Infection due to Nocardia farcinica in a woman with chronic granulomatous disease
Fijen, C.A.P.M.J.; Schrama, J.G.; Kuijper, E.J.; Boiron, P.; Gerritsen, W.R.; Speelman, P.

Published in:
Clinical infectious diseases

DOI:
10.1086/517033

Citation for published version (APA):
Infection Due to Nocardia farcinica in a Woman with Chronic Granulomatous Disease

Nocardiosis, an infecion due o an aerobic ac inomyce e, has a pronounced endency oward remission and exacerba ion. The Nocardia asteroides complex comprises three species: N. aster-

Purulen ma erial ob ained by CT-guided aspira ion was found o be smear posi ive af er bo h Ziehl-Neelsen and auramine s aining. PCR de ce ed DNA from non uberculous mycobac era. Cult-ure by use of he BACTEC sys em (Bec on Dickinson, Cockeys-ville, MD) revealed grow h of . xenopi, firs iden ified by specific PCR and secondarilly by biochemical es ing and gas chroma ogra-

An imycobac era erial herapy wi h ion s, rifampin, e hambo ol, and pyrazinamide was ini ed. In vi ro suscep ibile o es ing ha was performed on solid media by use of he propor ion echnique of Can e showed resis ance o ion s, e hambo ol, pyrazinam-

Ofloxacin was added o he pa ien ’s herapeu ic regimen when . xenopi was iden ified. Two mon hs la er, because of he en-

Medical rea men was con inued for 18 mon hs, wi h clinical improven men and biological normaliza ion. CT performed 17 mon hs af er comple ion of herapy revealed a significant persis en locula ed fluid collec ion, despi e he pa ien ’s is sa isfac ory clinical s a e.

Primary psoas muscle abscess is an uncommon infec ion and caused mos of en by infec ion due o Staphylococcus aureus. To our knowledge, his is he firs des cription of an case of primary psoas muscle abscess caused by . xenopi [2].

The por al of en ry of he bacillus remains an enigma; rea men; ed s udives (radiographic and scan graphic) failed o demons ra e a pulmonary, bone, or o her source for he psoas muscle infec ion. PCR me hods were very useful in ha hey allowed early de ec-

Nocardia asteroides complex comprises three species: N. aster-

References
A 56-year-old woman with CGD and a 1-month history of fever and a cough productive of greenish sputum failed to respond to therapy with erythromycin. She was admitted to the hospital. Her medical history included recurrent inguinal lymphadenitis, pulmonary sarcoidosis, and coliis. In 1988, she was read for breast carcinoma. In 1994, masiis due to infection with Aspergillus fumigatus was diagnosed, and she was read with amphotericin B; at home, therapy was changed to with itraconazole because her renal function had deteriorated. CGD due to p47phox deficiency was diagnosed. Treatment for CGD with interferon and prophylaxis for infection with co-rimoxazole were discontinued because of side effects.

On admission to the hospital, the patient’s temperature was 39°C. Auscultation over the upper lobe of the right lung revealed the presence of fine crackles. The erythrocyte sedimentation rate (71 mm/h) and the WBC count (14.4 × 10^9/L with left shift) were elevated, and anemia was present (hemoglobin level, 42.5 g/dL). A chest radiograph and a chest CT scan revealed a cavernous lesion, 7 cm in diameter, in the right upper lobe of the lung (figure 1). Bronchoscopic examination revealed no abnormalities, but Gram’s staining of the bronchoalveolar lavage fluid and biopsy specimens showed gram-positive, filamentous and branched bacteria, which were fluorescent with UV excitation (R&R, Kandern, Germany). With an assumed diagnosis of nocardiosis and consideration of the amphotericin B–induced renal damage, iv cefoxime (1 g q.i.d.) and imipenem (500 mg q.i.d.) were administered. The fever resolved, and, within 6 months, the pulmonary cavernous lesion resolved.

Aerobic incubation of the bronchoscopic specimens at 37°C for 4 days yielded folded, heaped, slightly orange colonies producing an aerial mycelium. The microorganism was partially acid-fast, was resistant to lysozyme, and produced urease and acid from glucose and rhamnose. N. farcinica was identified on the basis of negative decomposition of adenine, casein, xanthine, hypoxanthine, and yrosine; equal growth at 35°C and 45°C; opacification of Middlebrook agar; results of qualitative evaluation of cell-wall and carbon sources, and specific enzymatic activities; however, the young may be due to the residual killing activity present in the granulocytes. Consistent with this hypothesis, a recent report describes residual superoxide production of the granulocytes in seven of 11 adult CGD patients for whom the data of infecious complications was low during you h[10].

Sulfonamides are still the treatment of choice for infections due to N. farcinica[2, 3]. Good therapy is reflected by resolution of the CNS and bone involvement[2, 3, 6]. In our patient, the primary lesion was located in the right lung. Lack of dissemination in the head and low number of infective organisms in the you h may be due to the residual killing activity present in the granulocytes. Consistent with this hypothesis, a recent report describes residual superoxide production of the granulocytes in seven of 11 adult CGD patients for whom the data of infective complications was low during you h[10].

