infections especially emphysematous pyelonephritis is not known to occur in this age group. Emphysematous UTI are usually seen (if any) in older age group.

Because vascular insufficiency remains a very important risk factor in diabetic patients (beside hyperglycemia). This patient is 27y old, she had diabetes mellitus following several courses of steroids for the treatment of leprous reactions and she had no family history of diabetes mellitus. So, it is assumed that she has iatrogenic hyperglycemia/diabetes mellitus. Such type of diabetes in this age group and for this short duration (2 years) –although not exclusively- will not lead to autonomic neuropathy (one of the risk factors for susceptibility for this type of infection).

Acknowledgements

None.

Conflict of interest

The author declares no conflict of interest.

References