

Lymphoma Risk Increased with HCV



Alan Franciscus, Editor-in-Chief

The designation “Non-Hodgkin’s Lymphomas” (NHL) encompasses a variety of cancers of white blood cells that affect the lymphoid tissues. The exact cause of these cancers is not fully understood but it is believed to be caused by an altered or depressed immune system. Other conditions and medications that have been linked to NHL include HIV infection, immunosuppressive medications, rheumatic diseases and hepatitis C.

The lymphatic system is a circulatory system that collects white blood cells which are taken from veins, circulated throughout the body, and returned to the bloodstream. Once the lymphatic fluid is returned to the blood supply, the kidneys are responsible for removing the waste products. Lymphatic organs include the spleen, tonsils, appendix, and thymus. Moreover lymphatic glands are also found in patches located in the intestines. The lymphatic system carries white blood cells that help fight infection.

Typically, lymphoma occurs when white blood cells divide continuously without pause, which prevents them from maturing. This process can cause an overproduction of the immature cells which can crowd out the mature white blood cells, platelets and red blood cells.

It is not fully understood how HCV causes NHL. There are theories that the virus might be the causing agent, or that the constant immune system stimulation from hepatitis C causes NHL. However, we do know that the incidence of NHL in people with hepatitis C is higher than in the general population.

A recent study from the Veteran’s Administration will hopefully shed more light on the prevalence of this condition and other types of cancer in the hepatitis C population. The authors looked at the incidence of NHL, Waldenstrom macroglobulinemia, cryoglobulinemia, thyroid cancer

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and other types of malignancies. A retrospective study (examining information already available) by T.P. Giordano and colleagues was conducted that examined the incidence of various forms of cancer in veterans with hepatitis C compared to veterans without hepatitis C.

The study involved patients from U.S. Veterans Affairs health care facilities from 1997-2004. Medical records were analyzed from 146,394 HCV infected veterans and 572,293 non-HCV infected veterans. The mean or average age was 52 years old (± 8), and 97% were males. It was found that veterans infected with hepatitis C had a higher incidence of NHL (number = 1,359), Waldenstrom macroglobulinemia (number = 165) and cryoglobulinemia (number = 551) than veterans **without** hepatitis C. There was no

Incidence in HCV infected Veteran Population

Non-Hodgkin’s lymphoma	1,359 out of 146,394 patients (0.928317%)
Waldenstrom macroglobulinemia	165 out of 146,394 patients (0.1127095%)
Cyroglobulinemia	551 out of 146,394 patients (0.3763815%)

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Extrahepatic Manifestations: *Essential Cryoglobulinemic Vasculitis*



Alan Franciscus, Editor-in-Chief

Vasculitis refers to a group of conditions that are characterized by inflammation of the wall of blood vessels that include the veins, arteries and capillaries. *Essential mixed cryoglobulinemia* is a blood disorder that is caused by abnormal proteins in the blood called cryoglobulins that precipitate or clump together when blood is chilled and then dissolve when re-warmed. These proteins can be deposited in small and medium-sized blood vessels, which can lead to restricted blood flow to joints, muscles, and organs. This article will focus on the type of vasculitis that is associated with HCV-related essential mixed cryoglobulinemia. It should be noted that cryoglobulinemic vasculitis is **uncommon or rare** in people with hepatitis C. In addition, the prevalence of vasculitis varies based on geographic location – the condition is more common in Southern Europe than in Northern Europe or Northern America.

Vasculitis is sometimes referred to as a ‘hurting disease’ because it is commonly associated with pain. Vasculitis can affect almost every organ in the body. The more common symptoms and conditions produced by vasculitis include:

- **Skin** – rashes characterized by purplish red spots that are usually found on the legs
- **Joints**—joint aches, and ar-

thritis that includes swelling

- **Lungs**—Shortness of breath, cough, and lung infiltrates
- **Kidneys**—clumps of red blood cells in the kidneys, and loss of protein through the urine
- **Gastrointestinal tract**—abdominal pain, and bloody diarrhea
- **Blood**—anemia and/or elevated white blood cell counts
- **Sinuses and nose**—chronic sinus congestion and infections, hearing problems, and inflammation of nasal tissues
- **Eyes**—damage to the blood vessels of the eye
- **Brain**—headaches, difficulty with coordination, changes in mental status and strokes (rare)
- **Nerves**—shooting pains in the arms and legs as well as numbness and weakness

Other symptoms include fever, itching welts, fatigue, weight loss, muscle ache and pain, enlarged lymph nodes, and peripheral neuropathy.

