

Post-Transplant Lymphoproliferative Disorder (PTLD) of the Liver following an Allogeneic Renal Transplant

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Title - Post-Transplant Lymphoproliferative Disorder (PTLD) of the Liver following an Allogeneic Renal Transplant

Case Report

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Consent statement-

Written informed consent for publication of their clinical details and/or clinical images was obtained from the patient.

Abstract-

A 20-year-old man, a case of chronic kidney disease had had a renal transplant done in 2014, and presented with loss of weight and appetite since seven-eight months. Abdominal ultrasound showed two liver lesions with internal vascularity. CECT abdomen showed the same liver lesions with progressive contrast enhancement. MRI was done for further evaluation and showed enhancing lesions in segments VI and VIII with peripherally restricted diffusion and central necrotic component. Ultrasound-guided biopsy was performed and histopathology showed a high-grade Non-Hodgkin's lymphoma.

Keywords- Post-Transplant Lymphoproliferative Disorder, PTLD, renal transplant

Key clinical message- PTLD represents a variety of conditions with prognosis depending on the grade of lymphoid proliferation. Knowledge of the distribution and radiologic features of PTLD allows the radiologist to play a pivotal role in making an early diagnosis, in guiding biopsy, and in the surveillance of treatment response in patients with PTLD.

Case presentation :

A 20-year-old man, a case of chronic kidney disease with a renal transplant done in 2014 presented with loss of weight and appetite since seven-eight months. On examination, he was vitally stable and soft, non-tender abdomen. There was mild hepatomegaly on palpation. The patient was referred to our institution with a provisional diagnosis of liver abscesses with unresolving symptoms on treatment with antibiotics. On ultrasound abdomen (Figure 1), there were 10x6cm and 5x6cm sized two heterogeneously hypoechoic lesions in segment VIII and segment VI respectively. Both the lesions showed internal vascularity with RI=0.60.

CECT abdomen showed a well-defined 8.7x5.2x6cm (APxTRxCC) sized hypodense lesion in segment VIII (Figure 2) of the liver with progressive heterogenous contrast enhancement. Another well-defined 5.2x4.2x5.3cm (APxTRxCC) sized hypodense lesion in segment VI (Figure 3) of the liver with heterogenous progressive peripheral nodular contrast enhancement. The central part of the lesion was non-enhancing. Both kidneys were atrophic.

MRI of the abdomen was done for further characterization of the lesion. It showed two well-defined T2 hyperintense (Figure 4) heterogeneously enhancing lesions. They measured 9x5.5x6cm (APxTRxCC) in segment VIII and 4.8x5.2x5.6cm (APxTRxCC) in segment VI. There was diffusion restriction on DWI with a corresponding drop on ADC (Figure 5). The central necrotic component did not show enhancement (Figure 6) or diffusion restriction. The imaging diagnosis was made of post-transplant lymphoproliferative disorder (PTLD).

An ultrasound-guided biopsy of the larger lesion was done. Histopathological report showed a high-grade Non-Hodgkin's Lymphoma.

Discussion :

Post-transplant lymphoproliferative disorders (PTLD) is an umbrella term that involves lymphoid hyperplasia to lymphoid neoplasia. It occurs in the setting of immunosuppression after transplantation. The majority of PTLDs involve B cell proliferation and related Epstein-Barr virus (EBV) infection because the EBV-infected B cells proliferate when the T cells are depleted due to therapeutic immunosuppression [1,2]. In 1968, Starzl described PTLD in renal transplant recipients [3]. The prevalence differs with different organ allografts, with the highest prevalence recorded in multi-visceral transplant recipients (13%–33% of cases),

followed by bowel (7%–11%), heart-lung (9.4%), lung (1.8%–7.9%), heart (3.4%), liver (2.2%), and kidney (1%) recipients [4].

Since clinical features are non-specific. Imaging findings may help in identifying the lesions and guide biopsy for diagnosis. CT is the most commonly used modality because of its availability. MRI is also used depending upon the site and nature of the lesion. FDG-PET is specifically useful in the persistent lesions, and response evaluation to the therapy as it can help differentiate between residual tumor and fibrosis or necrosis [5].

The abdominal cavity is the compartment most frequently involved by PTLD. Extra-nodal involvement (80% of cases) is more common than nodal involvement (20%) in intraabdominal disease [6]. Hollow viscera involvement is similar to those of non-Hodgkin's lymphoma, including thickening and aneurysmal dilatation. The liver is the most frequently involved abdominal solid organ. The most common lesions are multiple hypodense nodules on the CT with variable peripheral enhancement. The kidney is the most commonly involved site in renal transplant recipients [7]. A pulmonary mass and nodules either solitary or multiple, are the most common thoracic manifestation of PTLD. The CT and MR imaging features of CNS PTLD closely resemble those of HIV-related lymphoma. But hemorrhages and necrosis are more common [8].

Classic treatment is the reduction of immunosuppression particularly in polyclonal PTLD [9]. Chemotherapy is the mainstay of treatment in monoclonal lesions and in lesions that are unresponsive to immunosuppression reduction.

Conclusion:

PTLD represents a variety of conditions with prognosis depending on the grade of lymphoid proliferation. Knowledge of the distribution and radiologic features of PTLD allows the radiologist to play a pivotal role in making an early diagnosis, in guiding biopsy, and in the surveillance of treatment response in patients with PTLD.

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Figures :

Figure 1-Ultrasonogram of liver:

A. Intercoastal axial view of the liver shows an approximately 10x6cm sized well-defined heterogeneously hypoechoic lesion in segment VIII of the liver.

B. Colour and Spectral Doppler of the same lesion show mild internal vascularity with RI=0.60.

Figure 2: Axial sections of the plain (A), arterial (B) and portovenous (C) phases of the CT abdomen show a well-defined 8.7x5.2x6cm (APxTRxCC) sized hypodense lesion in the segment VIII of the liver that shows progressive heterogenous contrast enhancement. There is no calcification. Figure 3: Axial sections of the plain (A), arterial (B) and portovenous (C) phases of the CT abdomen show another well-defined 5.2x4.2x5.3cm (APxTRxCC) sized hypodense lesion in the segment VI of the liver that shows heterogenous progressive peripheral nodular contrast enhancement. Central area of the lesion is non-enhancing. There is no calcification. Both the kidneys are atrophic. Figure 4: T2 weighted axial sections of the MRI abdomen show two well-defined heterogeneously hyperintense lesions in the segment VIII (A) and VI (B) of the liver. The lesion in the segment VIII measures 9x5.5x6cm (APxTRxCC) and the lesion in the segment VI measures 4.8x5.2x5.6cm (APxTRxCC). Figure 5: The lesions in the segment VIII (A) and VI (B) show diffusion restriction on DWI with a corresponding drop on ADC. The central area of the lesions does not show diffusion restriction. Figure 6: On contrast enhanced MRI of the abdomen, the lesion in segment VIII shows heterogenous enhancement (A), the lesion in segment VI shows peripheral nodular enhancement with central non-enhancing area (B).