Fig. 3. RV, Right ventricle.

right hepatic vein and then into the hepatic tissue. It was noticed that after the contrast medium filled the hepatic sinusoidal bed, it coalesced to fill the *left* hepatic vein and then the common atrium, thus showing a steal phenomenon through the liver (Fig. 2). This hepatic steal created a right-to-left shunt because of the hepatic vein exclusion that had been done with the Norwood stage III operation. The relatively lower pressure in the left hepatic vein and atrium was "stealing" from the Fontan circulation, which accounted for the increasing cyanosis. Presumably, the interconnecting hepatic venous network allowed this to occur. In addition to this intrahepatic right-to-left shunt, the effective pulmonary blood flow also was reduced by this steal (Fig. 3).

This child underwent reoperation. A triangular piece of the existing polytetrafluoroethylene material was excised at the lower end of the lateral tunnel, and a new baffle was constructed to divert the left hepatic venous drainage into the lateral tunnel. After the reoperation the child's oxygen saturation was normal.

In an attempt to reduce effusions, liver congestion, and other adverse effects of the Fontan operation, the Philadelphia group¹ introduced partial hepatic vein exclusion by partitioning the IVC. Although this modification is attractive for its lessening of the morbidity of the Fontan operation, due consideration should be given to the possibility that late hepatic steal may occur. Our patient raises the spectre of late malfunction of the Fontan circulation after hepatic vein exclusion.

REFERENCE

1. Jacobs ML, Norwood WT. Fontan operation: influence of modifications on morbidity and mortality. In: Abstracts, Thirtieth Anniversary Meeting. The Society of Thoracic Surgeons, 1993-1994:44.

Primary cutaneous *Nocardia farcinica* infection after heart transplantation: A case report

Wolfgang Rees, MD, Stephan Schüler, MD, Manfred Hummel, MD, Susanne Hempel, MD, Jörg Möller, MD, and Roland Hetzer, MD, Berlin, Germany

Nocardia is known to cause severe infection, especially in immunocompromized patients with malignant disease, viral immunodeficiency syndrome, or in those patients receiving immunosuppressive therapy.¹ To our knowledge, a total of 47 cases of nocardiosis in cardiac transplant recipients have been published,² but this is the first case of primary cutaneous infection caused by *Nocardia farcinica* in a transplant recipient, without dissemination of the infection and without pulmonary or cerebral affection.

In May 1992, a 57-year-old male patient underwent orthotopic cardiac transplantation because of end-stage ischemic heart disease. Revascularization operations had already been performed in 1977 and 1988. The treatment regimen consisted of triple drug immunosuppression with initial induction therapy with rabbit antithymocyte globulin 100 mg for 3 days. The intraoperative and perioperative course was uneventful, and the rehabilitation program was performed as usual. No rejection episodes had to be controlled.

Seven weeks after transplantation, the patient began having a backache. Computed tomography (CT) of the spine showed typical signs of severe osteoporosis and osteoporotic fractures of the eighth and eleventh thoracic vertebrae. Treatment was started with conservative drug therapy and a corset. One month later at home, low back pain increased again and an emergency physician started analgesic treatment by intramuscular injection of nonsteroidal antiphlogistics. Radiologic reinvestigation showed beginning osteoporotic fracture of the tenth thoracic vertebra. Three weeks later, swelling, tenderness, and increasing pain developed in the left groin and thigh, and the man was admitted to his local hospital because of high fever, chills, malaise, and a swollen left thigh. A CT scan revealed a circumscribed hypodense intramuscular mass medial above the left knee, reaching the left groin (Fig. 1). Physical examination on admission was unremarkable.

From the German Heart Institute Berlin, Department of Thoracic and Cardiovascular Surgery, Berlin, Germany.

J THORAC CARDIOVASC SURG 1995;109:181-3

Copyright © 1995 by Mosby-Year Book, Inc.

0022-5223/95 \$3.00 + 0 12/8/52622

Fig. 1. CT scans showing circumscript hypodense intramuscular mass.

except for the left thigh, which showed erythema with tenderness and severe pain from the hip to the knee. No abnormality could be detected neurologically or on the chest roentgenogram. Sonographic controlled puncture of the liquid mass revealed pus. A migrating abscess caused by an intramuscular injection was assumed. Antibiotic therapy was started immediately with ampicillin/sulbactam 4.5 gm/day and gentamicin 240 mg/day. Subsequently, the patient was transferred to our hospital.

