oped a tear in the intima of the aorta at the attachment of the commissure between the left and noncoronary cusps of the aortic valve after blunt chest injury. Attempts were made to repair the valve, but replacement was eventually required. Devineni and McKenzie,3 Schnitzelberg and Khalil,5 and German et al6 have also reported cases of aortic valve injury after blunt trauma. In those cases, successful treatment required valve replacement.

The mechanism of valve injury is thought to be a sudden increase in intrathoracic pressure during diastole when the valve is closed. Tears across a cusp and avulsion of a cusp from the annulus have been described. In contrast to aortic valve injury from blunt trauma, rupture of the thoracic aorta is not unusual, accounting for 15 to 20% of deaths among victims of automobile accidents.7 It is commonly thought that 80% of patients who suffer this injury die at the scene and another 10 to 15% die in the hospital. Many of these patients have second injuries that would prove fatal even if the aortic rupture had not occurred.

The most common mechanism of aortic rupture is the force generated by rapid deceleration of the body. The greatest strain in the thoracic aorta is at the isthmus where the mobile thoracic aorta joins the more fixed aortic arch. It is this area where most aortic ruptures are identified.

Aortic valve disruption and rupture of the thoracic aorta share a common mechanism of injury. It is not unusual that these injuries could occur together. Most of these patients die at the scene of the accident from the thoracic and cardiac trauma or from associated fatal second injuries.

In the case presented, the patient had an abnormal chest radiograph suggestive of aortic transection in addition to gross hemoperitoneum by lavage. We believed that the abdominal injury should take precedence because he was actively bleeding from this site. An arch aortogram was performed directly after surgery for diagnosis of aortic transection. Transesophageal echocardiography performed during the exploratory laparotomy would have been the diagnostically most valuable had the technology been available to us at the time. This would have easily demonstrated the aortic disruption as well as the valve insufficiency. We would then have proceeded to thoracotomy and repair of the aorta without delaying for an arteriogram.

Postoperatively, the patient developed congestive heart failure. This was not unexpected in the face of acute aortic regurgitation. Despite this, we felt it more prudent to manage his failure medically than to place a patient with multiple grade II liver lacerations on cardiopulmonary bypass immediately after exploratory laparotomy and thoracotomy. Aortic valve replacement was subsequently performed without complications.

The most unusual feature of this case is the favorable outcome for the patient. He had two routinely fatal injuries (cardiac and aortic) in addition to intra-abdominal and orthopedic trauma. Standard trauma protocol required emergency laparotomy to prevent exsanguination followed by diagnosis and repair of the thoracic aortic injury. The patient subsequently required aortic valve replacement for acute aortic insufficiency. Currently, at 3½ years postoperatively, the patient is leading a normal life.

References

Transmission of Invasive Aspergillosis From a Subclinically Infected Donor to Three Different Organ Transplant Recipients*

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Objective: To describe a cluster of donor-transmitted cases of invasive aspergillosis.

Design: Case series of epidemiologically linked cases of invasive aspergillosis.

Setting: Two tertiary care centers with solid-organ transplant programs.

Patients: Two kidney recipients, one heart recipient, and the single donor.

Measurements: Routine clinical, microbiological, and pathologic investigation as dictated for patient care. Epidemiologic analysis to establish linkage among cases.

Results: Three allografts (two kidneys and a heart) from a single donor transmitted invasive aspergillosis to the recipients. Three weeks after transplantation, the two kidney recipients had fever and urine cultures positive for Aspergillus fumigatus. The infected kidneys had multiple Aspergillus abscesses and had to be removed to cure the patients. The heart recipient had a negative workup when a diagnosis of aspergillosis was made for the kidney recipients but presented three months later with aspergillosis endocarditis with...

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hematogenous spread to the eyes and to the skin. Treatment included eye surgery, aortic valve replacement, and antifungal therapy; control of infection ensued. The donor was intensely immunosuppressed (17 days post-liver transplantation with death from intracerebral bleeding) but had no clinical or autopsy evidence of aspergillosis. Donor tracheal secretions obtained at the time of organ harvest later grew A fumigatus.