There are no standardized diagnostic tests or criteria for vasculitis. Vasculitis is usually diagnosed by various lab tests that include testing for cryoglobulins, skin biopsy, electromyography (detecting electrical signals in muscle cells), arteriography (pictures of blood vessels), and by

the clinical manifestations or the symptoms listed above.

Treatment of vasculitis is multi-faceted approach depending on the severity or disease progression. The treatment for HCV-related vasculitis usually starts with treating the underlying cause (HCV) with interferon and ribavirin therapy. Unfortunately, interferon plus ribavirin therapy does not usually result in long term resolution of vasculitis and could worsen current or underlying peripheral neuropathy – a condition that is associated with vasculitis. Additional strategies to treat vasculitis include the use of steroids or immunosuppressant drugs, and plasmapheresis (the removal, treatment and return of blood plasma).

In summary, essential cryoglobulinemic vasculitis is a rare condition in people infected with hepatitis C. It is a difficult condition to diagnose because there are no established diagnostic criteria, and current treatment strategies have had limited success. Fortunately, there is much research into the cause and treatment of vasculitis as well as new drugs to treat hepatitis C and a hepatitis C vaccine that would protect people from acquiring HCV-related extrahepatic manifestations such as vasculitis.



HealthWise:

Men's Health and HCV



Lucinda K. Porter, RN

This year, National Men's Health Week is June 11 - 17. Whether you are a man or a woman, this is an opportunity to increase your awareness about men's health. Although women have their own health problems, men also have to deal with potentially serious medical problems. Male life expectancy is shorter than that of females. Women outlive men by about 5 years. The average U.S. male born in 1950 may expect to live until age 79.¹

The leading causes of death for men are heart disease, cancer, accidents, and stroke. Suicide is the 8th leading cause. Liver disease is 10th on the list. Although the majority of those with chronic hepatitis C virus (HCV) are unlikely to die from it, roughly 8,000 - 12,000 people will die from HCV-related causes. Men outnumber women with this disease and are more likely to die from it.

A majority of veterans are men. In 1999, the Veterans Administration tested the veteran population and found that 6 to 8% of the participants tested positive for the HCV antibody. In Northern California, this leaps to a rate of over 17%. HCV in veterans is frequently complicated by other health problems, such as alcoholism and depression.

More than six million men suffer from depression. Since men are less likely than women to seek help for this illness, some wonder if the number is actually much higher. Minority and elderly men are particularly susceptible to depression.

Men tend to be less aware of the symptoms of depression and diagnosing it can be tricky. Men describe vague symptoms, such as having sleep or concentration problems, being easily irritated or feeling lousy. They may not report feeling sad, blue, down or depressed. They are less willing to seek help for illness in general and even less so for psychiatric ones. Yet

of those who do get help for depression, 90% report significant relief.

Sexual dysfunction is a problem for many. More men with HCV than those without it reported sexual dysfunction. This includes problems with sex drive, ejaculation, erectile dysfunction (ED) and sexual satisfaction. Thirty million men in the U.S. report ED. This is not just a sexual problem - it is also an emotional and physical one. A number of factors may cause ED, such as certain drugs and diseases. ED is associated with prostate problems, high blood pressure, high cholesterol and diabetes. Smoking, obesity, heavy drinking, stress, depression, lack of exercise, and sleep problems may contribute to ED.

Men are exposed to health risks through their occupations. More men than women work in construction, farming, mining, factories and the lumber and aviation industries. They work with power tools, drive taxis and trucks, work on highways, etc. This exposes them to greater risks of accidental death.

Men are more likely to die as the result of violence. The military is predominantly male. Paramedics, fire fighters and law enforcement officers tend to be men. There are more men in prisons and jails than there are women. Men are at increased risk for chemical dependence. Naturally, prostate cancer is restricted to males. Hemophilia, a clotting disease that is almost exclusive to men, also carries a high risk for HCV.

Although death is inevitable, an early death is not. We each have the power to take care of ourselves.

"If you are an average 57-year old American man reading this article, your life expectancy is roughly another 22 years."

Here are a few suggestions:

- Get a life - engage in activities that give you pleasure.
- If you don't have a doctor, find one and get regular health check-ups.