On admission, the patient was highly febrile. The white blood cell count was 16,900/ μ l and the plasma creatinine level was 1.29 mg/dl. Microbiologic investigation of the pus revealed growth of *Nocardia farcinica*. Antibiotic treatment was changed to high-dose imipenem (4 gm/day) and amikacin (1 gm/day) intravenously because the strain was sensitive to these drugs. Immunosuppressive therapy with azathioprine was stopped and cyclosporine whole blood trough levels were tapered to values between 150 and 200 ng/ml. No further sign of disseminated *Nocardia* infection could be detected by CT of the chest, spine, and brain.

Superficial division of the abscess was performed immediately. The fever disappeared, but swelling, erythema, and tenderness did not resolve. Eight days later, the patient had a convulsion with subsequent apnea. He was resuscitated successfully and imipenem treatment was stopped for 2 days, because imipenem was thought to be the cause of the convulsion, possibly as a result of the diminished excretion by the kidney (creatinine level increased to 1.97 mg/dl). After reduction of antibiotic therapy to imipenem 1.5 gm/day and amikacin 300 mg/day, swelling, tenderness, and pain in the left thigh increased again, and the fever recurred. Another CT scan of the left thigh showed multiple abscesses mainly involving the flexor muscles. Additionally, serum creatinine levels rose to 3.4 mg/dl and the patient had hypoacusis and

diplopia, which are typical signs of intoxication with amikacin. Antibiotic therapy was stopped for 2 days and continued thereafter with lowered dosages of imipenem 1 gm/day and amikacin 100 mg/day. Three days later, a second operation was performed. Excessive abscess dissection, wound débridement, and installation of a lavage-suction drainage were done. Intraoperatively, disseminated abscesses partially infiltrating the flexor muscles were detected.

The patient's condition improved dramatically during the next 3 weeks. Double drug antibiotic treatment was continued for a further month, and the patient was discharged on November 19, 1992, shortly after the antibiotic treatment had been changed to oral doxycycline monotherapy (100 mg/day). This was continued for another 3 months. A follow-up CT scan at time of discharge still revealed disseminated fluid accumulation in the left thigh. CT studies performed once a month after discharge showed slow regression of these structures. Three months later no abnormality could be revealed by CT scan. No recurrence of the infection has yet been seen.

Primary cutaneous disease is rare but may occur as a result of iatrogenic or traumatic inoculation because of the organism's wide resistance, especially to disinfectants, such as alcohol. Because no signs of pulmonary or cerebral manifestation of *Nocardia* infection could be detected, an iatrogenic cause due to intramuscular application of analgesics is the most probable source of this *Nocardia* infection. Silent infection of the saphenotomy wound where the bypass operation was performed some years earlier, with late reactivation of *Nocardia* because of aggressive immunosuppression, can be discussed but does not seem likely.

Because a high lethality for nocardiosis (40% to 80%)

has been described,³ prompt and effective surgical and drug therapy is essential.

REFERENCES

1. Krone A, Schaal KP, Brawanski A, Schuknecht B. Nocardial cerebral abscess cured with imipenem/amikacin and enucleation. *Neurosurg Rev* 1989;12:333-40.
2. Perschak H, Gubler J, Speich R, et al. Pulmonary nocardiosis concurrent with *Pneumocystis carinii* pneumonia in two patients undergoing immunosuppressive therapy. *J Infect* 1991;23:183-5.
3. Forbes GM, Harvey FA, Philpott-Howard JN, et al. Nocardiosis in liver transplantation: variation in presentation, diagnosis and therapy. *J Infect* 1990;20:11-9.

Myasthenia gravis complicated by sarcoidosis

Iwao Takanami, MD,^a Tetsuo Imamura, MD,^b and
Susumu Kodaira, MD,^a Tokyo, Japan

Myasthenia gravis (MG) complicated by sarcoidosis is rare. Whether this complication represents the association between the two diseases or a simple coincidence is unclear. We encountered a patient in whom MG seemed to have induced sarcoidosis.