Conclusion: Expanded criteria for organ donation have to be balanced against infectious risk to organ recipients. A fumigatus can be transmitted from a subclinically infected donor to solid-organ transplant recipients. (CHEST 1996; 109:1119-24)

Key words: aspergillosis; Aspergillus fumigatus; heart transplantation; liver transplantation; opportunistic infections; tissue donors and mycoses

Donor-transmitted infections occur after organ transplantation, although less commonly than nosocomial infections and opportunistic infections. Recipients are at highest risk during intensive induction immunosuppression. Bacterial contamination of the donor organ is rare with current organ preservation techniques. More commonly, latent or subclinically present microorganisms in the donor organ are knowingly or unknowingly transferred to the new host. For example, cytomegalovirus and Toxoplasma-sero-negative recipients are at high risk of developing symptomatic primary infections when their organ donor is seropositive. The risk of these primary infections is knowingly accepted although it is reduced as much as possible by various treatment strategies. In contrast, hepatitis B and C and HIV are never knowingly transmitted. With current donor evaluation protocols, the risk of transmitting these viruses has been drastically reduced. Tuberculosis, histoplasmosis, and cryptococcosis on rare occasions have been unknowingly transmitted through organ transplantation.

We report a small outbreak of four related cases of invasive aspergillosis that occurred when two kidneys and the heart were harvested from a donor with unsuspected invasive aspergillosis. The recipients all developed invasive aspergillosis in their respective allografts, and in one case there was hematogenous spread to the eye and the skin. The organ donor was intensely immunosuppressed (17 days after liver transplant, death occurred as a result of intracerebral bleeding), but there was no clinical or autopsy evidence of aspergillosis.

Overview of the Outbreak

Recipient one (index case) presented three weeks after cadaveric renal transplantation with persistent low-grade fever and urine cultures positive for Aspergillus fumigatus. The course was subacute over several weeks. There was high suspicion of renal aspergillosis, but imaging studies were negative. The patient clinically deteriorated and developed acute respiratory failure due to cytomegalovirus infection. He remained febrile and toxic despite therapy with ganci clovir. The urine cultures were persistently positive for A fumigatus despite intravenous therapy with amphotericin B. The kidney was removed, showing multiple Aspergillus abscesses (Fig 1, A). As soon as renal aspergillosis was documented, the allograft was traced back to the donor, who
was an intensely immunosuppressed liver transplant recipient. Investigation revealed that donor tracheal secretions obtained at the time of organ harvest later grew A fumigatus and that the other kidney and the heart had also been transplanted. The identities of the other two allograft recipients were obtained. The second kidney recipient had low-grade fever without localized findings. Fungal cultures of the urine were obtained and were positive for A fumigatus. Cultures remained positive despite therapy with intravenous amphotericin B. An MRI study showed multiple luencies in the allograft, and the kidney was removed. Gross pathologic findings were similar to those of the first case, with multiple abscesses (Fig 1, B). Neither kidney recipient had distant spread from the allograft to other sites, and both recovered after removal of the allograft and after continued treatment with antifungal therapy. The heart recipient was evaluated shortly after the two infected kidneys were removed. No evidence of aspergillosis was found. Three months later, Aspergillus endocarditis developed with hematogenous spread to the skin and to the eye. The patient recovered after surgical (including aortic valve replacement and eye surgery) and medical therapy for invasive aspergillosis. Table 1 highlights the clinical course of illness in the three recipients.

**CASE REPORTS**

**RECIPIENT 1 (INDEX CASE)**

A 42-year-old man with end-stage renal disease from remote postinfectious glomerulonephritis received a cadaveric renal transplant on August 12, 1993. Details of the initial immunosuppression are included in Table 1. The early postoperative course was uneventful, and the patient returned to work within 2 weeks. Three weeks after transplantation, he developed mild fevers with no obvious source. The serum creatinine value increased from 2.0 to 2.6 mg/dL. Percutaneous biopsy of the allograft on September 7, 1993, revealed minimal focal acute cellular rejection and no vascular rejection. He was treated with a short course of high-dose methylprednisolone (3.5 g over a period of 6 days). A chest roentgenogram was negative, and blood cultures were negative for bacteria, fungi, and cytomegalovirus. Urinalysis showed more than 50 white blood cells per high-powered field, but urine cultures were negative for bacteria and for cytomegalovirus. On September 15, 1993, urine cultures for fungus were obtained and were positive for A fumigatus. Fever persisted. On September 17, 1993, blood cultures were positive for cytomegalovirus. Subsequent urine cultures were positive for A fumigatus, and direct smears of the urine showed fungal hyphae. Outpatient therapy with oral itraconazole, 200 mg/d, was begun.

