12-1987

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Recommended Citation
Maeda, Koichi; Bricker, Leslie; Ma, Chan K.; and Deegan, Michael J. (1987) "T-cell Lymphoma in Renal Transplant Recipient," Henry Ford Hospital Medical Journal : Vol. 35 : No. 4 , 256-258. Available at: https://scholarlycommons.henryford.com/hfhmedjournal/vol35/iss4/18

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T-cell Lymphoma in Renal Transplant Recipient

Koichi Maeda, MD,* Leslie Bricker, MD,† Chan K. Ma, MD,* and Michael J. Deegan, MD*

A 35-year-old woman, who had a renal transplant five years ago, developed malignant lymphoma of the mediastinum. The lymphoma was of lymphoblastic type and had T-cell immunophenotype. Most transplant-related lymphomas are of B-cell type. T-cell lymphoma in this setting is extremely rare, and the mechanism of development may be different from that of B-cell lymphomas. (Henry Ford Hosp Med J 1987;35:256-8)

An increased incidence of malignant lymphomas has been seen in renal transplant recipients (1-3). This phenomenon is partially explained by the immunosuppression of these patients and their predisposition to neoplasm (4). The malignant lymphomas occurring among the immunosuppressed individuals are usually of B-cell type. Histopathologically, the majority are either large cell type or undifferentiated lymphomas. Recently, we encountered a case of T-cell lymphoma that had developed in a renal transplant recipient. T-cell lymphoma is extremely rare in this setting and is the basis for this report.

Case Report

A 35-year-old white woman had a renal transplant due to end-stage chronic renal failure (chronic glomerulonephritis) in 1981 and received subsequent immunosuppressant therapy with azathioprine for five years. Cyclosporin was not used. In April 1986, she complained of chills, fever, and cough, and was found to have widening of the mediastinum. Subsequent mediastinal biopsy showed lymphoblastic lymphoma. She was discharged once, but was readmitted on May 12, 1986, after developing increasing shortness of breath, chest pain, and weakness.

Physical examination at admission revealed a thin woman in moderate distress with a temperature of 36.5°C, pulse of 150 beats/min, respiration of 32 breaths/min, and blood pressure of 110/80 mm Hg. Her skin showed scars from the previous graft. Chest examination revealed decreased breath sounds over the lower lung fields. Heart examination revealed normal S1 and S2 with increased rate. Other pertinent findings included 4 + pitting edema to the thighs bilaterally, and no hepatosplenomegaly or superficial lymphadenopathy. The neurologic examination was normal. Results of laboratory studies included WBC count of 19,500/µL, hemoglobin of 13.4 g/dL, platelet count of 214,000/µL, and differential count of 41% neutrophils, 35% bands, 6% lymphocytes, 17% monocytes, and 1% myelocytes. Serum chemistries included sodium of 131 mEq/L, potassium of 4.9 mEq/L, chloride of 93 mEq/L, CO₂ of 31 mEq/L, BUN of 21 mg/dL, creatinine of 1.3 mg/dL, glucose of 103 mg/dL, total lactic dehydrogenase of 714 IU/L, SGOT of 258 IU/L, glutamate-pyruvate transaminase of 86 IU/L, total alkaline phosphatase of 120 IU/L, total cholesterol of 113 mg/dL, triglyceride of 111 mg/dL, total bilirubin of 4.0 mg/dL, corrected calcium of 11.3 mg/dL, and total protein of 5.3 g/dL. Prothrombin time was 20.5 seconds with a control of 11.5 seconds, and activated partial thromboplastin time was 36 seconds. Urinalysis revealed clear, yellow urine with specific gravity of 1.02, pH of 5.0, and it was negative for albumin, glucose, ketone, blood, and bilirubin. Human immunodeficiency virus-antibody test and serum immunoglobulin levels were not done. Pleural fluid had WBCs of 7,800/µL with predominantly neutrophils. The culture revealed numerous Staphylococcus aureus. The patient was also considered to have chronic active hepatitis with hepatitis B antigenemia. Bone marrow was negative for lymphoma.

