

Fatal Transfer of Malignant Melanoma From Multiorgan Donor To Four Allograft Recipients¹²

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Received 6 December 1999.

Accepted 15 March 2000.

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² Supported in a part by NCI Cancer Center Grant CA46934.

¹ Presented in part at the 87th Annual Meeting of the United States and Canadian Academy of Pathology Meeting, Boston, MA, March 4, 1998.



Abstract

Background. In this report we describe the transfer of malignant melanoma from a single donor to four solid organ transplant recipients, all of whom died from metastatic melanoma.

Methods and Case Histories. The donor of a heart, liver, and two kidneys to four separate recipients died of intracerebral hemorrhage. The donor had no history or clinical evidence of melanoma. All four recipients, treated with standard immunosuppression protocols, developed metastatic malignant melanoma within 1 year

after transplantation Three patients died within 14 months after transplantation, although the fourth, whose immunosuppressive therapy was discontinued, died of metastatic melanoma 30 months after renal transplantation.

Findings: Tumors from all recipients were histologically identical. Donor origin of tumor cells was confirmed by polymerase chain reaction (PCR)-based DNA analysis for polymorphic short tandem tetrameric repeats (Geneprint STR, Promega Corp., Madison, WI). DNAs from nontumorous donor tissue and tumor tissue available from three recipients tested positive for CSF1P0 alleles 10 and 12 and for TH01 alleles 6 and 7, although DNAs from nonneoplastic recipient tissues all exhibited different allelotypes.

Interpretation. Transmission of fatal or potentially fatal malignant tumors, notably malignant melanoma, from donor to recipient is an uncommon complication of solid organ transplantation. PCR-based genetic analysis permits definitive assignment of the source of posttransplant tumors.

Malignant tumors are a significant cause of long-term morbidity and mortality in allograft recipients. The incidence of cancer in patients undergoing solid organ transplantation ranges from 4 to 18% (¹, ²). Tumors at increased incidence in transplant recipients include squamous and basal cell carcinomas of the skin, hepatocellular carcinoma, sarcoma, carcinomas of the uterine cervix, vulva, and kidney (¹⁻⁴) and lymphoproliferative disorders (⁵). Most posttransplant solid tumors are assumed to arise primarily from recipient tissues but Penn and co-workers (⁶) have found that nearly half of patients receiving allografts from donors known to have cancer, develop tumors thought to be of donor origin. Donor tumors that have been transferred to recipients include lung cancer, choriocarcinoma, malignant melanoma and renal carcinoma (⁷⁻¹³). Recipients to whom donor tumors are inadvertently transferred commonly develop widespread metastatic disease that has been treated by graft removal, reduction of immunosuppression, and/or tumor-targeted chemotherapy (⁶). Only a small percentage of these recipients have survived despite treatment. Early recognition of the donor origin of transferred tumors may improve chances of survival, but may be difficult if tumor is not clinically recognized in the donor.

Recently it has become possible to definitively identify the source of tumor cells by polymerase chain reaction (PCR)-based molecular analysis. Using such methods, Loh and co-workers (¹³) have previously reported the occurrence of donor-derived prostate carcinoma in a transplant recipient. They raise the question of whether the incidence of donor-derived malignancy is higher than previously suspected. We report the occurrence of malignant melanoma in multiple recipients received from a single donor. The donor died of cerebral hemorrhage and had no history or physical signs of melanoma. Immunohistochemical and PCR-based genetic analysis conclusively determined that the tumors were of donor origin.

In our study, the first recipient was a 70-year-old woman who underwent renal transplantation for end-stage renal failure due to hypertension and type II diabetes mellitus. She initially did well, experiencing no episodes of rejection. However, 15 months after transplantation she developed a urinary tract infection complicated by sepsis and poor renal function. The transplanted kidney was removed and found to contain malignant melanoma. She died 17 months after transplantation. An autopsy documented widespread malignant melanoma. Recipient 2 was a 50-year-old white man

who underwent liver transplantation for cryptogenic cirrhosis. He presented 15 months after transplantation with shortness of breath. A chest x-ray showed diffuse lung infiltrates; a CT scan of the chest and abdomen showed multiple lung and liver lesions. A transjugular liver biopsy revealed metastatic malignant melanoma.