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Steatosis and Liver Cancer



Alan Franciscus, Editor-in-Chief

A simple definition of steatosis is “fatty liver” or “fatty infiltrates in the liver.” Steatosis is a common condition found in the general population that is most likely caused by a high fat diet and lack of exercise. Steatosis is more common in the hepatitis C population compared to the general population. The reason for the higher prevalence in the hepatitis C population is unknown but there is a theory that while the hepatitis C virus does not necessarily cause steatosis it may make it easier for steatosis to form.

Infection with hepatitis C can lead to liver cancer, but the period of time that it usually takes for people to develop liver cancer is 20, 30 years or longer. However, it is important to know that the vast majority of people with hepatitis C will never develop liver cancer.

It has been suggested that the combination of steatosis and HCV has a synergism – that is the combination of the two conditions produces an effect that neither condition could produce alone or results in one condition that is greater than the total effects of each condition operating by itself. This would suggest that having hepatitis C and steatosis would accelerate HCV disease progression.

The evidence on the role of steatosis on HCV disease progression has been conflicting with some data showing a faster disease progression while other data has shown that steatosis has had little or no effect on disease progression in someone with hepatitis C.

A recently published study by

J.R. Pekow and colleagues titled “Hepatic steatosis is associated with increased frequency of hepatocellular carcinoma in patients with hepatitis C-related cirrhosis” is giving us a better picture of the relationship between steatosis, cirrhosis, and hepatocellular carcinoma (liver cancer).

In this retrospective study, 94 consecutive patients with HCV cirrhosis who underwent liver transplantation from 1992 to 2005 were identified. Data on age, race, sex, HCV genotype, presence of HCC (pathology records from the transplanted liver), and stage of fibrosis was collected. All pathology records were reanalyzed by a single, blinded pathologist who evaluated the tissue for steatosis. The following grading system was used:

- Grade 0 = absent
- Grade 1 = 1-5% of hepatocytes (liver cells) affected
- Grade 2 = 6-32% of hepatocytes affected
- Grade 3 = 33-66% of hepatocytes affected
- Grade 4 = greater than 66% of hepatocytes affected.

The majority of patients were men (78%), white (80%) and infected with genotype 1 (57%).

Four patients had received HCV therapy before liver transplantation, of whom 2 achieved an SVR (1 in each group), and two were nonresponders (1 in each group). Thirty-two patients had evidence of HCC.

In total, 53 (56%) of patients in the analysis had evidence of

steatosis. Nineteen percent of patients had grade 2 steatosis or higher. The authors pointed out that the percentage of patients with steatosis was consistent with previous studies. However, the authors found that the percentage of patients with grade 2 steatosis or higher was lower than in previous studies, but could be explained by the inclusion of noncirrhotic patients in those studies.

The authors found that there was a significant association between increasing steatosis grade ($P = .005$), higher ALT (alanine aminotransferase) ($P = .002$), higher alpha-fetoprotein (a marker for liver cancer) ($P < .001$), lower HCV viral load ($P = .02$), higher biologic MELD score ($P = .03$) and the risk for liver cancer. In multivariate analysis (a collection of procedures involving analysis and observation), age, AFP and steatosis were significantly associated with liver cancer.

This study is important because it is the first study that examines the association between steatosis and liver cancer in patients with HCV-related cirrhosis.

The authors concluded that “[i]n patients with HCV-related cirrhosis, the presence of hepatic steatosis is independently associated with the development of hepatocellular carcinoma.” The authors also noted that since steatosis poses an additional risk for liver cancer in people with hepatitis C, steatosis should be monitored closely and strategies should be developed to reduce steatosis, which will lower the risk for developing liver cancer.

Reference

Pekow JR, Hepatitis steatosis is associated with increased frequency of hepatocellular carcinoma in patients with hepatitis C-related cirrhosis. *Cancer*, May 8 [epub ahead of print].



Management of HIV/HCV Coinfection: *New Guidelines*

■■■
Liz Highleyman

In the May 31, 2007 issue of *AIDS*, an international panel of experts presented updated guidelines for the management of HIV/HCV coinfection. The revised recommendations reflect an improved understanding of how the two infections interact and the challenges of concurrent therapy since the previous guidelines were issued in 2004.

It is estimated that one-third of HIV positive people also have HCV. Coinfected patients tend to experience more rapid liver disease progression, especially if they have advanced HIV disease and low CD4 cell counts. Since the advent of potent combination antiretroviral therapy (HAART), liver disease has become a leading cause of death in people with HIV.

