

# Society of Radiologists in Ultrasound 2012 Toshiba Residents Program

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## 1. History

69-year-old male status post kidney transplant one month prior for ESRD and now with elevated LFTs.

## 2. Figures

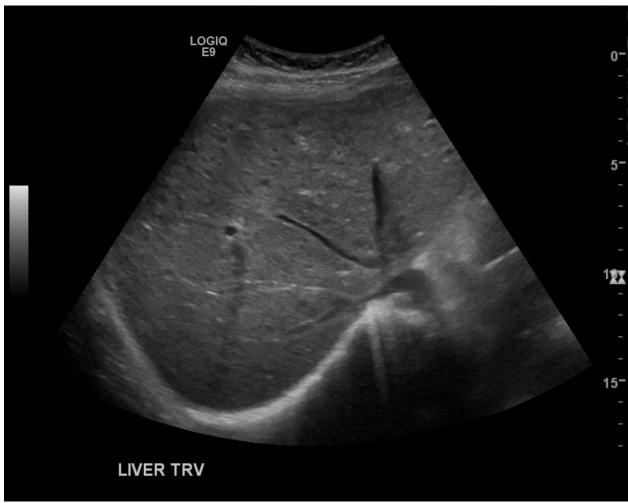


Figure 1 – Transverse sonographic image of liver at level of hepatic veins with heterogeneous echotexture and multiple small hypoechoic masses. Smooth liver contours. No biliary dilatation.



Figure 2 – Sagittal sonographic image of the gallbladder and liver. Similar liver findings as in figure 1. Minimal gallbladder wall thickening and cholelithiasis.

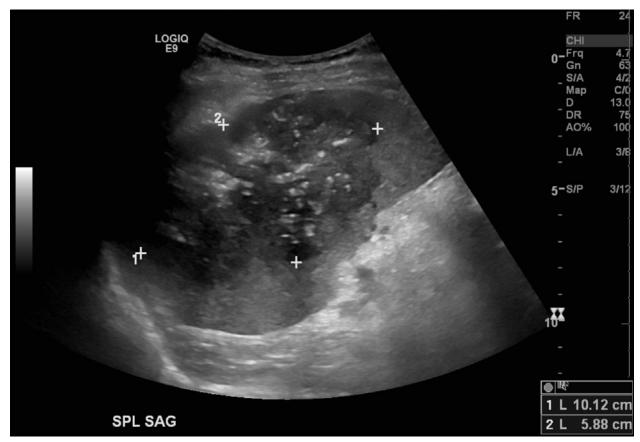


Figure 3 – Sagittal sonographic image of the spleen. Large lobulated 10x6x8 cm complex mass in the spleen with scattered calcification.

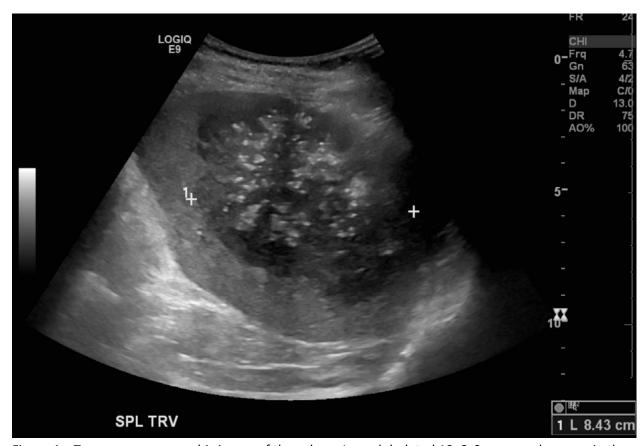


Figure 4 – Transverse sonographic image of the spleen. Large lobulated 10x6x8 cm complex mass in the spleen with scattered calcification.

