were deliberately contaminated with *S. aureus* to determine the extent of survival of bacteria. *S. aureus* survived up to 48 hours on rubber grip pens, whereas the minimum duration of survival was observed on plastic pens and pens with metal body (24 hours and 18 hours, respectively). Our findings indicate that the pens used by healthcare personnel in intensive care units can be contaminated with bacteria. This is in agreement with the findings of a previous study. [5] However, another group of workers could not show bacterial contamination of pens. [6] Several factors such as duration of usage, type of pen, number of persons using the pen may influence the rate of contamination of pens. We showed that *S. aureus* survives longer on rubber grips of the pens. Isolation of methicillin resistant *S. aureus* is a matter of concern. One critical aspect of bacterial transmission from person or from environment to a person is the ability of the microbe to survive on environmental surface. Careful use of pens and handwashing will help prevent transmission of bacteria from contaminated pens. Usage of pens with metal body may be encouraged in hospitals.

**Acknowledgments**

The authors thank the Dean, Chief Operating Officer, and all healthcare personnel who provided the used pens for the study.

**References**


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**Table 1: Bacteria isolated from used pens**

<table>
<thead>
<tr>
<th>Bacteria isolated</th>
<th>No. of pens</th>
<th>Rate of contamination (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Staphylococcus epidermidis</em></td>
<td>16</td>
<td>21.3</td>
</tr>
<tr>
<td><em>Staphylococcus aureus</em></td>
<td>6</td>
<td>7.9</td>
</tr>
<tr>
<td>Bacillus spp</td>
<td>4</td>
<td>5.3</td>
</tr>
<tr>
<td>Diphtheroids</td>
<td>2</td>
<td>2.7</td>
</tr>
<tr>
<td>Micrococcus spp.</td>
<td>1</td>
<td>1.3</td>
</tr>
</tbody>
</table>

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**Visceral Leishmaniasis Simulating Chronic Liver Disease: Successful Treatment with Miltefosine**

**Dear Editor,**

A 45-year-old non-alcoholic male resident of Bihar, an Indian state endemic for Kala-azar, was admitted with high-grade fever associated with chills and rigor, pain in the left upper abdomen, anorexia and weight loss for 1½ months. He had severe pallor, bilateral pedal oedema, non-tender hepatosplenomegaly and ascites. Investigations revealed haemoglobin of 5.5 g/dL, total leucocyte count 1.4x10³/mm³, platelet count 60x10³/mm³, ESR 78 mm/h and peripheral smear suggestive of pancytopenia. Bone marrow smear showed plasma cells and Leishman donovan (LD) bodies. Indirect immunofluorescent antibody test (IFAT) for Kala-azar was positive (titre 1:1600). Serum bilirubin was 1.2 mg/dL, serum alanine aminotransferase 20 U/L, serum aspartate aminotransferase 16 U/L, serum alkaline phosphatase 240 U/L and total serum protein 10.7 g/dL (albumin 1.44 g/dL). Prothrombin time (PT) was 25 (13 s) and activated partial thromboplastin time (aPTT) was 50 (28 s). Protein electrophoresis was suggestive of chronic inflammation and hypoalbuminaemia without any M-spike. Urine examination revealed albumin ++++, Bence–Jones protein negative and 24-h urinary protein was 1312 mg. Hepatitis B surface antigen, antihepatitis C virus and enzyme-linked immunosorbent assay for human immunodeficiency virus were negative. Ultrasound of the abdomen showed hepatomegaly (14 cm), massive splenomegaly (> 19 cm), borderline portal hypertension (portal vein size 13 mm) and ascites. Ascitic fluid was transudative in nature. Upper gastrointestinal endoscopy was normal. Liver and renal biopsies could not be performed as patient refused to consent. The patient was put on tablet Miltefosine 50 mg
BD for 4 weeks. He responded well to treatment and became afebrile after 10 days, with marked regression in the size of the liver and spleen. Repeat investigations performed after 8 weeks showed near normal haemogram, normal liver function tests and normal size portal vein on ultrasound and the bone marrow smear became negative for LD bodies. IFAT titre (1:400) and proteinuria (476 mg/24 h) decreased significantly.

Our patient had evidence suggestive of significant liver disease in view of hepatosplenomegaly, elevated serum alkaline phosphatase, marked hypoalbuminaemia, impaired PT/aPTT, transudative ascites and ultrasonographic evidence of hepatosplenomegaly, ascites and borderline portal hypertension along with definitive evidence of Kala-azar. Most of these findings did revert after successful treatment with oral miltefosine.

Although liver involvement is not unusual in Kala-azar, presentation as cirrhosis, portal hypertension and chronic liver disease is always a curiosity. Extensive literature search revealed only one case report with visceral leishmaniasis along with histological evidence of leishmanial hepatitis and portal hypertension. On the contrary, a series of 60 Kala-azar cases did not have any case of chronic liver disease, although one quarter of the patients had biochemical evidence of hepatitis. Our patient also had non-nephrotic range proteinuria at the time of diagnosis, which is a common finding usually reported as a late complication.

The Indian subcontinent has been plagued by resistance to classical antileishmanial drugs and the approval of miltefosine in this regard has been a significant milestone in the treatment due to ease of administration, few toxic effects and excellent cure rate.

References

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Received: 10-03-2008
Accepted: 11-05-2008