The updated recommendations incorporate new data from recent trials of interferon-based therapy for coinfecting individuals. While HIV positive patients generally do not respond as well to pegylated interferon (Pegasys or PegIntron) plus ribavirin as people with HCV alone, these studies shed new light on ways to optimize hepatitis C treatment in this population.

The revised guidelines cover 11 areas:

- Management of patients with persistently normal aminotransferase (ALT) levels;
- Assessment of liver fibrosis, including new noninvasive tests;
- Predictors of response to anti-HCV therapy;

- Optimal doses of pegylated interferon and ribavirin;
- Optimal duration of interferon-based therapy;
- Treatment of coinfecting nonresponders and relapsers;
- Care of coinfecting patients with end-stage liver disease;
- Treatment of acute hepatitis C in HIV positive individuals;
- Management of patients with multiple hepatitis viruses;
- Interactions between anti-HCV medications and antiretroviral drugs;
- Hepatotoxicity of antiretroviral drugs.

PERSISTENTLY NORMAL ALT

The panel noted that about 7%-9% of coinfecting patients have persistently normal ALT, compared with about 25% of HCV mono-infected individuals. Further, research suggests that 25%-40% of coinfecting patients with normal ALT have significant liver fibrosis or cirrhosis, compared with 10%-30% of HCV mono-infected patients. Given that the prevalence of fibrosis is higher and liver disease progression is faster in coinfecting individuals, the panel recommended that these patients should be considered for hepatitis C treatment regardless of ALT level.

LIVER FIBROSIS ASSESSMENT

The panel reviewed noninvasive methods for assessing fibrosis

using various serum biomarkers and imaging techniques. While these newer methods are generally good at diagnosing absent or mild fibrosis versus severe fibrosis or cirrhosis, they are less able to distinguish between intermediate stages. Due to the inflammatory nature of HIV disease and the effect of antiretroviral drugs on biomarker levels, serum tests may be less reliable in coinfecting patients, though elastometry (FibroScan) seems quite accurate. Given the improved rate of response to anti-HCV therapy, the faster rate of fibrosis progression in HIV positive patients, and the ability to assess early virological response to therapy, the panel stated that in most cases "liver biopsy is not mandatory for considering the treatment of chronic HCV infection," adding that a combination of noninvasive methods "accurately predicts hepatic fibrosis in most cases."

PREDICTING RESPONSE TO THERAPY

The panel also reviewed the various factors that predict response to interferon-based therapy. Many of these are the same in coinfecting and HCV mono-infected individuals (younger age, female, Caucasian, HCV viral load, genotype 2 or 3, lower body mass index, minimal liver fibrosis or steatosis), but there are some additional factors associated with poor

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COINFECTION

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response in coinfecting patients, including lower CD4 cell count and use of certain antiretroviral drugs. Insulin resistance is also a concern, given its association with protease inhibitors.

Good adherence is especially crucial for coinfecting patients, who must receive adequate doses of pegylated interferon and ribavirin for a sufficient length of time. Studies have shown that rapid virological response (RVR) at Week 4 predicts eventual sustained virological response (SVR) in coinfecting patients – as it does in those with HCV alone – but coinfecting patients appear less likely to achieve RVR, possibly due to higher baseline HCV RNA levels or slower response to therapy. Patients who do not achieve early virological response (at least a 2-log reduction in HCV RNA by Week 12) are unlikely to achieve SVR and should stop treatment early to avoid further side effects and cost.

OPTIMAL DOSE AND DURATION

Coinfection treatment trials have shown that weight-based ribavirin (1000 mg/day if < 75 kg and 1200 mg/day if > 75 kg) is superior to an 800 mg fixed dose. Adequate ribavirin dosing seems especially important for coinfecting patients, since response to interferon may be compromised by immune deficiency. Though some studies have tested higher doses of pegylated interferon, the benefits have not been established in coinfecting patients, and the panel recommended the standard dose. If necessary, blood cell growth factors should be used to enable full dosing of both pegylated interferon

and ribavirin.

While studies have shown that treatment duration may be safely shortened for HCV mono-infected individuals who achieve RVR, this strategy has not been adequately studied in coinfecting patients. The panel recommended 48 weeks of combination therapy for coinfecting patients with all genotypes, but allowed that 24 weeks may be adequate for those with genotypes 2 or 3. Although data are not yet conclusive, they suggested that slow-responding coinfecting patients with genotypes 1 or 4 (those who achieve EVR but not RVR) “might benefit from extended (60-72 weeks) courses of therapy.”