On September 25, 1993, the patient was admitted to the hospital with a grand mal seizure. A CT scan and MRI studies of the head both showed two areas of infarction, possibly old because of some associated atrophy. A lumbar puncture was negative. Another blood culture was positive for cytomegalovirus and repeat urine culture again grew A fumigatus. Therapy with intravenous ganciclovir (200 mg qd) and intravenous amphotericin B (0.7 mg/kg/d=50 mg/d) was begun. Immunosuppression was reduced (prednisone reduced to 20 mg/d, azathioprine stopped, and cyclosporin continued at 200 mg twice daily). Fever persisted and increased in magnitude with daily temperature spikes to 40°C. The patient developed progressive respiratory distress and diffuse pulmonary infiltrates. He required mechanical ventilation with positive end-expiratory pressure. Bron-
choscopy with BAL revealed lymphocytic alveolitis. Cultures of BAL fluid were positive for cytomegalovirus but negative for bacteria and for fungi. The immunosuppression was reduced further. Therapy with ganciclovir was continued and the amphotericin B was changed to a liposomal preparation (Amphotericin B Lipid Complex; The Liposome Company; Princeton, NJ) to preserve renal allograft function. Urine cultures remained positive for *A. fumigatus*. Several attempts to document fungal abscesses in the allograft were not successful. A non-contrast CT study of the abdomen and a renal ultrasound study disclosed no abnormalities. Respiratory failure improved slowly though ventilatory support was still necessary.

Transplant nephrectomy was performed on October 13, 1993. Gross pathologic specimens showed multiple large abscesses throughout the kidney (Fig 1, A). Histopathologic samples showed large areas of necrosis. Numerous fungal hyphae were seen with septae and acute branching, morphologically consistent with *Aspergillus* species. The patient slowly improved and became afebrile within 1 week, after having had persistent fever from September 1, 1993. He was weaned from mechanical ventilation 10 days after transplant nephrectomy. Antifungal therapy was changed back to regular amphotericin B, which was continued through November 11, 1993, to a total cumulative dose of 2 g. At follow-up 25 months after the unsuccessful renal transplantation, the patient was well while receiving long-term outpatient hemodialysis.

### Transplant Donor

A 56-year-old man with end-stage liver failure due to α-1-antitrypsin deficiency and cirrhosis underwent orthotopic liver transplantation on July 25, 1993. Immunosuppression included methylprednisolone, 1.0 g twice on the first day, followed by 100 mg of methylprednisolone daily for the next 2 days. After that, the patient received 40 mg of prednisone daily with a planned slow tapering toward 20 mg/d. He also received azathioprine (2 mg/kg/d) and cyclosporine (cyclosporin A) (initially 2 mg/kg twice daily with dosage adjustments dictated by serum levels), but he did not receive any antilymphocyte therapy. Thrombocytopenia, which antedated transplantation, persisted postoperatively. Apart from large intraoperative transfusion requirements, the transplantation was uneventful and the immediate postoperative course was notable only for mild renal impairment and cardiac rhythm disturbances. A liver biopsy, performed on August 2, 1993, because of increasing serum transaminase and bilirubin values, showed cholestatic hepatitis consistent with moderate cellular rejection. Bolus methylprednisolone therapy was given on three successive days and triple immunosuppression was continued. In spite of antirejection therapy, there was further deterioration in liver function and the patient became mildly confused and deeply jaundiced. A Doppler examination of the liver on August 5, 1993 showed arterial flow in the left lobe of the liver but uncertain flow in the right lobe. On August 9, 1993, the Doppler examination was repeated and failed to detect any hepatic artery flow. A hepatic arteriogram on the same day confirmed proximal occlusion of the hepatic artery graft, and the patient was activated for retransplantation. On August 10, 1993, the patient developed sudden loss of consciousness. A CT scan of the head revealed massive intracranial bleeding with blood in the ventricular system. Brain death was diagnosed later in the day, and he was considered for organ donation because heart and kidney function were normal.

