radiation therapy to the mediastinum (36 Gy). The patient achieved CR in February 1995. Over the next 14 months she remained well and in complete hematologic remission. In May 1996 she noted extensive ecchymoses and petechiae. Her hematocrit was 43.2%, leukocyte count 0.64x10^9/L and the platelet count 1.0x10^9/L. The spleen was not palpable and there was no evidence of HD relapse. A bone marrow biopsy revealed normal erythroid and myeloid maturation. The diagnosis of ITP was suspected and a trial of corticosteroids was begun. After 30 days the platelet count was normal (224x10^9/L) and corticosteroids were stopped. To date, the patient remains in complete hematologic (ITP) and HD remission. Although some studies have reported that the presence of ITP in HD patients could signify active lymphoma, especially with splenic involvement, other reports have shown that ITP can occur in the absence of active HD, and that the HD status is independent of the onset of ITP.

Our two patients did not have active HD when they developed ITP, and the development of ITP did not indicate subsequent relapse, supporting the hypothesis of two independent diseases with different onsets. Furthermore, as for primary ITP occurring in association with HD, so thrombocytopenic purpura also seems to respond to conventional therapy. Both patients responded with a single course of oral prednisolone, but one relapsed after 15 months and splenectomy was necessary for CR.

The recognition of a picture of ITP as a complication of HD could have important implications, as described by Doan and Bouroncle. We suggest, therefore, that an occult HD should be considered in any patient who presents with thrombocytopenic purpura of the ITP type, and particularly in a patient known to have been previously treated for HD, where the ITP picture may signal the recurrence of lymphoma. It should be noted that we have also observed thrombocytopenic purpura in chronic myeloid leukemia (CML) during treatment with interferon-α.

In conclusion, ITP associated with HD responds to therapy in a similar manner to that of primary ITP. Its presence seems to confer no prognostic significance, although the possibility of localization of splenic HD disease has to be considered.

Correspondence
Giovanni Martinelli, MD, Institute of Hematology and Medical Oncology "Seràgnoli", University of Bologna, Policlinico S. Orsola, via Massarenti 9, 40138, Bologna, Italy. Phone: international +39-051-6363680 • Fax: international +39-051-398973 • E-mail: seragnol@kaiser.alma.unibo.it

References

Bacteremia caused by CDC Group IV c-2 in a patient with acute leukemia
ANTONIO SALAR, JORDI CARRATALÀ, AMADEO ZURITA, EVA GONZÁLEZ-BARCA, ALBERTO GRÀNENA

Department of Clinical Hematology, Institut Català d’Oncologia and *Department of Infectious Diseases, Hospital Príncipes de España, Ciudad Sanitaria y Universitaria de Bellvitge, Hospital de Llobregat, Barcelona, Spain

Human infections due to CDC group IV c-2, a gram-negative bacillus, are rare. We describe a case of nosocomial bacteremia caused by this organism in a neutropenic patient with acute lymphoblastic leukemia and include a literature review of CDC group IV c-2 infection in patients with hematologic malignancies.

Human infections due to CDC group IV c-2, a gram-negative bacillus, have been recently reported.1,8 Bacteremia caused by this organism is rare in patients with hematologic malignancies.2,5,6,8

We report here a case of septicemia caused by CDC group IV c-2 in a neutropenic patient with acute leukemia and review the literature on this entity (MEDLINE, National Library of Medicine, Bethesda, MD covering the years 1965-97).

A 71-year-old previously healthy woman was admitted to our hospital with a temperature of 37.4ºC, dyspnea and pleuritic chest pain. The patient had a history of fatigue, weakness and weight loss of 10 kg over the three previous months. Physical examination revealed cutaneous pallor, shortness of
breath and a few crackles at the left lung base. White blood cell count was 188 × 10^9/L with 95% blastic forms, the hemoglobin level was 6.8 g/dL and platelet count 44 × 10^9/L. A chest radiograph showed a left lower lobe infiltrate and a small left pleural effusion. Empirical therapy with amoxicillin/clavulanate was administered intravenously. On the second day of admission the patient was afebrile. Sputum and blood cultures were negative.

Microscopical examination of a bone marrow aspirate revealed acute lymphoblastic leukemia, L-2 according to the criteria of the FAB classification. Immunophenotype was consistent with ALL-preB. An induction chemotherapy regimen consisting of daunorubicin, vincristine, L-asparaginase and prednisone was initiated.

After two weeks of antibiotic treatment, the patient’s condition improved and a new chest radiograph was normal. Amoxicillin/clavulanate was stopped and oral prophylaxis with norfloxacin was then initiated. Twelve days later, while neutropenic, the patient’s temperature rose to 38.5°C and her blood pressure fell to 80/50 mm Hg. After acquisition of two specimens for blood culturing, fluid replacement was initiated and, ceftazidime and amikacin were given intravenously. Twenty-four hours after beginning empirical antibiotic therapy she became afebrile with no clinical signs of infection. A gram-negative rod was isolated from all the blood cultures. Cultures from urine and indwelling line specimens were negative.