Immunosuppressive agents were discontinued and chemotherapy was begun. However, he died within a few days of the initiation of treatment, 16 months after transplantation. An autopsy confirmed the presence of widespread metastatic tumor. The third recipient was a 62-year-old man who underwent cardiac transplantation for severe ischemic cardiomyopathy with recurrent atrial arrhythmias. He suffered one episode of rejection, which was treated with OKT3. Ten months after transplantation, a routine chest x-ray revealed multiple pulmonary nodules. CT scan of the chest, abdomen, and pelvis revealed liver lesions. MRI of the brain showed numerous metastatic lesions. Biopsy of one of the lung nodules revealed metastatic melanoma. Immunosuppression was decreased. The patient declined further treatment and died 13 months after transplantation. No autopsy was obtained. Recipient 4 was a 19-year-old man with Alport's disease who did well for 17 months after transplantation at which time his physician was notified of the possibility that tumor may have been transmitted to him with the allograft. A CT scan performed at that time indicated that multiple masses were present in the kidneys, liver, and lungs. Immunosuppressive therapy was promptly discontinued but his disease progressed and he expired 30 months after receiving his allograft, more than 1 year after the other recipients.

For histological studies, fixed, paraffin-embedded tissue was stained with hematoxylin and eosin and examined by conventional light microscopy. Immunohistochemical stains were performed by a Streptavidin technique (Biogenix) using antibodies against S100 (polyclonal, Dako Corporation), melanoma specific antigen (HMB-45, monoclonal, Dako Corporation), pan-cytokeratin (monoclonal, Signet Corporation), leukocyte common antigen (monoclonal, Shandon Corporation), vimentin (monoclonal, Dako Corporation), and desmin (monoclonal, Dako Corporation). Cells were graded for intensity, percentage of cell positive, and intracellular localization of stain precipitate.

Nonneoplastic donor tissue was available as well as tumor and nonneoplastic tissue from three of the four recipients. Tumor tissue was isolated from nonneoplastic tissue by dissection of wet formalin-fixed tissue or paraffin blocks using a scalpel or razor blade. Paraffin-embedded tissue was then thoroughly deparaffinized in xylene and rehydrated through graded ethanol solutions to water. Cellular fragments weighing 100 to 300 mg. from both wet tissue and paraffin blocks were then washed with tris-EDTA buffer. DNA was extracted by digestion with proteinase K followed by purification on a DNA binding membrane (QIAamp Tissue kit, Qiagen Inc., Santa Clarita, CA). DNA fingerprinting was performed to determine the source of each tumor, by analysis of polymorphic short tandem repeats in DNA extracted from tumor and from multiple uninvolved donor and recipient organs. Polymorphic short tetrameric repeat loci were amplified using primers provided in the Geneprint STR System (CTT Triplex, Promega Corp., Madison, WI) according to the protocol of Lins et al. (14). This PCR amplification system contains three multiplexed primer pairs flanking tetrameric tandem repeats in the CSF1PO, TPOX, and TH01 genes. The PCR products, and appropriate allelic markers were then electrophoresed on a 6% sequencing gel at 50°C and stained by a silver staining technique to visualize amplified allelic bands (14).

Hematoxylin and eosin stained sections of tumors were available for review from recipients 1, 2 ([Figs. 1, A and B](#)), and 3, and were histologically identical. All tumors were composed of sheets of cells with large nuclei, conspicuous nucleoli, a moderate amount of eosinophilic cytoplasm and a high mitotic rate. Necrosis was a prominent finding. Immunohistochemical stains for melanoma-associated antigen (HMB-45) were strongly positive ([Fig. 1C](#)), and S-100 protein was focally expressed by tumor cells. Additional immunohistochemical stains performed on tumor tissue from recipients 2 and 3 for cytokeratin, leukocyte common antigen, vimentin, and desmin were negative. Based on histological appearances and immunohistochemical profiles, tumors were classified as metastatic melanoma.