A 32-year-old woman visited our department because of blepharoptosis in February 1990. The blepharoptosis was improved by the tension test. X-ray examination and computed tomographic scanning of the chest showed no abnormalities. Results of the serum acetylcholine receptor antibody test were positive. Electromyography showed the typical waning phenomenon after continuous peripheral nerve stimulation. On the basis of these findings, a diagnosis of MG was made. She was followed up without administration of anticholinesterase drugs.

In November 1992, she noticed fatigability of the skeletal muscles, difficulty in speech and swallowing, and frequent choking. Because of the exacerbation of MG, pyridostigmine bromide (60 mg) was administered daily. In July 1993, she was admitted to our department because of an abnormal shadow on chest X-ray films. Physical examinations revealed no palpable lymph nodes in the neck, axilla, or inguen. Examination of the eyes showed no uveitis.

Examination of peripheral blood samples showed no abnormalities in the leukocyte, erythrocyte, or platelet counts but an increase in the CD₄/CD₈ ratio among lymphocyte subsets. Urinary findings were within normal limits. The aspartate aminotransferase, alanine aminotransferase, alkaline phosphatase, and γ -guanosine triphos-

phate levels were within normal limits, but the thymol turbidity test level was 14.6 U, and the zinc turbidity test level was 24.2 U. The total protein concentration was 8.8 gm/dl with 51.0% α_2 -globulin and 29.8% γ -globulin. The serum level of angiotensin-converting enzyme was 23.4 U/ml, and the lysozyme level was 14.3 μ g/ml. The erythrocyte sedimentation rate was 56 mm/hr. The serum levels of triiodothyronine, thyroxine, and thrombin-stimulating hormone were within normal limits. Tests for thyroglobulin and mitochondria antibodies were negative. Rheumatoid factor and antinuclear antibody tests were negative, as was the positive protein derivative reaction.

Chest X-ray examination showed bilateral hilar lymphadenopathy. Chest computed tomographic scanning also showed marked bilateral hilar lymphadenopathy but no enlargement of the thymus or thymoma. Gallium 67 scintigraphy revealed accumulation-positive images on the bilateral hilar areas. A diagnosis of sarcoidosis complicated by MG (Osserman IIA) was made.

An operation was performed on October 19, 1993. After a T-shaped skin incision and longitudinal sternotomy had been done, the mediastinum was reached and extended thymectomy and biopsy of the hilar lymph nodes were performed. Histopathologic examination of the hilar lymph nodes demonstrated noncaseous granuloma, supporting the diagnosis of sarcoidosis.

Four months after the operation, she showed improvement in symptoms of MG with decreased blepharoptosis and diplopia and no fatigability of the skeletal muscles without administration of pyridostigmine bromide. Sarcoidosis has been observed after the operation without treatment, but no substantial changes have occurred in serum levels of angiotensin-converting enzyme or lysozyme.

MG is an autoimmune disease induced by the appearance of antiacetylcholine antibody. T cell abnormality is observed in the peripheral blood,¹ and germinal center formation is frequently observed in the thymus tissue. MG is closely associated with thymus abnormalities. On the other hand, sarcoidosis is a systemic disease characterized by formation of epithelioid cell granulomatous lesions not accompanied by necrosis, and its pathogenesis is unclear. However, recent information has suggested the following mechanism of the establishment of epithelioid cell granulomatous lesions, although the details are still unknown²: (1) T cells are activated after nonspecific stimulation or stimulation as an immune response to unknown cause, and activated T cells accumulate in the lesion; (2) accumulated T cells produce lymphokine; (3) lymphokine promotes the differentiation of monocytes/macrophages to epithelioid cells, resulting in the formation of epithelioid granuloma. Because the thymus plays an important role in the differentiation and growth of T cells, it is speculated that MG as "an unknown cause" activates the T cell line, inducing the formation of sarcoidosis lesions.

Some case reports of MG complicated by sarcoidosis have been reported. Sarcoidosis developed during the remission of MG in some cases³ but during the exacerbation period in others,⁴ showing no consistent tendency. Regression of MG and sarcoidosis after thymectomy was also reported.⁴ In our patient, MG became less severe after thymectomy, but sarcoidosis did not.

From the First Department of Surgery,^a First Department of Pathology,^b Teikyo University School of Medicine, Tokyo, Japan.

J THORAC CARDIOVASC SURG 1995;109:183-4

Copyright © 1995 by Mosby-Year Book, Inc.

0022-5223/95 \$3.00 + 0 12/8/55535