A left chest tube placement was done on May 12, 1986, with subsequent drainage of 1,300 mL of purulent fluid. Throughout the remainder of her hospitalization, the patient continued to experience shortness of breath with decreasing neurologic status responsive only to painful stimuli. A diagnosis of septic shock was considered on May 16, with blood pressure changes occurring rapidly due to a poor control of peripheral circulation. Chest x-rays showed her continuing worsened condition and revealed pneumonia. She was treated by antibiotics, but died on May 17, 1986.

Autopsy

An autopsy limited to the chest only was granted. There was no superficial lymphadenopathy. Lymphoblastic lymphoma was noted in the anterior mediastinum. The tumor, which measured 20 x 13.5 x 13 cm, involved the pericardium, bilateral pleura.

Accepted for publication: October 9, 1987.
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Fig 1—Lymph node biopsy, mediastinum. Sheets of lymphoblasts with frequent mitoses (arrows) (hematoxylin-eosin stain, X128).

and bilateral main stem bronchi. The heart was normal in size, but was compressed by the large tumor mass. Diffuse interstitial pneumonia and patchy bronchopneumonia were present in both lungs. Bilateral fibrinopulent pleuritis was also present. Mild to moderate arteriosclerotic coronary artery disease was observed. Bone marrow was negative for tumor. Superior vena cava was negative for tumor infiltration, but possibly it was partially compressed by the large mediastinal tumor.

**Surgical Specimen**

Biopsy of the mediastinal mass showed sheets of lymphoblasts with frequent mitoses (Fig 1). A small number of scattered phagocytic histiocytes were seen. No nodular pattern was recognized. Imprints were stained for acid phosphatase activity, and the golgi areas showed cap-like positivity. The terminal deoxyribonucleotidyl transferase test was negative, but technical difficulty was encountered during the test.

Electron microscopy examination showed sheets of lymphoblasts. Nucleoli were seen, but were not prominent. The cytoplasm showed scattered mitochondria and polyribosomes and occasionally prominent golgi apparatus (Fig 2).

Cell surface markers were studied by fluoroctyometric and immunoperoxidase study. The fluoroctyometric analysis was as follows: T11 (CD2-pan T) 92%, T4 (CD4-helper) 91%, T8 (CD8-suppressor) 94%, polyvalent for B-lymphocyte 4%, Kappa light chain 3%, and lambda light chain 4%. Frozen sections of immunoperoxidase staining additionally confirmed the T-cell nature of this tumor. The majority of cells was strongly positive for the T-cell antigens, T6 (CD1), T9, and T10. These results showed a typical profile of a common thymocyte in which both T4 and T8 antigens were expressed.

**Comment**

Malignant lymphomas occurring among renal transplant recipients are usually B-cell lymphomas (5). Histopathologically, they are intermediate or high grade and have an appearance of large cell immunoblastic type or large cell noncleaved type (6). A small number of patients with Hodgkin’s disease have been reported (7). T-cell lymphoma is extremely rare among the renal transplant-related lymphomas, and we were able to find only one case reported in the literature (8). The Epstein-Barr virus infection may play an important role in the development of post-transplantation B-cell lymphomas (6,8-13). It is postulated that the Epstein-Barr virus-infected B-cells proliferate due to T-cell immunosuppression and result in monoclonal and polyclonal lymphomas.

Rare cases of T-cell lymphoproliferative disorders associated with immunodeficiency or immunosuppressed state have been reported (8,14,15). Harper et al (14) described concomitant infection with HTLV-I and HTLV-III in a 65-year-old black man with T8 lymphoproliferative disease. In this unique case, it is not possible to conclude a causative relationship of lymphoproliferative disease due to immunodeficient state. It is conceivable, however, that prior infection with HTLV-III and immune
deficient states predisposed this patient to subsequent infection of HTLV-I and development of lymphoma.

Knowles (15) reported three individuals with the acquired immunodeficiency syndrome (AIDS) and chronic lymphoid leukemia derived from the T-lymphocyte lineage. The T-cell chronic lymphoid leukemias appeared to be of low virulence, and the patients have not suffered the progressive downhill course characteristic of AIDS-related lymphomas which are usually B-cell type.

These findings suggest that the development of lymphomas in transplant recipients and immunosuppressed individuals may be due to more than one mechanism. Different mechanisms may account for the development of T-cell lymphomas in immunosuppressed individuals in contrast to those involved in B-cell lymphoma development in the same setting.

References