The chemotherapy course was complicated by several episodes of intestinal bleeding due to severe thrombocytopenia. Several days later, the patient became agitated and severe hypotension and oliguria developed despite fluid replacement and inotropic drug therapy. The patient died because of a new bleeding episode. At that time blood cultures were negative.

The isolate was identified as CDC group IV c-2 by conventional methods from the blood cultures after 72 hours. Its microbiological characteristics were consistent with those previously defined for the bacillus. With standard disk diffusion testing, the organism was susceptible to trimethoprim-sulfamethoxazole, ciprofloxacin and ceftazidime, but was resistant to ampicillin, amoxicillin plus clavulanate, piperacillin, ticarcillin, ticarcillin plus clavulanate, cephalothin, cefoxitin, cefotaxime, ceftriaxone, imipenem, aztreonam, gentamicin, tobramycin, amikacin and tetracycline.

CDC Group IV c-2 is a gram-negative environmental bacillus rarely isolated from clinical specimens. However, several cases of well-documented infections caused by this organism have been reported, with eight cases of septicemia (five of them occurring in a hospital outbreak), two cases of peritonitis related to peritoneal catheter infection, one case of planar abscess complicated with bacteremia and one case of tenosynovitis. All but one of these patients had underlying disease. We found five previously reported cases of infection caused by CDC Group IV c-2 in patients with hematologic malignancies in our review of the literature. These 5 cases with the addition of our case are summarized in Table 1. Four patients had acute leukemia and two non-Hodgkin’s lymphoma. Our review shows that most of the infections were nosocomial-acquired and presented with bacteremia, mainly catheter-related, occurring during chemotherapy-induced neutropenia.

In spite of the well-known severity of gram-negative bacteremia in patients with cancer, all reported patients with bacteremia caused by CDC Group IV c-2 were cured from infection following appropriate antimicrobial therapy, probably indicating a low level of pathogenicity of this organism. It should be noted that catheter removal was only necessary in one of these cases.

It has been shown that CDC Group IV c-2 is susceptible to a wide range of antimicrobial agents, including aminopenicillins, antipseudomonal penicillins, cephalosporins, carbapenems and fluoroquinolones. However, this organism is usually resistant to aminoglycosides. In our case, the organism showed an unusual pattern of antibiotic-susceptibility, showing resistance to a broad spectrum of antimicrobial agents. To our knowledge, this is the first report of infection caused by CDC Group IV c-2 in a patient with acute lymphoblastic leukemia. The isolate was successfully treated with a combination of ceftazidime and amikacin.

Table 1. CDC Group IV c-2 infection in patients with hematologic malignancies.

<table>
<thead>
<tr>
<th>Reference</th>
<th>Age/Sex</th>
<th>Underlying condition</th>
<th>Neutropenia</th>
<th>Specimen cultured</th>
<th>Source of infection</th>
<th>Acquisition</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dan et al.</td>
<td>37/M</td>
<td>Plasma cell leukemia</td>
<td>No</td>
<td>Blood</td>
<td>Unknown</td>
<td>Community</td>
<td>Recovery</td>
</tr>
<tr>
<td>Ramos et al.</td>
<td>10/F</td>
<td>Acute leukemia</td>
<td>Yes</td>
<td>Blood</td>
<td>Catheter</td>
<td>Nosocomial</td>
<td>Recovery</td>
</tr>
<tr>
<td>Moissenet et al.</td>
<td>11/NR</td>
<td>Non Hodgkin’s lymphoma*</td>
<td>Yes</td>
<td>Blood</td>
<td>Catheter</td>
<td>Nosocomial</td>
<td>Recovery</td>
</tr>
<tr>
<td>Moissenet et al.</td>
<td>14/NR</td>
<td>Acute leukemia*</td>
<td>Yes</td>
<td>Blood</td>
<td>Catheter</td>
<td>Nosocomial</td>
<td>Recovery</td>
</tr>
<tr>
<td>Salar et al.</td>
<td>77/F</td>
<td>Acute leukemia</td>
<td>Yes</td>
<td>Blood</td>
<td>Unknown</td>
<td>Nosocomial</td>
<td>Recovery</td>
</tr>
</tbody>
</table>

PR, present report; NR, not reported; *cases reported in a hospital outbreak of CDC Group IV c-2 infection.
first report in which resistance of this bacillus to imipenem is noted.

In summary, CDC group IV c-2 bacillus is an opportunistic pathogen that rarely causes bacteremia in patients with hematologic malignancies and indwelling intravenous lines.

**Key words**

CDC group IV c-2, bacteremia, acute leukemia, neutropenia.

**Funding**

This work was supported by a grant (AG/97) from the José Carreras International Leukemia Foundation.

**Correspondence**

Antonio Salar, MD, Department of Clinical Hematology, Institut Català d’Oncologia, Autovía de Castelldefels km 2.7, 08907 Hospitalet de Llobregat, Barcelona, Spain. Fax international + 34-3-263-4579 • E-mail: 28609ass@comb.es

**References**