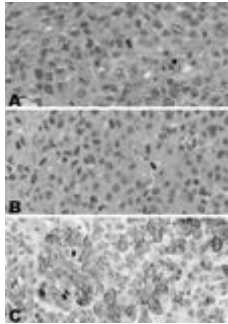


Figure 1

[Image Tools](#)

Results of genetic fingerprint analyses are summarized in [Table 1](#) and illustrated in [Figure 2](#). Two loci, CSF1PO and TH01, were informative in all cases. Recipient alleles at these loci differed from donor alleles in all specimens tested. Tumor DNA displayed donor alleles and often minor bands representing recipient alleles and reflecting the presence of DNA from benign stromal cells, leukocytes, or benign epithelial cells admixed with the tumor cells. These results confirmed the donor origin of all of the tumors in the series.

Table 1 is a small table with multiple columns and rows, likely containing genetic fingerprint analysis results for various samples. The text is too small to read.

Table 1

Figure 2

[Image Tools](#)[Image Tools](#)

Organ transplantation is a vital option for many patients. It is accepted that rejection and disease recurrence may lead to graft failure, and that immunosuppression carries with it a risk for development of various malignancies. Our study illustrates a potentially lethal complication of transplantation in which previously undiagnosed tumors within donated organs may be inadvertently transferred to recipients. The course of such tumors may be aggressive or even fatal in immunosuppressed hosts. We report cotransplantation of malignant melanoma with heart, liver, and kidney allografts into four recipients, all of whom developed biopsy proven evidence of metastatic melanoma and died. It is presumed that tumor cells were present within each of the donated organs and were not removed by the perfusion process used to maintain allograft homeostasis before transplantation. Because of the immunosuppression received by the allograft recipients and in the absence of significant episodes of rejection, the metastatic cells were apparently not destroyed as would be expected in an immunocompetent host ([6](#), [13](#)). Using PCR-based DNA analysis for polymorphic short tandem tetrameric repeats, the

common donor origin of these metastatic tumors was confirmed. The finding of donor alleles within the recipient tumor is not likely to be a result of donor cell migration with microchimerism ([15-17](#)), because analysis of normal tissue DNA from three of the four recipient in this series contained no donor alleles.

Although transfer of malignant tumor from donor to recipient is thought to be a rare event, its true incidence is unknown because molecular methods for assessing tumor source have become available only recently. Donor origin of tumors occurring in organ transplant recipients may be suspected in those who develop tumors within 2 years of transplantation and whose tumor involves the donated organ ([6](#), [13](#)). In cases of multi-organ donation, information regarding the occurrence of tumor in other recipients may be crucial to raising the suspicion of donor origin. Molecular analysis should be performed expeditiously because prompt determination of donor origin will be crucial to clinical management ([13](#)). For recipients of kidneys or kidney-pancreas transplants, immunosuppression should be discontinued immediately and, if necessary, the transplanted organ(s) removed. In recipients of heart or liver transplants, immunosuppression may be significantly decreased or discontinued. Surgical removal of tumor and chemotherapy may be considered in all patients with transplanted tumors ([6](#)).

The importance of thorough screening of potential donors for malignancy is highlighted by these cases ([6](#), [13](#)). The screening process should include a thorough review of the medical record including radiographic and laboratory studies. At the time of harvest, the surgical team should carefully examine the body for possible tumor, enlarged lymph nodes, or other abnormalities. Any suspicious tissue should be examined by frozen section. With these efforts, a balance should be achieved that allows for maximum use of donor organs and successful long-term engraftment.

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Acknowledgments.

The authors thank Dr. Cass Franklin, Methodist Hospital, Des Moines, IA for clinical follow-up information.

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