

# Primary Renal Lymphoma- A Case Report

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## Abstract

Primary renal Non-Hodgkin's lymphoma is rare. Renal involvement though common in disseminated Non-Hodgkin's lymphoma (NHL), the incidence of extra nodal primary renal non-Hodgkin's lymphoma is less than 1%. An elderly, immunocompetant, chronic alcoholic male symptomatic since one month was presented with acute abdominal pain with fever and acute renal failure. The provisional diagnosis both clinically and radiologically was liver abscesses with enlarged bright kidneys. On the complete autopsy examination, diagnosis of primary renal lymphoma with liver metastasis was made. Also an incidental finding of microfilariae of *Wuchereria bancrofti* was noted.

## Introduction

Renal involvement though common in disseminated non-Hodgkin's lymphoma (NHL), the incidence of extra nodal primary renal Non-Hodgkin's lymphoma is less than 1% . Primary renal Non- Hodgkin's lymphoma is rare, because the renal parenchyma does not have lymphatics. Patients with bilateral renal involvement of the renal lymphoma tended to have poorer survival. We report a rare case of primary renal lymphoma with liver metastasis presented as acute renal failure and liver dysfunction. Also an incidental finding of microfilariae of *Wuchereria bancrofti* was noted.

## Case Report

A 65 years old male, immunocompetant, chronic alcoholic was admitted in medicine ward with jaundice, abdominal distension, fever and oliguria. There was no past history of drug intake or chronic liver disease. There were no significant findings on general and systemic examination except icterus and hepatomegaly. The haemoglobin was 11.2 gm%, BUN-140 mg%, S. creatinine – 7.2 mg%, alkaline

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phosphatase- 1605 IU/L, SGOT/SGPT- 509/311 mg%, Total bilirubin- 11 mg%.

Ultrasonography of abdomen was reported as hepatomegaly with multiple hypoechoic lesions suggestive of abscesses or metastasis with enlarged bright kidneys.

Clinical diagnosis of alcoholic liver disease with viral hepatitis with acute renal failure was made. Another possibility of liver metastasis with hepatic encephalopathy was kept and advised CT scan.

Patient died on the same day of admission with respiratory failure as terminal event.

A complete autopsy was performed. Icterus was present. On gross examination enlarged liver (3.2 kg) with multiple well defined white, soft to firm lesions seen varying in size from 1 x 1 cm to 5 x 5cm with prominent central umbilication (necrosis) noted. Surrounding liver parenchyma showed cholestasis and venous congestion changes (nut-meg liver).

Both the kidneys were enlarged (600 grams), bosselated and showed diffuse enlargement of cortex and medulla which is white firm suggestive of tumour infiltration (Fig. 1).

A thorough examination was done to find the enlarged lymph nodes in the body. However, no lymph nodes were noted and there were no nodules seen in any of the parenchymal organs like spleen, lungs, brain, stomach and intestines.

Liver and kidney lesion's scrapes were taken for cytological examination. Cytology and

histopathological examination of kidney sections (Fig. 2) and liver sections was suggestive Non-Hodgkin's lymphoma, extranodal type which was confirmed on Immunohistochemistry to be Diffuse large B cell type. There was a unusual finding of microfilariae of *Wuchereria bancrofti* seen in peripheral smear and in the scrapes taken from liver lesions (Fig. 3).

Considering the pattern of tumour infiltration and extensive review of literature the diagnosis of Extranodal- primary renal lymphoma with liver metastasis was made.

### Discussion

Primary renal Non-Hodgkin's Lymphoma (NHL) is defined as a Non-Hodgkin's Lymphoma arising in the renal parenchyma and no invasion from an adjacent

lymphomatous mass.<sup>1</sup>

Renal NHL is rare and ante mortem diagnosis of primary renal lymphoma is difficult.

Renal involvement with NHL is more commonly found in the setting of disseminated disease. Primary renal NHL could be more common than previously recognized because they are usually intermediate and high-grade B-cell NHL that metastasize rapidly.<sup>2</sup> This is supported by Kandel *et al* in their review of primary renal lymphoma, who showed that all 17 autopsy cases of presumed primary renal lymphoma had evidence of extra renal lymphomatous disease.<sup>2</sup>

Because the normal kidney does not have lymphoid tissue, how can primary renal lymphoma be explained? Freeman *et al* 19 in their review of 1467 cases of extranodal NHL hypothesized "the usual precursor of extranodal lymphoma is a specific pathologic proliferative response of lymphoid tissue at the site." Puente Duanay postulated that a pre-existing inflammatory processes recruit lymphoid cells into the renal parenchyma, and while there, "the untimely oncogenic event takes place."<sup>2</sup> Others postulate that because the renal capsule is rich in lymphatics, the



Fig. 1 :Gross section of the enlarged kidney showing diffuse tumour infiltration.

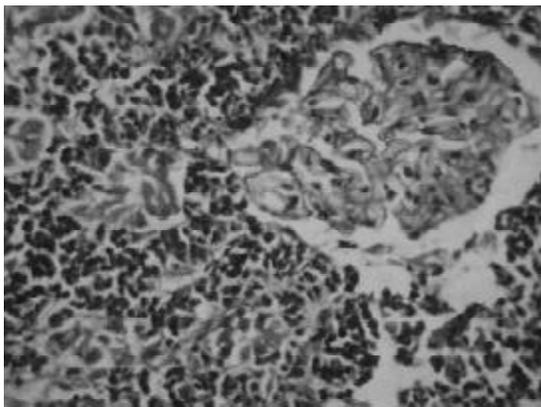


Fig. 2 :Microscopy of kidney section shows extensive tumour infiltrate.