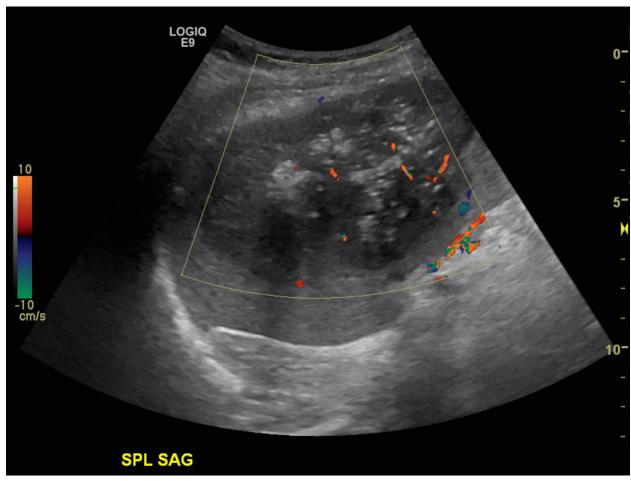


Figure 5 – Sonographic color Doppler image of the spleen. Complex splenic mass with internal blood flow.

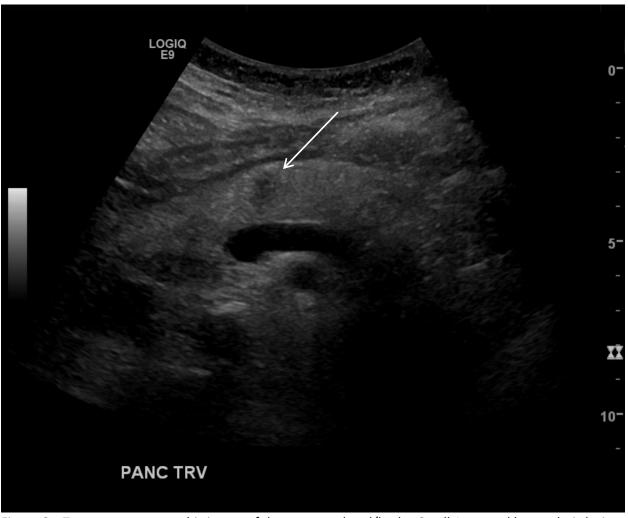


Figure 6 – Transverse sonographic image of the pancreas head/body. Small 1 cm oval hypoechoic lesion in the pancreatic body (arrow). No significant blood flow on color Doppler (not shown).



Figure 7 – Contrast enhanced axial CT in portal-venous phase at the level of the pancreas. Heterogeneous, hypodense, lobulated splenic mass with heterogeneous enhancement and tiny calcifications correlate with ultrasound findings. Hypodense infiltrating mass with irregular borders at the pancreatic tail measuring up to 3.1 x 2.5 cm. This lesion is not clearly visualized on ultrasound. The 1 cm hypoechoic mass at the pancreatic head is at a lower axial slice (not shown). The multiple small hypoechoic masses in the liver are better visualized on the ultrasound.



Figure 8 – Coronal reconstructed slice of the upper abdomen again noting the large complex splenic mass.

#### **Diagnosis**

Transjugular liver biopsy show high grade diffuse large B cell lymphoma.

#### **Discussion**

Hodgkin disease is classically differentiated from non-Hodgkin lymphoma (NHL) by its propensity for involving axial lymph nodes. Peripheral nodal involvement has never been seen and mesenteric nodal involvement is rare. It classically involves a single nodal group and has contiguous nodal extension. Extranodal masses are also rare in Hodgkin disease. Non-Hodgkin lymphoma covers the remaining lymphomas and includes more than 30 subtypes (1). NHL incidence has increased 75% in the past 20 years with increasing mortality. Median age for NHL is 55 with greater than 90% occurring in adults (2).

Diffuse large B-cell lymphoma is the most common NHL subtype in adults accounting for 31% of cases. It is also one of the most aggressive with 60% of patients having disseminated disease at diagnosis (2). Extranodal lymphoma is more common in NHL vs. Hodgkin disease (20-40% vs. 4-5%), which is defined by disease in sites other than lymph nodes, thymus, tonsils and pharyngeal lymphatic ring (caveat: spleen is considered nodal in Hodgkin disease and extranodal in NHL) (3). Extranodal involvement is also more common in recurrent disease and immunodeficiency-related disease including posttransplantation lymphoproliferative disorder (PTLD) (4).

PTLD occurs most frequently within the first year following transplantation. Majority of PTLD B-lymphocyte proliferation are thought to be related to Epstein-Barr virus (EBV) infections, although the exact pathogenesis is unknown. There is data to suggest increased risk of PTLD in patients receiving tacrolimus after a kidney transplant which our patient received. However, PTLD in kidney recipients is rare accounting for approximately 1%. Highest prevalence of PTLD is in multivisceral transplant recipients followed by bowel, heartlung and liver recipients. Probability of PTLD also increases with more aggressive immunosuppressive induction therapy particularly in bowel, lung and heart transplant recipients (5).

Anatomic distribution of PTLD highly correlates with the transplanted organ. With kidney transplant, allograft involvement is reported in 10-75% of patients. The variability is thought to be related to varying populations and immunosuppression protocols. Interestingly our patient's transplanted kidney, which he received from his daughter, has a normal appearance on CT and ultrasound. Otherwise, liver and spleen are the most frequently involved abdominal solid organ in PTLD as well as NHL (5).

Splenic involvement usually has diffuse infiltration or focal nodules. Nodules in the spleen are characteristically hypoechoic on ultrasound. There is usually no posterior acoustic enhancement. On CT they are usually hypoattenuating with decreased enhancement when compared to normal splenic tissue. Calcifications are usually a result of prior treatment and are therefore unusual in our patient's case who has not received any treatment. Diffuse infiltrative involvement of the spleen without nodules is difficult to detect and absence of splenomegaly does not rule out tumor infiltration (3).

Hepatic involvement has a similar appearance and pattern involvement to that of the spleen. It is usually difficult to differentiate it from metastatic disease and fungal abscesses. Pathologic sampling is always required for diagnosis (4). In our patient a transjugular liver biopsy is performed.

Pancreatic involvement is rare in PTLD and has only been described in pancreatic allografts. However, the pancreas is involved in up to 30% of NHL cases. Patterns are similar and include well circumscribed mass and diffuse glandular enlargement which may mimic acute pancreatitis. Biliary and pancreatic duct obstruction is usually rare. Vascular invasion, tumor calcification and necrosis are unusual at presentation of lymphoma and may help differentiate it from adenocarcinoma (4).

Lymph node involvement in PTLD is less likely than in NHL. Otherwise, its appearance is indistinguishable from other lymphoproliferative processes (5). Our patient did not have significant lymphadenopathy in the chest or abdomen.

Unfortunately, our patient died four days after the ultrasound from sepsis and multi-organ failure. The large complex splenic mass with calcifications is atypical in appearance for an initial presentation of PTLD or NHL. The hypoechoic masses in the liver and pancreas are usually non-specific, but in a post-transplant patient PTLD should be considered as it is the most likely case in our patient. Biopsy is always required for diagnosis of PTLD. Recognizing its appearance and pattern of involvement will aid in early diagnosis and treatment.

#### References

- 1. Leite N et al. Cross-sectional Imaging of Extranodal Involvement in Abdominopelvic Lymphoproliferative Malignancies. *RadioGraphics* November 2007 27, 1613-1634.
- 2. Blodgett T, Ryan A, Almusa O. StatDx Premier: Non-Hodgkin Lymphoma. Available at: https://my.statdx.com/STATdxMain.jsp?rc=false#dxContent;non\_hodgkin\_lymphoma\_dx
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- 4. Lee W. Abdominal Manifestations of Extranodal Lymphoma: Spectrum of Imaging Findings. AJR July 2008 191, 198-206
- 5. Borhani A et al. Imaging of Posttransplantation Lymphoproliferative Disorder after Solid Organ Transplantation. *RadioGraphics* July 2009 29, 981-1000.

### <u>Authors</u>

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#### Institution

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