Sulfonamides are still the treatment of choice for infections due to N. farcinica[2, 3]. Good therapy is reflected by resolution of the CNS and bone involvement[2, 3, 6]. In our patient, the primary lesion was located in the right lung. Lack of dissemination in the head and low number of infective organisms in the you h may be due to the residual killing activity present in the granulocytes. Consistent with this hypothesis, a recent report describes residual superoxide production of the granulocytes in seven of 11 adult CGD patients for whom the data of infective complications was low during you h[10].

Sulfonamides are still the treatment of choice for infections due to N. farcinica[2, 3]. Good therapy is reflected by resolution of the CNS and bone involvement[2, 3, 6]. In our patient, the primary lesion was located in the right lung. Lack of dissemination in the head and low number of infective organisms in the you h may be due to the residual killing activity present in the granulocytes. Consistent with this hypothesis, a recent report describes residual superoxide production of the granulocytes in seven of 11 adult CGD patients for whom the data of infective complications was low during you h[10]. Sulfonamides are still the treatment of choice for infections due to N. farcinica[2, 3]. Good therapy is reflected by resolution of the CNS and bone involvement[2, 3, 6]. In our patient, the primary lesion was located in the right lung. Lack of dissemination in the head and low number of infective organisms in the you h may be due to the residual killing activity present in the granulocytes. Consistent with this hypothesis, a recent report describes residual superoxide production of the granulocytes in seven of 11 adult CGD patients for whom the data of infective complications was low during you h[10]. Sulfonamides are still the treatment of choice for infections due to N. farcinica[2, 3]. Good therapy is reflected by resolution of the CNS and bone involvement[2, 3, 6]. In our patient, the primary lesion was located in the right lung. Lack of dissemination in the head and low number of infective organisms in the you h may be due to the residual killing activity present in the granulocytes. Consistent with this hypothesis, a recent report describes residual superoxide production of the granulocytes in seven of 11 adult CGD patients for whom the data of infective complications was low during you h[10]. Sulfonamides are still the treatment of choice for infections due to N. farcinica[2, 3]. Good therapy is reflected by resolution of the CNS and bone involvement[2, 3, 6]. In our patient, the primary lesion was located in the right lung. Lack of dissemination in the head and low number of infective organisms in the you h may be due to the residual killing activity present in the granulocytes. Consistent with this hypothesis, a recent report describes residual superoxide production of the granulocytes in seven of 11 adult CGD patients for whom the data of infective complications was low during you h[10]. Sulfonamides are still the treatment of choice for infections due to N. farcinica[2, 3]. Good therapy is reflected by resolution of the CNS and bone involvement[2, 3, 6]. In our patient, the primary lesion was located in the right lung. Lack of dissemination in the head and low number of infective organisms in the you h may be due to the residual killing activity present in the granulocytes. Consistent with this hypothesis, a recent report describes residual superoxide production of the granulocytes in seven of 11 adult CGD patients for whom the data of infective complications was low during you h[10]. Sulfonamides are still the treatment of choice for infections due to N. farcinica[2, 3]. Good therapy is reflected by resolution of the CNS and bone involvement[2, 3, 6]. In our patient, the primary lesion was located in the right lung. Lack of dissemination in the head and low number of infective organisms in the you h may be due to the residual killing activity present in the granulocytes. Consistent with this hypothesis, a recent report describes residual superoxide production of the granulocytes in seven of 11 adult CGD patients for whom the data of infective complications was low during you h[10]. Sulfonamides are still the treatment of choice for infections due to N. farcinica[2, 3]. Good therapy is reflected by resolution of the CNS and bone involvement[2, 3, 6]. In our patient, the primary lesion was located in the right lung. Lack of dissemination in the head and low number of infective organisms in the you h may be due to the residual killing activity present in the granulocytes. Consistent with this hypothesis, a recent report describes residual superoxide production of the granulocytes in seven of 11 adult CGD patients for whom the data of infective complications was low during you h[10]. Sulfonamides are still the treatment of choice for infections due to N. farcinica[2, 3]. Good therapy is reflected by resolution of the CNS and bone involvement[2, 3, 6]. In our patient, the primary lesion was located in the right lung. Lack of dissemination in the head and low number of infective organisms in the you h may be due to the residual killing activity present in the granulocytes. Consistent with this hypothesis, a recent report describes residual superoxide production of the granulocytes in seven of 11 adult CGD patients for whom the data of infective complications was low during you h[10]. Sulfonamides are still the treatment of choice for infections due to N. farcinica[2, 3]. Good therapy is reflected by resolution of the CNS and bone involvement[2, 3, 6]. In our patient, the primary lesion was located in the right lung. Lack of dissemination in the head and low number of infective organisms in the you h may be due to the residual killing activity present in the granulocytes. Consistent with this hypothesis, a recent report describes residual superoxide production of the granulocytes in seven of 11 adult CGD patients for whom the data of infective complications was low during you h[10]. Sulfonamides are still the treatment of choice for infections due to N. farcinica[2, 3]. Good therapy is reflected by resolution of the CNS and bone involvement[2, 3, 6]. In our patient, the primary lesion was located in the right lung. Lack of dissemination in the head and low number of infective organisms in the you h may be due to the residual killing activity present in the granulocytes. Consistent with this hypothesis, a recent report describes residual superoxide production of the granulocytes in seven of 11 adult CGD patients for whom the data of infective complications was low during you h[10].
Neisseria case-conrol studies comparing the efficacy of various regimens.

