Table 1 Review of T cell lymphomas affecting the heart after organ transplantation

Case	Sex	Age at Dx (years)	Transplant organ	Immunosuppressive Tx	Time between Tx and Dx	Survival after Dx	T cell lymphoma type	Location	Viral association	Reference
1	M	11	Bone marrow	MTX/prednisone	21 months	6 months	WF: lymphoblastic	Pleural effusion, myocardium	EBV+ve HTLV+ve	Zutter <i>et al</i> ^a
2	F	30	Kidney	CIT	3 years	17 months	ALCL T cell type	Multiorgan infiltration	EBV+ve HTLV-ND	Coyne <i>et al</i> ^b
3	M	30	Kidney	Cy-A, Aza/prednisone	5 years	Few days	T cell	Pleural effusion	ND	Canoz <i>et al</i> ^c
Index case	M	23	Kidney	CIT	7 years	Dx at autopsy	Peripheral extranodal T cell unspecified	Multiorgan infiltration with mass lesion only in heart	EBV+ve HTLV-ND	—

ALCL, anaplastic large cell lymphoma; Aza, azathioprine; CIT, conventional immunosuppressive treatment (azathioprine and/or cyclophosphamide combined with prednisone); CyA, cyclosporin A; Dx, diagnosis; EBV, Epstein-Barr virus; F, female; HTLV, human T cell lymphocyte virus; M, male; MTX, methotrexate; ND, not done; Tx, treatment; WF, Working Formulation classification; +ve, negative; +ve, positive.

that polyclonality is seen in a lower percentage.³ Only one case in the literature shows human T cell lymphocyte virus-1 genome.⁴

Our case of PTLT is unusual for several reasons: its primary pericardial involvement, T cell phenotype, pattern of spread and the late onset. The literature search revealed that the involvement of heart and pericardium was seen in only three cases in the previously recorded T cell PTLTs (table 1).⁵⁻⁷ The index case is a 30-year man who developed T cell PTLT 7 years after renal transplantation. This is in accordance with T cell PTLT, which usually is of late onset. The reasons for bicytopenia in the index case are anaemia of a chronic disorder, chemotherapy, megaloblastosis and haemophagocytosis. Kaplan *et al*⁸ have described haemophagocytosis associated with post-transplantation T cell lymphoma arising in the vulva. The direct cause-and-effect relationship of haemophagocytosis and T cell lymphoma is unclear. T cell PTLT has a poor prognosis when it is monoclonal and/or possesses multiorgan involvement, as seen in the index case.

The mode of spread of lymphoma cells to various visceral organs and the brain is interesting in our case. This seems to be vascular in nature—that is, through the systemic circulation.

This case exemplifies that post-transplantation lymphoma can develop in various unusual sites with protean clinical manifestations. In a post-transplant patient who presents, a long time after transplantation, with serous cavity effusions or pericarditis, the possibility of lymphoproliferative disease apart from the opportunistic infections should be considered and investigated, so that the immunosuppression can be reduced or the patient treated with specific therapy such as interferon α or monoclonal antibodies.

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A novel rapid and sensitive BRCA1/2 mutation detection method based on pooled DGGE and targeted sequencing

In 2005, we published a paper in the *Journal of Clinical Pathology* describing a novel BRCA2

mutation in an Indonesian family detected using a novel rapid and sensitive BRCA1/2 mutation detection method based on pooled denaturing gradient gel electrophoresis and targeted sequencing.¹ In this paper, we described the method used in detail as there was, at that time, only a published abstract from the Groningen group to refer to as the original source.² However, we did not realise that a contract signed by one of us some years earlier prohibited us from describing this method in detail before the developers of this technique had published it themselves. In the meantime, this has been realised.³ It was, therefore, unjustified for tables 1–4 to enclose such detail in our paper, for which we apologise to the Groningen group.

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