The patient had remained afebrile throughout the posttransplantation course except for a single temperature elevation of 38.4°C 8 h prior to the intracranial bleeding. No source of fever was defined and no treatment was given. A chest roentgenogram showed a focal area of increased density in the left lower lobe suggesting a possible early infiltrate. Cultures of blood and urine for bacteria, fungi, and virus and cultures of abdominal fluid for bacteria and fungi were negative. No sputum was produced or cultured, but culture of a tracheal aspirate obtained at the time of organ donation later grew *A. fumigatus*. The results were not available until after the organs had been transplanted. An autopsy performed on the donor revealed evidence of bronchopneumonia in dependent areas of the lungs. There was no gross or microscopic evidence of pulmonary or systemic *Aspergillus* infection at the time of autopsy.

### Recipient 2

A 47-year-old woman with end-stage renal disease from glomerulonephritis received a cadaveric renal transplant on August 12, 1993, from the same donor as recipient 1 (index case). Details of the initial immunosuppression are included in Table 1. The early postoperative course was complicated by hypertension and decreased renal function. Renal function improved with antirejection therapy which consisted of several days of high dose (500 to 1,000 mg) intravenous methylprednisolone with rapid tapering. Low-grade fever began about September 1, 1993. Cultures of blood and urine for bacteria and for cytomegalovirus were negative on September 14, 1993. On September 28, 1993, fever increased to 39°C. A transplant biopsy was done on September 28, 1993, and showed some chronic changes but no acute cellular rejection and no vascular rejection. Bacterial, cytomegalovirus, and fungal blood cultures were negative. On September 28, 1993, urinalysis showed 15 to 19 WBCs per high-powered field. Urine cultures for bacteria and cytomegalovirus were negative, but urine culture was positive for *A. fumigatus* and direct smears of the urine showed fungal hyphae. Therapy with intravenous amphotericin B was started (0.7 mg/kg/50 mg every other day). The patient remained febrile, and urine cultures remained positive for *A. fumigatus* during the first 200-nm cumulative dose of amphotericin B. An MRI study showed multiple round areas of increased T2 signal intensity compatible with abscesses, and the patient had a transplant nephrectomy on October 15, 1993. The pathologic findings (Fig 1, B) were remarkably similar to those of the index case with numerous large abscesses. Numerous fungal hyphae were identified with septae and acute branching, morphologically consistent with *Aspergillus* species. The patient became afebrile within 3 days of transplant nephrectomy. Intravenous amphotericin B therapy was continued to a total cumulative dose of 2 g. At follow-up 25 months after the unsuccessful renal transplantation, the patient was well while receiving long-term outpatient hemodialysis.

### Recipient 3

A 64-year-old man with ischemic cardiomyopathy underwent orthotopic heart transplantation on August 11, 1993. Details of the initial immunosuppression are included in Table 1. The postoperative course was uneventful. He was discharged from the hospital on August 22, 1993, in excellent condition. Over the ensuing 5 months, he did very well. There were no episodes of infection, and ten serial surveillance right endocardial biopsies showed no evidence of rejection. A thorough evaluation in late October 1993 was prompted by the clinical courses of recipients 1 and 2. Results of physical examination were within normal limits, and a chest roentgenogram was normal. The patient was afebrile and clinically well.

On January 30, 1994, he was admitted to the hospital with a 1-day history of blurred vision. Two days prior to admission, he had noticed double vision. Five days prior to admission, he had noticed a tender nodule on the right palm. On admission, he was afebrile with normal vital signs. The right eye was swollen and erythematous. Funduscopic examination revealed a lazy vitreous. There was a tender, 1-cm, purplish nodule in the right palm. The heart sounds were normal and no murmur was detected. Results of the remainder of the physical examination were within normal limits.