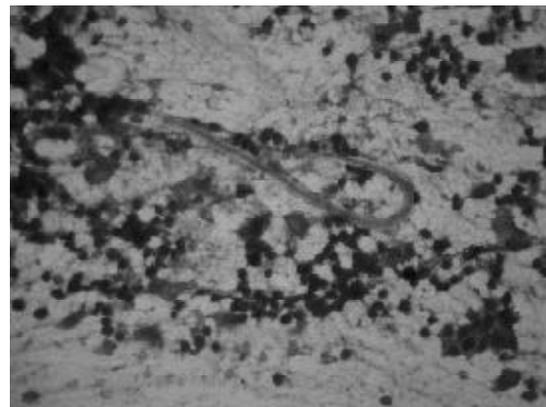


Fig. 3 :Microfilariae of *Wuchereria bancrofti* seen in the scrapes taken from liver lesions.

tumour is able to penetrate the renal parenchyma. The presenting symptom in all patients was flank pain and normal laboratory data, except an elevated serum creatinine, were not helpful in the diagnosis of primary renal NHL.<sup>2</sup>

Tumour proliferation begins in the interstitium and the underlying nephrons; however, collecting systems and blood vessels provide a framework for tumour growth.<sup>3</sup> Infiltrative growth results in preservation of the parenchymal structures and renal contour; consequently, detection is often difficult, and renal involvement can be easily missed. As lymphomatous tumour enlarges, the surrounding renal parenchyma is compressed and destroyed, and continuous tumour infiltration results in the formation of expansible renal masses.<sup>4</sup> Non uniform growth can result in single or conglomerate masses that extend beyond the renal contour and displace the collecting system.

Most primary renal lymphomas disseminate rapidly from their renal origin, and mean survival is reportedly less than a year after the diagnosis. Although the standard management of a renal mass is nephrectomy, Non-Hodgkin's lymphoma should be considered in the differential diagnosis.

The disease may present with progressive renal failure of either the oliguric or non-oliguric type. In primary renal lymphoma, survival is extremely poor: 75% of patients die in less than 1 year.<sup>3</sup> The prognosis may be improved by early detection of the disease and by performing systemic chemotherapy.<sup>4</sup> With appropriate treatment, renal lesions may completely regress, often with minimal scarring within the renal parenchyma.<sup>4</sup>

In our case, there was liver involvement. Liver in systemic lymphomas is involved in approximately 50% of all cases. Involvement of the Lymphoma of liver is usually

secondary. Liver involvement is common in disseminated Non-Hodgkin's lymphoma.<sup>5</sup>

Acute Liver failure (ALF) as a presenting feature of a haematolymphoid malignancy is extremely rare and therefore not usually considered in the differential diagnosis. This occurs when the hepatic parenchyma is massively replaced by tumour cells with diffuse intrasinusoidal propagation.<sup>5</sup> This infiltration can result in hepatic ischaemia due to massive sinusoidal infiltration or tumour cell replacement of the hepatic parenchyma. Establishing this diagnosis can be very difficult and requires a high index of suspicion. Given the poor prognosis, it should be considered in any patient presenting with ALF especially if associated with lactic acidosis, hepatomegaly and without an obvious aetiology. Liver biopsy is crucial to the process of early diagnosis and initiating chemotherapy, which may be lifesaving.

In our case, we found microfilariae of *Wuchereria bancrofti*, as an incidental finding. Gupta *et al* reported six cases where microfilariae were found in body fluid cytology and fine needle aspiration smears in association with tubercular pleural effusion, Non-Hodgkin's lymphoma, malnutrition and pregnancy.<sup>6</sup>

Although the finding of microfilariae in cytology smears is considered incidental, the association of microfilariae with debilitating conditions suggest that it is an opportunistic infection.<sup>6</sup>

### **Conclusion**

Our case is unique because a rare entity of Extranodal primary renal lymphoma presenting as acute renal failure and acute liver failure. With awareness and high index of suspicion, Primary Renal Lymphoma, though rare should be included in the differential diagnosis of renal masses

presenting with acute renal failure.

#### References

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#### TUBERCULOSIS DRUG RESISTANCE COMES FULL CIRCLE

In today's *Lancet*, Anne von Gottberg and colleagues present a report that could bring us full circle, suggesting that fluoroquinolone treatment of multidrug-resistant (MDR) tuberculosis causes pyogenic bacterial resistance. The reverse has long been thought to be true.

The study, from South Africa, found levofloxacin resistance in children with MDR tuberculosis treated with a fluoroquinolone and in children with nosocomially acquired resistant pneumococci.

WHO also reported the existence of extensively drug-resistant (XDR) tuberculosis in no fewer than 45 countries; although this is certainly an underestimate because few countries have laboratory capability to detect it.

Drug resistance, including MDR and XDR forms, is essentially a man-made problem.

WHO's Green light committee has stopped recommending ciprofloxacin to treat drug-resistant tuberculosis. The difference between MDR and XDR tuberculosis is additional resistance to fluoroquinolones and an injectable drug in the latter.

This has changed with fluoroquinolones, which are widely used to treat pyogenic infections, and besides being highly potent drugs for MDR disease are being studied as first-line treatment for tuberculosis.

Nosocomial spread occurs in both directions.

The impending adoption of moxifloxacin as a first-line drug for tuberculosis has even more serious ramifications.

If we continue along this path we will have lost one of our leading drugs for the treatment of tuberculosis and perhaps, soon, an important treatment for community-acquired pneumonia.

**Lee B Reichman, *The Lancet*, 2008; 371 : 1052-53.**