Yakima Health District BULLETIN

Volume 12, Issue 2 June, 2013

Hepatitis C Update: Clinical Evaluation and Prevention Messages

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Background

The March 2013 edition of the *YHD Bulletin* addressed surveillance, epidemiology and testing for chronic viral hepatitis C (HCV) infection. This issue complements that discussion with summary recommendations for evaluation and management of HCV-infected patients and a review of what is known about the prevention of HCV transmission. Due to space limitations, the promised hepatitis B article is deferred to a future issue of the *Bulletin*.

Clinical Manifestations

Although acute hepatitis C virus (HCV) infection is usually asymptomatic, it leads to chronic infection in about 80% of cases. Approximately 25% of chronically infected individuals will progress to cirrhosis over the ensuing two or three decades. Within another decade, 15% of these will develop hepatocelluar carcinoma (HCC) and 30% will progress to liver failure. Meanwhile, the precirrhotic experience is far from a benign waiting game. Chronically HCV-infected patients commonly experience fatigue, arthralgias, and depression. Furthermore, HCV infection has now also been associated with hepatic steatosis, diabetes, renal disease, lymphoma, and other extrahepatic manifestations (e.g., cryoglobulinemia, porphyria cutanea tarda).

Laboratory

Detection of anti-HCV antibodies by enzyme immunoassay (EIA) approaches 95% sensitivity after a 60-day window and 100% after 180 days. Confirmation of infection is by HCV RNA testing, which will be positive in about 80% of EIA-positive patients. When used for detection of acute infection, HCV RNA can be detected as early as 2–3 weeks after infection. EIA-positive, RNA-negative patients generally need no further testing or follow-up for HCV.

Initial Evaluation and/or Referral after Detection of HCV

Initial evaluation after detection of chronic HCV infection typically addresses the elements in Table 1 (see page 2).This is best done by or in consultation with an expert in HCV management.

Treatment Options

The decision to initiate therapy is a function of the patient's clinical and histopathologic status, clinician judgment, and the patent's willingness and ability to proceed. This medical decision-making should be done by or in consultation with an expert in HCV management. In general, the motivation to treat is proportional to the stage of progression of disease, up to the point of decompensated cirrhosis (when pharmacotherapy is generally not pursued outside of experimental protocols). Also, many (if not most) treatment centers require patients to be clean-and-sober for 6-12 months prior to initiating therapy. This increases the probability of adherence to the regimen and appears to reduce the incidence of regimen-interrupting neuropsychiatric adverse effects.

Typical regimens for treatment-naïve patients are set forth in Table 2 (see page 2). The goal of therapy is to eradicate HCV RNA, which corresponds to reduced risk of hepatic and extra-hepatic sequelae, as well as to improve overall status. Genotypes 1a and 1b, the predominant types circulating in North America and Europe, respectively, have historically been less responsive to

standard therapy than types 2 and 3. Now, with the addition of protease inhibitors (PIs), sustained viral response (i.e., negative HCV RNA 24 weeks after completion of therapy) can be achieved in proportions comparable to those with standard therapy for types 2 and 3: 70-80%. However, adverse effects (mostly hematologic) and costs are also raised by the addition of PIs.

Details on timing and duration of the PI vary depending on history of prior treatment, response to therapy, and which PI is used. Quoted response rates assume good tolerance and adherence. In addition to genotypes 1a/1b and non-adherence to the regimen, risk factors for poor response to therapy include cirrhosis, HIV infection, genotype 4, African-American or Latino race/ethnicity, and polymorphisms in host protein IL28B.

Table 1. Typical Elements of HCV Baseline Evaluation

Element	Components
History	Alcohol and other substance use Psychiatric history and mental status exam
Laboratory	PCR for confirmation (if not already done) and determination of viral burden Liver* and renal function Complete blood count and platelets PT/INR Other viral hepatitis serologies (anti-HAV, HBsAg, anti-HBs, anti-HBc) Genotyping of HCV (1a, 1b, 2, 3, 4, 5, 6)
Direct Assessment of Liver One or more of the following depending on patient's clinical status, laboratory results, clinician judgment, and patient preference	Abdominal ultrasound (ascites, tumors) Liver biopsy (inflammation, necrosis, fibrosis, Fe, steatosis) Non-invasive tests (elastography, specialized serum tests) MELD score (need/candidacy for liver transplantation)
Immunizations	If susceptible and/or not completely vaccinated as appropriate: • Hepatitis A • Hepatitis B • Pneumococcal If asplenic, add meningococcal and Hib
Counseling	Avoid these risk factors associated with progression of liver injury: • Alcohol consumption • Obesity • Marijuana smoking Advise about what is known regarding risks of sexual transmission and relative safety for most patients Do not donate blood, semen, tissues, or organs Relatively good outlook for most patients (see Treatment) Clean-and-sober criteria typically required for treatment Address depression or ongoing chemical dependency, if present
*Note: Beware normal ALT results, particularly on a single examination. Inflammation and fibrosis can be ongoing or even quite advanced in the setting of normal or modestly abnormal transaminases.	

Table 2. Typical Regimens for Treatment Naïve Patients

Type (prevalence)	Regimen	Sustained Viral Response (%)
1a, 1b (75%)	Ribavirin + pegylated interferon + protease inhibitor (boceprevir or telaprevir) x 24-48 weeks	65-83%
2,3 (20%)	Ribavirin + pegylated interferon x 24 weeks	70-80%
4,5,6	Ribavirin + pegylated interferon x 24 weeks	50-70%

Up to 80 percent of treated patients experience adverse effects, including but not limited to: cytopenias, integumentary changes (e.g., rash, hair loss), neuropsychiatric disturbance (e.g., fatigue, irritability, depression), thryoid dysfunction, and autoimmune disorders.

The arrival of protease inhibitors for HCV therapy offers an opportunity to treat patients with mid- to later-stage disease who cannot wait for future agents in the drug development pipeline. Future hopes center around compounds targeting viral and host proteins (e.g., viral protease and polymerase, host cyclophilin). Furthermore, some of these agents in the drug development pipeline may offer better efficacy against previously difficult-to-treat genotypes. Consequently, some experts are recommending that relatively healthy, stable patients with early disease forego therapy at the current time and remain under observation while awaiting new regimens that are likely to be more effective and hopefully better tolerated than the current ones. Likewise, waiting for newer agents is a standard recommendation for patients with genotypes 2-6 who have failed to respond to previous therapy with ribavirin and pegylated interferon.

Prevention

Drug Injectors

Because drug injection is the dominant risk factor for transmission of HCV in our setting, any successful public health approach for its prevention must focus here. A meta-analysis of interventions aimed to reduce transmission through multiple combined strategies (e.g., syringe exchange programs, safe injection education, behavioral counseling, opiate replacement therapy) showed a statistically significant 75% reduction in seroconversion among drug injectors. In this analysis of multiple studies, single-method interventions did not show any clear trend toward effectiveness in preventing seroconversion. This finding is consistent with the diverse factors that favor HCV transmission and create an unforgiving environment for imperfect drug injection practices: a large pool of infected individuals with high titers of a virus that can persist on injection equipment for many days. Table 3 shows a case in point: seroconversion rates for HIV and HCV in Baltimore across two decades with implementation of combination interventions (syringe exchange, education-and-referral, and opiate replacement therapy). Success in interrupting HIV transmission was profound; not a single case of HIV acquisition was noted in the cohort after 1998. HCV transmission was reduced, but could not be eliminated as it was for HIV.

Table 3. Incidence On Follow-up Among IDU with Initially Negative Serology (cases/100 person years)

	1988-1989	1994-1995	1998	2005-2008
HIV	5.5	2.0	0.0	0.0
HCV[1]	22.0	17.2	17.8	7.2

[1]P=.07, X2 test for trend

The authors of this study noted that such interventions "*may reach IDUs too late in their injecting careers to have a significant impact on HCV infection incidence. Furthermore, although these measures may reduce the frequency of needle sharing that may be sufficient to impact HIV transmission, they may not completely eliminate risk behavior, making them insufficient to effectively prevent HCV transmission throughout an IDU's injection career."* Crude local estimates show a similar picture: during 2006-2011, IDU was the chief risk factor for approximately 5-10 newly reported HIV cases but at least several hundred chronic HCV cases in Yakima County.

Where does this leave us? The evidence base suggests that a diverse approach to HCV prevention among drug injectors will yield substantial but imperfect gains: clean injection equipment, safe injection practices, behavioral counseling to reduce drug use, and referrals for drug treatment or opiate replacement therapy. With the potential arrival of HCV treatment regimens that are easier to administer, adhere to, and tolerate, it is also conceivable that in the near future treatment of infected individuals could also serve as a mode of prevention, reducing risk of transmission to others.

Infected Health Care Workers

The Centers for Disease Control and Prevention offers no specific recommendations to restrict the practice of a health care worker who is infected with HCV. However, the Society for Healthcare Epidemiology of America (SHEA) recommends that "*HCV-infected providers who have circulating HCV viral burdens of greater than or equal to 10^4 GE/mL routinely use double-gloving for all invasive procedures, for all contact with mucous membranes or nonintact skin, and for all instances in patient care for which gloving is routinely recommended, and that they not perform those Category III activities identified as associated with a risk for provider-to-patient transmission of bloodborne pathogen infection*

despite the use of appropriate infection control procedures." SHEA provides a pathway for HCV-infected health care workers with lower viral burdens to engage in Category III procedures under a specified contract of responsibilities subject to the discretion of an Expert Review Panel that coordinates its oversight with an occupational health care provider, facility infection control, and a personal physician. Category III procedures include but are not limited to most general, oral, and specialty surgical procedures.

Sexual Contacts

Barrier protection is not recommended for serodiscordant, HIV-negative couples in a mutually monogamous relationship unless there is concern about disruption of mucosal or epithelial integrity. On the other hand, condoms are definitely recommended for dually HIV/HCV-infected patients, even if they are having sex with HIV-seroconcordant partners ("sero-sorting"). It is also prudent to recommend that HCV-serodiscordant couples consider the likely increase in risk of transmission that might be associated with menses, unprotected ano-genital intercourse, or disruptions in skin or mucosal integrity (e.g., recurrent genital herpes) and that they take appropriate precautions under such circumstances.

Challenges

The immediate disease control challenges associated with HCV include the large reservoir of infection, the ease with which it is spread through drug injection, and the high proportion of infected individuals who are unaware of their infection. These challenges are exacerbated by the absence of any substantial, dedicated funding for surveillance, investigation, epidemiology, and control by local and state health public health agencies for this disease that causes as many or more deaths each year than does HIV.

On a second level, HCV represents not only a legacy of the past drug injection experiences from a cohort now largely removed from drug use, but also an ongoing threat involving the current generation of active drug injectors, as well as migrants from parts of the world where health care exposures (not illicit drug injection) are the dominant mode of transmission.

Finally, HCV presents a health services delivery challenge. Identifying, diagnosing, evaluating, and (where appropriate) treating the estimated 3,200 HCV infections in Yakima County calls for health care financing, system capacity, and patient readiness that simply does not exist. While much of this is beyond our individual or even collective control locally, as health care providers we can at least try build appropriate capacity within ourselves and our care systems to recognize patients who are at risk, diagnose them, provide initial evaluation and counseling, and make referrals for further care when indicated and to the extent financial resources and patient readiness permit.

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Extended-Spectrum Beta-Lactamase Producing Infections Noted Locally

Microbiology

Initially recognized 25-30 years ago following the introduction of oxyimino cephalosporins in Europe and North America, extended-spectrum beta-lactamases (ESBLs) are plasmid-mediated enzymes conferring resistance to a broad range of beta-lactams. This causes *in vitro* and clinical resistance to penicillins, extended-spectrum cephalosporins (e.g., ceftazidime, cefotaxime, ceftriaxone) and the monbactam, aztreonam. ESBLs primarily have been associated with *Klebsiella spp.* and *Escherichia coli*, but other gram-negative organisms can also harbor such resistance (e.g., *Salmonella*, *Proteus,* other *Enterobacteriaceae, Pseudomonas*). Common sites of involvement with ESBL-producing organisms are the bloodstream, urinary tract, respiratory tract, and surgical wounds.

ESBLs are plasmid-mediated enzymes (TEM-type, SHV-type, CTX-M) that evolved by mutation and selection of beta-lactamase genes naturally present in many gram-negative bacteria. A less common but similar mechanism of ESBL activity is conferred by AmpC beta-lactamases that are either chromosomal or plasmid-mediated. ESBLs are inhibited *in vitro* by beta-lactamase inhibitors(e.g., clavulanate, sulbactam) and are typically sensitive *in vitro* to cephamycins (e.g., cefoxitin, cefotetan, cefmetazole), whereas AmpC beta-lactamases are the converse (not inhibited by beta-lactamase inhibitors and resistant *in vitro* to cephamycins). Most ESBL-harboring organisms also carry other antimicrobial resistance genes (e.g., gentamycin, ciprofloxacin).

Laboratory detection of ESBLs is a complex and changing arena of microbiologic practice. Because the enzymes are heterogeneous in their sensitivity to the various cephalosporins, susceptibility testing involves conducting assays against a panel of such agents in both the presence and absence of clavulanic acid. Organisms that demonstrate resistance to a cephalosporin but whose growth is inhibited when clavulanic acid is added are suspected to have ESBL activity. The National Clinical Laboratory Standards Institute has released new, lower minimal bacterial inhibition (MIC) cut-offs for these organisms to raise sensitivity in detection of ESBL; however, some experts have expressed concern about the impact of that policy on specificity and positive predictive value.

Clinical Outcomes and Treatment

Patients with ESBL-producing infections have higher mortality, lower clinical microbiologic cure rates, longer hospital stays, and higher costs than their counterparts with similar non-ESBL-producing infections—especially when inadequate therapy is prescribed. At this time, the sole class of agents that is proven be effective are carbapenems (e.g., imipenen, meropenem, ertapenem, doripenem). The fourth generation cephalosporin, cefipime, may be effective in some settings when given in higher doses. Despite in vitro activity of beta-lactamase inhibitors and cephamycins, clinical outcomes in patients treated with these agents are inferior to those obtained with carbapenems.

Epidemiology

In the United States, an estimated 5-10% of *Klebsiella pneumoniae* isolates and 3-4% of *E. coli* isolates show ESBL activity. Observed rates are 2-3 times higher in Latin America, Asia and Europe. Incidence of acquisition during an intensive care unit stay is estimated at approximately 0.5%. The primary risk factor for acquisition of an ESBL-producing organism is antimicrobial pressure, especially use of cephalosporins. However, it seems that prior therapy with other antimicrobials that are not an enzyme target (e.g., ciprofloxacin, trimethoprim-sulfamethoxazole, vancomycin, others) can still select for ESBL-producing organisms. Delayed or inadequate antimicrobial therapy for a previous infection has also been shown to increase risk for a subsequent ESBL-producing infection. Other leading risk factors include urinary catheterization, surgical procedures (e.g., tracheostomy, gastrostomy, jejunostomy, abdominal surgery), chronic disease, hospitalization, and long term care residence. Concern also exists about the potential for ESBLs to enter the food chain through indiscriminate use of cephalosporins in animal food production.

Spread of ESBLs in health care facilities can occur via clonal transmission of the same organism or by plasmid transfer among unrelated organisms. Infection control studies suggest that both contact precautions and restricted use of cephalosporins can reduce the prevalence of colonization in health care facilities.

Up to two-thirds of ESBL infections in some settings are now attributable to *E. coli*, and about one-third of ESBL infections are community- (not health care-) associated. This raises concern for an increase in ESBL infections in the future and is reminiscent of the transition a decade ago of methicillin-resistant *Staphylococcus aureus* (MRSA) from being primarily a health care associated infection to now an endemic community-acquired one.

Local Occurrence and Control

In Yakima County, ESBL-producing infections have been recognized in newly hospitalized patients who were recently

treated in long term care facilities or in outpatient clinics. Given that delayed, inadequate or indiscriminate antimicrobial use are demonstrated risk factors for the occurrence of such infections, prudence is warranted in evaluation and management among patients with other risk factors for ESBL: pre-treatment specimen collection for culture and sensitivities, targeted antimicrobial therapy, and early infectious diseases consultation for complicated, unresponsive, or proven ESBL-producing infections. Please note that isolation of ESBL-producing organisms from sputum and urine specimens may represent colonization rather than disease; in the absence of a compatible clinical syndrome, a positive culture on its own does not necessarily indicate the need for treatment.

Infection with an ESBL-producing organism is not a notifiable condition under Washington State law. However, YHD strongly recommends that diagnosing providers collaborate with facility infection control staff, infectious diseases specialists, and microbiology staff in the management and control of ESBL-producing organisms. In the event of a documented cluster of community-acquired ESBL infections among patients seemingly linked in some manner, YHD would want to be notified in order to facilitate an investigation of possible causes and to support control measures in collaboration with involved health care providers and facilities.

Similar to MRSA, vancomycin-resistant enterococci (VRE), and carbapenem-resistant *Enterobacteriaciae* (CRE), the wide antimicrobial resistance spectrum of ESBL-producing organisms poses a formidable challenge in humankind's ongoing battle with microorganisms. The dearth of new antimicrobials in the development pipeline calls upon us to prevent and control their further emergence as much as possible because therapeutic options will remain limited for some time to come.

Acknowledgement

YHD thanks Neil Barg, MD, for notifying us about the local occurrence of ESBL and for commenting on a draft of this article.

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Notifiable Condition *(includes confirmed and probable cases)*	Cases Jan - May 2013	Cases Jan - May 2012	Cases Jan - May 2011	Total Cases by Year 2012	Total Cases by Year 2011
Campylobacteriosis	32	21	30	108	122
Chlamydia	579	563	501	1302	1224
Cryptosporidiosis	0	1	0	5	1
Genital Herpes - Initial	19	27	35	61	74
Giardiasis	3	6	4	15	16
Gonorrhea	46	33	39	81	99
Hepatitis A acute	0	0	0	2	0
Hepatitis B acute	0	0	0	0	0
Hepatitis B chronic	*NA	*NA	3	*NA	8
Hepatitis C acute	0	2	0	2	0
Hepatitis C chronic	*NA	*NA	82	*NA	206
HIV/AIDS Cumulative Living	186	179	178	185	182
HIV/AIDS Deaths	1	5	0	6	4
HIV/AIDS New	2	1	5	9	12
Meningococcal Disease	0	0	0	2	0
Pertussis	106	104	1	493	10
Salmonellosis	10	7	11	26	18
Shigellosis	3	0	3	1	11
STEC (enterohemorrhagic E. coli)	4	2	2	7	10
Syphilis - Primary and Secondary	8	2	5	6	9
Tuberculosis	1	2	6	5	6

*NA=Not Available

Notifiable Conditions Summary Jan - May 2013

Firland Foundation

CURRY INTERNATIONAL TUBERCULOSIS CENTER

UCSF University of California San Francisco advancing health worldwide

Tacoma - Pierce County Health Department
Healthy People in Healthy Communities

Spokane Regional Health District

Public Health Prevent • Promote • Protect

Washington State Department of Health

SAVE the DATE

October 23, 2013
Washington State
Annual TB Meeting

This year's Annual TB Meeting will be held at the Tacoma/Pierce County Health Department.

This year's **Keynote Address** will be given by Dr. Michael Kimerling of the Gates Foundation.

Also presenting will be Dr Tara Wildes, Chief of Jacksonville Florida Sheriff's Office Prisons Division

You may attend at the main meeting site in Tacoma or you have the option of viewing through video conference at one of the Eastern Washington locations (Benton-Franklin or Spokane Regional Health Districts). You may also participate by iLinc.

CMEs/CEUs will be provided through the Curry International Tuberculosis Center for those participating in-person or by video conference.

We hope that you, our partners in the elimination of TB, are able to attend.

Public Health: Always Working for a Safer and Healthier Washington

October 23, 2013
WA State Annual TB Meeting

Planning Notes	**(Electronic TB Packet)**

7:30 –8:00	REGISTRATION & BREAKFAST *(Moderator: SheAnne Allen)*
8:00-8:05	**Welcome and State of the State** SheAnne Allen, Washington State TB Controller Washington State Department of Health
8:05-9:05	**KEYNOTE: ?** Dr. Michael Kimerling Bill and Melinda Gates Foundation
9:05-9:30	**WA State TB Epidemiology** Shawn McBrien, MPH, Epidemiologist Washington State Department of Health TB Program
9:30-10:00	**TB & Pediatrics Refresher** Scott Lindquist, MD Washington State TB Program Medical Consultant
10:00-10:10	**BREAK**
10:10-11:00	**Understanding the Laboratory Network in Washington** State PH Lab, PHSKC Lab, Harborview Lab, a Commerical lab
11:00-Noon	**New Drugs/12 Dose Regimen** Sundari Mase, MD, MPH, Center for Disease Control
12:00–1:00pm	**WORKING LUNCH & NETWORKING**

Planning Notes	

1:00-1:10 **Curry International Tuberculosis Center**
Kay Wallis, ???

1:10-2:10 **Florida Correction regarding Local Jail Health & TB Suspects**
Tara Wildes, Chief Jacksonville Sherriff's Office, Florida

2:10-2:30 **Visual Stories**
Erica Berg, Refugee Youth Empowered

2:30 – 3:00 **EVALUATION & CLOSURE**

WA State TB Program LHJ Business Meeting

(Attendance: LHJ TB staff only)

3:00-3:10 **Introduction: History of the MDR Workgroup**
SheAnne Allen, