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Case Blog
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Diffuse Large B cell Lymphoma of Liver in the Setting of Prior HCV

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Case Blog

40 year gentleman with a past medical history significant for hepatitis C infection and T2DM presented to his primary care physician on 1/30/2013 He was complaining of fevers for 10 days. He returned 9 days later with continued fevers and sore throat and treated with azithromycin for a bacterial pharyngitis. Other symptoms at this time included, significant nighttime sweats, and loss of appetite. He was noted to have marked submandibular lymphadenopathy.

On 02/18/2013, the patient returned with the same symptoms but a worsening of the night sweats and anorexia. Additionally, he was noted to have a weight loss of 8 pounds since his previous visit. Blood work was drawn at this time. He began developing abdominal pain on 02/25 at which time was repeated revealing an ESR=101 and CRP=85.1. On examination, he was found to have moderate right flank tenderness. Anuitrasound was ordered but pain became progressively worse and patient went to the ER. At this time, he rated the flank pain as 8/10. He was found to have a temperature of 100.9. An abdominal ultrasound was performed revealing innumerable varying sized hepatic hypodensities with some peripheral enhancement throughout the liver. The largest lesion was found in segment IIb and measured 2.3 cm. Many enlarged lymph nodes were also identified in the upper, central abdomen as well as mildly enlarged retroperitoneal nodes. Labs at this time showed an elevated total protein of 9.3, Na+ of 134. Other labs were normal. An MRI was then ordered, which showed new masses throughout the liver, with the largest lesion being in the left lobe.

His pain continued to progress and he underwent a liver biopsy. A fine needle and core biopsy of the liver revealed diffuse large B cell lymphoma, germinal center B cell subtype. Morphology revealed atypical lymphoid infiltrate composed of medium to large cells. Immunohistochemical stains showed that the infiltrate was positive for CD20, CD10, BCL6 and CD3, CD5, BCL2, MUM1, cyclin D1, CD30, c-MYC. Proliferation index by ki67 was more than 90%. Patient was referred to an oncologist and underwent chemotherapy. Patient remains cancer free as of this date.

Hepatitis C is well-established as a causative factor of liver cirrhosis and hepatocellular carcinoma. The link between HCV and non-Hodgkin's lymphoma has not been so well determined. Morphologically, NHL related to HCV includes varying subtypes, of which large B cell lymphoma is one.

Interestingly, the association between HCV and NHL has been found to be higher in some countries (Italy, US, Japan) but not in other countries. However, some of these studies were limited because they did not use an appropriate control group or control for confounding variables. One study performed in Italy used a multi-center case controlled study to remove these limitations. The study included patients in 10 different hospitals across Italy. The results of this study showed that patients with NHL were 3.1 times more likely to be infected with HCV than the control group. The genotype of HCV did not appear to make any difference. Approximately 2/3 of these patients were men, consistent with previously known data. This study affirmed that there is indeed an association between HCV and NHL.

There are some studies that seem to suggest that the association with HCV involves only indolent forms of NHL. The association between HCV and the development of large B cell NHL has been demonstrated in many countries. The fact that anti-viral medication seems to playa curative role in B cell NHL in patients with HCV tends to suggest that the virus plays some part in the proliferation of B cells. The exact mechanism is unknown. Cumulative evidence shows an antigen driven process in lymphoma development. Further evidence seems to demonstrate that the link between HCV and more aggressive NHL

is also associated with a translocation of BCL2. Lymphomas in the setting of HCV appear to have a specific molecular signature. This fact may be key in developing treatments.

Across the globe, approximately 180 million people, or about 3% of the world's population is infected with HCV. There have been several proposed theories as to the association between HCV and B-NHL. One is antigen drive as noted above. Another suggests that the virus exerts a direct effect on hematopoietic cells. The third theory is referred to as "hit and run", where a transforming event is required. The fact that so many are infected with HCV makes prevention imperative. Evidence seems to suggest that treatment of HCV prevents further progression to lymphoma.

Reviewing studies from a decade or more in the past, the association between HCV and lymphoma was controversial. We now have a multitude of evidence showing a clear association between the 2 and suspected mechanisms of causality. However, there remain gaps in our knowledge base regarding this association and the body of evidence is not so clear cut as the association between HCV and hepatocellular carcinoma. More research is needed to determine the precise pathway HCV follows to induce NHL. Once that is known, targeted treatments can be developed. Additionally, since anti-viral medications seem to play some curative role in treating the NHL, more research needs to be conducted on the eradication of this virus as this may also prevent NHL.

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