A transthoracic echocardiogram was normal. A pars plana vitrectomy of the right eye was performed. A fungal smear of vitreous humor revealed fungal hyphae, and culture grew *A. fumigatus* in 1 day. A biopsy of the lesion of the right palm showed abscesses.
and necrosis with negative fungal stains, but fungal cultures ultimately grew *A. fumigatus*. On February 1, 1994, a transesophageal echocardiogram showed a large vegetation on the aortic valve, and the patient was prepared for urgent thoracotomy. Aortic valvular vegetations and an adjacent intramyocardial abscess were encountered. The aortic valve was excised and replaced with a homograft. The abscess cavity was debrided and painted with phenol. Cultures of aortic valve tissue, coronary cuff, and abscess material all grew *A. fumigatus*. The patient was treated with intraaortic amphotericin B and also, in sequence, systemic amphotericin B followed by liposomal amphotericin B (Amphotericin B Lipid Complex) followed by oral itraconazole. Serial CT studies of the abdomen, pelvis, chest, and head did not detect evidence of further dissemination. The patient was well at follow-up 18 months after the aortic valve replacement.

**DISCUSSION**

Latent or subclinically present microorganisms in a donor organ can be knowingly or unknowingly transmitted to the recipient.\textsuperscript{1,2} Most cases involve transmission of latent viruses including cytomegalovirus, hepatitis B and C viruses, and HIV, among others. However, tuberculosis, histoplasmosis, and cryptococcosis all have been transmitted from donors with unsuspected infections to the recipients of their infected organs. Candida and monosporium infections have been transmitted in a similar manner.

The cluster of cases reported here provides strong evidence that Aspergillus infection also can be transmitted with allografts from an infected donor to the new hosts, even without clinically apparent infection in the donor at the time of surgery. The evidence is strong but is based entirely on the epidemiology. It would have been better if the isolates could have been genetically linked, but this was not possible because the Aspergillus isolates had all been discarded before the unusual nature of the outbreak was manifested.

The evidence that the kidney infections were donor-transmitted is very strong. Although we believe strongly that the heart infection was also donor-transmitted, it is remotely possible that the Aspergillus endocarditis could have been an isolated event related to immunosuppression. This would have required a remarkable coincidence. The most common type of invasive aspergillosis in heart recipients is pulmonary aspergillosis, which was not documented in this recipient. Rather, he had Aspergillus endocarditis in the transplanted heart. For the Aspergillus endocarditis to be independent, the remarkable history of the severely immunosuppressed donor and the unusual fate of the kidney recipients from that donor would have to be totally unrelated, which is highly unlikely. This small outbreak serves to emphasize that unusual opportunistic infections appearing in any allograft should prompt review of the donor history and notification of other recipients from the same donor.

Donor tracheal secretions grew *A. fumigatus* after transfer of the organs. Although preventive antifungal therapy is recommended for lung transplant recipients whenever Candida or Aspergillus organisms are recovered from donor respiratory tract secretions,\textsuperscript{13,14} the situation in lung transplantation is quite different because the lung itself is transferred to the recipient.

Virtually all unrelated solid-organ donors are in ICUs, are intubated, and are receiving mechanical ventilation. These donors almost universally have colonization of the respiratory tract with various bacterial and fungal organisms. The natural history of this outbreak suggests that in this case the Aspergillus organisms were not merely colonizing the airway. Rather, there was invasion of the donor lung and early hematogenous dissemination at least to the kidneys and the heart. The chest radiograph of the donor showed a minimal infiltrate in the left lower lobe. Autopsy showed bronchopneumonia in dependent areas of the lung, with no evidence of aspergillosis even with special stains. Although the initial donor autopsy and a subsequent reexamination of all autopsy material (including lung tissue from multiple lobes) did not reveal any evidence of invasive aspergillosis, only selected samples of the donor lung were examined microscopically. Either the infiltrate was early aspergillosis but was not sampled or the infiltrate was simply bronchopneumonia, and the Aspergillus infection involved areas of the lung that were normal through radiographic means and by gross inspection.

The intense immunosuppression of the donor had allowed Aspergillus organisms to invade; had he survived, clinically apparent invasive aspergillosis would likely have evolved. In one large retrospective study of liver transplant recipients with sputum cultures positive for Aspergillus species, 72% had invasive pulmonary aspergillosis and 28% only were colonized.\textsuperscript{13,14} No patient had clinically apparent dissemination to distant sites. This data base for this study included all liver transplant recipients (more than 2,000) over a 10-year period at the University of Pittsburgh. By protocol, any diagnostic sputum sample obtained from these patients was always cultured for fungus as well as for bacteria.

Urinary cultures positive for Aspergillus species and direct smears of the urine positive for septate hyphae are very unusual findings and strong evidence for renal aspergillosis. The urine cultures in the two kidney recipients remained positive despite amphotericin B therapy, perhaps not surprising in view of the poorly perfused necrotic Aspergillus abscesses in the allograft kidneys. The poor sensitivity of standard imaging techniques was not expected. For the first kidney recipient, a CT study with contrast might have been more sensitive. Fear of contrast-induced renal injury prohibited this study. For the second kidney recipient, MRI of the kidney was helpful. (The first kidney recipient was too sick for an MRI study.) The heart recipient had a negative clinical evaluation in October 1993. He had a negative transthoracic echocardiogram on admission to the hospital on January 30, 1994. A transesophageal echocardiogram 2 days later showed vegetation. However, the hospital admission was prompted by eye findings and skin findings and the diagnosis of invasive aspergillosis had been proven from these distant sites before the transesophageal echocardiogram was done.

The Aspergillus infections acquired with the allografts were subacute with incubation periods ranging from 3 weeks to 6 months. The infections in the kidneys did not clinically spread beyond the kidneys despite many weeks of active infection. Even more surprising, the heart infection remained subclinically present for 6 months. Ten weeks post transplant, the heart recipient was evaluated after renal aspergillosis developed in the two kidney recipients. He was afebrile without a heart murmur. Lacking clinical indications of endocarditis, transesophageal echocardiography was not done. This test is highly sensitive for vegetations\textsuperscript{15} and...
among available tests, would have had the best chance of showing an early change on the valve. Transthoracic echocardiography at that time would not have helped since it was negative even in late January when the transesophageal study showed large vegetations.

There is a great shortage of organs for transplantation. Many potential recipients die because a suitable organ is not available. There is a natural tendency to maximally expand the donor pool and to relax exclusion criteria to the limits of safety. Regulatory requirements also dictate external determination of donor suitability so that potential donor opportunities are not wasted. Organs are now harvested from noninfected immunosuppressed patients and even harvested a second time if a recipient dies a noninfectious death without graft rejection. Organs are sometimes taken from donors with systemic bacterial infections when the infection is highly responsive to antibiotics and does not directly involve the donated organ. This outbreak serves to emphasize that there still is a limit to safety. Liver transplantation is associated with a higher risk of invasive aspergillosis than transplantation of other solid organs (4 to 10% in some series). Exclusion of subclinically present infection in distant organs may not be totally possible. Surveillance cultures positive for Aspergillus organisms should prohibit serial organ donation from a liver recipient, but as in this outbreak, the results may lag the organ transfer. This donor, although lacking specific donor exclusion criteria, was in retrospect too immunosuppressed to serve as an organ donor. Perhaps any liver transplant recipient at day 17 is too immunosuppressed to reliably exclude subclinically present infections and recipient risk from direct transfer of microorganisms.

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**REFERENCES**


**Noninvasive Diagnosis and Treatment of a Saddle Pulmonary Embolism**

A Case Report in Support of New Trends in Management of Pulmonary Embolism

Adam Torbicki, MD; Ryszard Pacho, MD; Piotr Jedrusik, MD; and Piotr Pruszczyk, MD

Transesophageal echocardiography and contrast-enhanced spiral CT of the chest helped to avoid a pulmonary angiography in an elderly patient with saddle pulmonary thromboembolism and allowed for direct evaluation of its resolution during treatment with subcutaneous low molecular weight heparin.

(CHEST 1996; 109:1124-26)

**LMWH=low molecular weight heparin; PE=pulmonary embolism; TEE=transesophageal echocardiography**

**Key words:** computed tomography; low molecular weight heparin; pulmonary embolism; transesophageal echocardiography

Definitive confirmation of pulmonary embolism (PE) is still reserved for pulmonary angiography. Only then is