C. A. P. Fijen, J. Schrama, E. J. Kuiper, P. Boiron, W. Gerritsen, and P. Speelman
Department of Medical Microbiology and Department of Internal Medicine/Infectious Diseases, Academic Medical Centre, University of Amsterdam, and Department of Hematology/Oncology, Antoni van Leeuwenhoek Hospital, Amsterdam, the Netherlands; and Unité de Virologie, National Center for Ycooses and Antifungal Agents, Institut Pasteur, Paris, France

References
2. Peers BR, Sanbolle MA, Cosandino JM. Disseminated and cerebral infection due to Nocardia farcinica: diagnosis by blood culture and cure.

Neurosyphilis and Syphilitic Gumma of the Adrenal Gland

Syphilitic gummas have been described in different body organs, but syphilis of the adrenal gland is rare; only two cases were reported in the first half of the century [1]. As far as we know, ours is the first case of syphilitic gumma of the adrenal gland to be reported in recent years.

A 66-year-old male was admitted to hospital in 1996 with a 2-week history of abdominal pain and consipaion and diminished vision in the right eye; he had had a hard, polydipsia, and gain of several kilograms.

On admission he was afebrile and had angular cheilosis and a small keraoid lesion on the right cheek; he had gained weight over the past year. He was anemic and his hemoglobin was 11.5 g/dL, and his white blood cell count was 6.8 x 10^9/L, with a normal differential. A chest X-ray showed no abnormalities.

Laboratory studies revealed the following values: sodium, 137 mEq/L; potassium, 5.2 mEq/L; chloride, 102 mEq/L; hyroxine, 0.82 µg/dL; and thyroid-stimulating hormone, 33.86 µg/dL. A serum creatinine was 0.82 mg/dL.

A CT scan of the abdomen showed a 3-cm mass in the adrenal gland, with a central hypodense area and multiple hemorrhages. Microscopic examination of the mass revealed focal areas of necrosis with inflammatory changes.

Therapy with hyroxyine was started. Six days later, he


This work was presented in a paper at the Annual Scientific Meeting of the American College of Physicians held on 10–11 January 1997 in Long Branch, New Jersey.

Reprints or correspondence: Dr. Gary Gar enberg, Highland Park Medical Associæes, 205 Nor h Second Avenue, Highland Park, New Jersey 08904.

Clinical Infectious Diseases 1998;26:224–5 © 1998 by The Universi y of Chicago. All rights reserved.
1058-4838/98/2601-0058$03.00

pa ien suddenly became somnolent. His vi al signs were: emperatura, 102.4°F; pulse, 85/min and regular; and blood pressure, 84/44 mm Hg. He was resuscitated with hydroxy and other medications. The cor isol level was 18 µg/dL [2, 3], and he resulted from a cosyn ropin es were normal.

Additionnal work-up revealed a rapid plasma reagin (RPR) and a complement fixation test (CFT) for syphilis were normal. A serum IgG and IgM antibody for Borrelia burgdorferi was positive, but PCR assays of blood and CSF were negative. A CSF examination revealed a normal cell count and glucose concentration.

Laboratory studies revealed the following values: sodium, 137 mEq/L; potassium, 5.2 mEq/L; chloride, 102 mEq/L; hyroxine, 0.82 µg/dL; and thyroid-stimulating hormone, 33.86 µg/dL. A serum creatinine was 0.82 mg/dL. A CT scan of the abdomen showed a 3-cm mass in the adrenal gland, with a central hypodense area and multiple hemorrhages. Microscopic examination of the mass revealed focal areas of necrosis with inflammatory changes. Therapy with hyroxyine was started. Six days later, he

Lef adrenalec omy was performed, revealing a 3 x 2 x 1-cm adrenal mass with a mullple hemorrhages. Microscopic examina ion of the mass revealed focal areas of necrosis with in inflammatory cells surrounded by fibrous tissue. The diagnosis of neurosyphilis was confirmed by the presence of Treponema pallidum in a biopsy. A CSF examination revealed a normal cell count and glucose concentration.

The clinical manifestations of neurosyphilis in our pa ien are consis en wi h neurosyphilis; the ypes of neurosyphilis commonly described are asymptomotica, meningeval, meningoovascular, and parenchymal, and here is of en overlap among the ypes [5]. Our pa ien ’s manifes a tions are predomina nly due to the presence of meningovascular syphilis, as manifested by memory impairment, weakness in the leh upper extremity, and ischemic op ic neuropathy in associaion with hyroid s imula ing hormone, 33.86