NONRESPONDERS AND RELAPERS

To date, retreatment of coinfecting nonresponders and relapsers has not been adequately studied. Even among patients who do not achieve sustained response, however, research suggests that interferon maintenance therapy may help slow liver disease progression. Based on available data, the panel stated that “[n]onresponders and relapsers to prior courses of HCV therapy are a heterogeneous population and therapeutic interventions in them should be individualized.”

The panel also suggested that investigational drugs – including new types of interferon and directly targeted antiviral agents such as HCV protease and polymerase inhibitors – offer the prospect of improved outcomes. “Trials exploring the efficacy and safety of these drugs in coinfecting patients should

be prioritized, without waiting for the final results of Phase III trials conducted in HCV-mono-infected,” they wrote.

ANTIRETROVIRAL THERAPY

Finally, the panel looked at interactions between anti-HIV and anti-HCV medications and hepatotoxicity of antiretroviral therapy. The major interaction concerns relate to concurrent use of ribavirin with ddI (Videx) or AZT (Retrovir). Both ddI and ribavirin can cause mitochondrial damage, while both AZT and ribavirin can cause anemia. The panel recommended that ddI and ribavirin should never be used together, and that AZT “should also be avoided when possible.”

With regard to hepatotoxicity, the panel noted that various antiretroviral drugs affect the liver in different ways. Certain nucleoside analogs (especially ddI and d4T [Zerit]) can cause mitochondrial toxicity (which can lead to liver steatosis). Nevirapine (Viramune)

can cause hypersensitivity reactions that damage the liver, and some protease inhibitors, such as full-dose ritonavir (Norvir), can

“The panel recommended 48 weeks of combination therapy for coinfecting patients with all genotypes”

directly damage the liver. Atazanavir (Reyataz) can cause elevated bilirubin, but this does not reflect liver damage. In addition, as HAART enables CD4 cell recovery, immune reconstitution can worsen liver inflammation (mainly a concern for patients with HBV).

Despite these concerns, the panel concluded, the benefits of

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COINFECTION

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antiretroviral therapy outweigh the risks, given that several studies have demonstrated lower rates of liver-related mortality in coinfecting patients taking antiretroviral therapy. Indeed, they wrote, “[s]ince severe immunosuppression accelerates HCV-related liver fibrosis progression, it may be advisable to start HAART without unnecessary delays in coinfecting patients and even consider earlier initiation of treatment.”

CONCLUSION

Due to space limitations, this summary of the new guidelines is necessarily incomplete. Coinfecting patients should talk to their doctors about the latest recommendations for treatment, monitoring, and management of symptoms and side effects.

For more information on HIV/HCV coinfection, see the Hepatitis C Support Project’s new booklet, *HIV and Hepatitis Coinfection*, by Raymond M. Johnson, MD, PhD, available in versions for both patients (http://www.hcvadvocate.org/co_web2.pdf) and clinicians (http://www.hcvadvocate.org/co_web.pdf).

Reference

V. Soriano, M. Puoti, M. Sulkowski, et al. Care of patients coinfecting with HIV and hepatitis C virus: 2007 updated recommendations from the HCV-HIV International Panel. *AIDS* 21(9): 1073-1089. May 31, 2007.



MEN’S HEALTH

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- Get an annual flu shot and make sure all vaccinations are up to date. If you have HCV, be immunized against hepatitis A and B
- Strive to be as physically active as you can be on a regular basis.
- Maintain a healthy weight.
- Eat a low fat, high fiber diet. Include fruit, vegetables, and whole grains. Avoid trans-fatty acids and saturated fats.
- Avoid alcohol, tobacco and recreational drugs. If you cannot quit, try to cut back or get some help.
- If you are not finding much pleasure in your life, ask your doctor to screen for depression.
- Learn how to avoid, reduce or manage stress.
- Practice safer sex.
- Maintain friendships and social contacts.
- If you are facing retirement or are already there, take on meaningful activities, such as volunteer work. Get involved with life.

Life expectancy calculators can be frightening but motivating. They can help detect those areas that need improvement and may extend our lives. There are many life expectancy calculators on the Internet. I use them as motivational tools. For example, if I keep my weight in a certain range, I may extend my life by three years. Therefore, when I look at a sugary dessert, I ask myself if the indulgence is worth three years of my

life – then I pass on the calories. A calculator I like is *moneycentral.msn.com/investor/calcs/n_expect/main.asp*

If you are an average 57-year-old American man reading this article, your life expectancy is roughly another 22 years. Does that make you gulp? The good news is that you can do something about this. Don’t sit there and take it like a man. Get off the couch and get moving. Now, what are you going to do with the rest of your one and only life?

Reference

¹Centers for Disease Control National Vital Statistics Reports, Vol. 54, No. 14, April 19, 2006

Resources

- *Younger Next Year: A Guide to Living Like 50 Until You are Eighty and Beyond*, by Chris Crowley and Henry S. Lodge
- www.cdc.gov/men
- *A Guide to Healthy Living with HCV* – www.hcvadvocate.org/hepatitis/factsheets_pdf/healthy_Living.pdf

Men’s Health

- www.menshealthweek.org
- www.menshealthnetwork.org

National Institutes of Health

- www.nimh.nih.gov/publicat/publiclisting.cfm?dID=48
- www.nlm.nih.gov/medlineplus/menshealthissues.html



Alabama Organ Donor Registry

The state of Alabama has done much to promote organ donor awareness. They offer two easy-to-use websites where you register your wishes. The state also offers “Recycle Yourself” license plates. You can purchase a plate for a mere \$50 fee, of which more than \$41 goes to the Alabama Organ Center’s educational program. Just think – you can turn your gas-guzzling car into a vehicle for promoting organ and tissue donation awareness.

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Alabama Organ Center *www.alabamaorgancenter.org*

Legacy Organ and Tissue Donor Registry *www.legacyalabama.org*

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LYMPHOMA RISK

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increase in the number of thyroid cancers in veterans with hepatitis C when compared to veterans without hepatitis C.

The authors concluded that “hepatitis C virus infection confers a 20% to 30% increased risk of non-Hodgkin’s lymphoma overall, and a 3-fold higher risk of Waldenstrom macroglobulinemia,” and that “although the clinical significance of these findings is unknown, it is possible that screening of individuals infected with HCV could identify early stage lymphoproliferative conditions suitable for early intervention strategies, including chemoprevention trials on premalignant disease.”

It is important to know that, even though there is a 20% to 30% increased risk of NHL and a 3-fold higher risk of WM, these conditions are uncommon. Putting a bit of perspective on the actual percentage of cases in HCV infected veterans gives us a better picture of the incidence of these conditions in the hepatitis C population (*see table on page 1*).

WHAT IS WALDENSTROM MACROGLOBULINEMIA?

Waldenstrom macroglobulinemia (WM) is a chronic low-grade type of cancer of the lymph cells. WM is considered a rare or uncommon condition in people with hepatitis C. The prevalence of WM in the hepatitis C population is unknown, but it has been found to be higher than in the general population.

WM occurs when the body produces large amounts of an abnormal protein (macroglobulin) that causes plasma cells to multiply out

of control and invade bone marrow, lymph nodes, and the spleen. WM is more common in men than women and among whites than blacks and is more often seen in people over the age of 65.

Symptoms of WM include swelling of the lymph nodes, liver or spleen, weakness, muscle numbness or tingling, loss of appetite, low grade or mild fever, abnormal bleeding, fatigue, headaches, problems with vision, kidney problems, infections, dizziness, and/or confusion.

Treatment of WM depends on the progression of the disease. The most common treatment consists of chemotherapy. Plasmapheresis (the removal, treatment and return of blood plasma), interferon therapy, and monoclonal antibody (Rituxan) have also been used to treat WM, but with varying degrees of success. There are also many drugs in clinical development to treat and manage WM.

References:

T.P. Giordano et al, Risk of Non-Hodgkin Lymphoma and Lymphoproliferative Precursor Diseases in US Veterans with Hepatitis C Virus, JAMA, May 9, 2007 – Vol 297, No. 18

Extrahepatic Manifestations: Non-Hodgkin’s Lymphoma (NHL), by Alan Franciscus - http://www.hcvadvocate.org/hepatitis/factsheets_pdf/NHL.pdf

For more information about Extrahepatic Manifestations of HCV check out our Factsheet page <http://www.hcvadvocate.org/hepatitis/factsheets.asp>

For more information about WM visit the American Cancer Society’s Web site at www.cancer.org



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