reducing costly complications, improving efficiencies of care, and reducing resource utilization.

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TRANSMISSION OF HUMAN T-LYMPHOTROPIC VIRUS TYPE I BY BILATERAL LIVING-DONOR LOBAR LUNG TRANSPLANTATION

To the Editor:

Human T-lymphotropic virus type I (HTLV-I) is an etiologic agent for adult T-cell leukemia/lymphoma and HTLV-I–associated myelopathy/tropical spastic paraparesis. Most HTLV-I infections are attributable to transmission from mother to child or to sexual contact later in life, whereas transfusion is perhaps the most efficient mode of viral transmission. Seroprevalence of HTLV-1 among high-risk populations is 10% to 30% in the southern area of Japan, such as Okinawa, and approximately 5% in the Caribbean region compared with 1% in Europe and the United States.

There have been several reports of HTLV-1 transmission in association with organ transplantation (Table 1).[1-4] However, no report describes a transmission of HTLV-1 by lung transplantation. We describe the first case of HTLV-1 transmission from a seropositive donor via lung transplantation.

A 38-year-old woman had abnormal shadows on a chest x-ray at a healthcare examination. Video-assisted thoracoscopic surgical lung biopsy was performed, and usual interstitial pneumonia was diagnosed. Despite some drug treatments, she had dry cough and dyspnea that gradually worsened. In 2003, at the age of 42 years, she was considered eligible for bilateral living-donor lobar lung transplantation at Okayama University. The patient was negative for anti-HTLV-I antibody. The younger sister was positive less than 16 times for anti-HTLV-I antibody. Because the patient's condition was severe and deteriorating, she was considered to be in urgent need of lung transplantation. In addition, there are no reports of HTLV-I transmission by lung transplantation. Therefore, lung transplantation was performed using lungs from an elder brother and a younger sister in August 2003. Before the transplant, the lungs were flushed with 1 L of Euro-Collins solution, which consists of 115 mmol/L monopotassium, 10 mmol/L sodium, and 3.6% glucose, both antegradely and retrogradely.[5]

The patient's general condition improved after the lung transplantation. In November 2008, her oxygen saturation was 98% to 99% at room air. However, serologic follow-up demonstrated that she had acquired HTLV-I infection posttransplantation. Antibody titers were as follows: 256 times after 9 months, more than 8192 times in August 2006, 2048 times in April 2008.

Remesar and colleagues[1] reported the first case of HTLV-1 infection transmitted by organ transplantation (a child who received a kidney from her infected mother). The seroconversion was observed 83 days after transplantation. Nakatsuji and colleagues[2] described a kidney transplant recipient who presented with tropical spastic paraparesis 4 years posttransplantation. Toro and colleagues[3] reported the transmission of HTLV-1 from a single donor to 3 organ (2 kidneys and 1 liver) transplant recipients, who subsequently developed tropical spastic paraparesis within 2 years posttransplant.

Although the lungs were pretreated with preservative solution, transmission of HTLV-1 could not be prevented. Approximately 5 years posttransplant, our patient demonstrates neither neurologic nor hematologic evidence of HTLV-I–related disease. However, this patient should be followed up for adult T-cell leukemia/lymphoma and tropical spastic paraparesis.

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TABLE 1. Review of the literature about transmission of human T-lymphotropic virus type I after solid-organ transplantation

First author	Year	Age and sex	Organ	Clinical features	References
Remesar	2000	Daughter	Kidney	None	1
Nakatsuji	2000	50 M	Kidney	HTLV-1–associated myelopathy	2
Toro	2003	53 F*	Kidney	Subacute myelopathy	3
Toro	2003	55 M†	Kidney	Subacute myelopathy	3
Toro	2003	44 F‡	Liver	Subacute myelopathy	3
Zarranz	2003	54 F*	Kidney	Myelopathy	4
Zarranz	2003	57 M†	Kidney	Myelopathy	4
Zarranz	2003	44 F‡	Liver	Myelopathy	4
Yara	2008	42 F	Lung	None	Present report

HTLV, Human T-lymphotropic virus type I. *, †, ‡The same patients.

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DOES HYPERCHOLESTEROLEMIA IMPROVE OPERATIVE OUTCOMES IN PATIENTS WITHOUT CORONARY ARTERY DISEASE WHO UNDERGO CARDIAC SURGERY?

To the Editor:

Tabata and colleagues[1] found that preoperative use of statins was associated with lower operative mortality in patients without coronary artery disease who underwent cardiac valve surgery and suggested that the pleiotropic effects of statins may be an important biological mechanism underlying this beneficial effect.

Although the authors did not include preoperative serum cholesterol levels in the analysis because they were not measured in all patients, they speculated that a beneficial effect of a decreased serum cholesterol might not be ruled out.[1] However, the study suggests that higher baseline cholesterol levels might actually improve operative outcomes in patients without coronary artery disease who undergo cardiac surgery. In fact, baseline total cholesterol levels of statin-treated populations are higher than those of the general population.[2]

Critical care literature provides strong evidence for a survival advantage associated with higher lipid levels in critically ill surgical patients.[3] Furthermore, in surgical patients, hypocholesterolemia, beyond being a strong predictor of in-hospital death, also predicts nosocomial infections and length of hospital stay.[4] In particular, it has been shown that critically low cholesterol concentrations in patients after cardiothoracic surgery are associated with a significantly increased risk for mortal outcome.[5]

Therefore, we suggest that in individuals studied by Tabata and colleagues,[1] statin use selected the healthy statin user or unselected the unhealthy patients with low cholesterol.

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Reply to the Editor:

We appreciate the comments from Mascitelli and colleagues. We have shown that preoperative statin use is associated with lower operative mortality in cardiac surgical patients without coronary artery disease.[1] As they pointed out and as we described in the original article,[1] serum cholesterol level could be a potentially confounding factor of the association between preoperative statin use and operative outcomes. However, the effect of baseline hypocholesterolemia on operative outcomes in patients having cardiac surgery remains unknown. The previous studies, including ones that Mascitelli and colleagues referenced, did not reveal an effect. In another study, Horwich and colleagues[2] have shown that lower serum cholesterol level at the time of admission is associated with increased mortality in patients with heart failure, and that patients with lower serum cholesterol levels were more likely to be on lipid-lowering medication. Although the study population of this study is not patients having surgery, their findings indicate that serum cholesterol level may be a negative confounding factor in our analysis, which would not affect our conclusion. Further investigation is necessary to determine the effect of preoperative statins on operative outcomes in cardiac surgery.

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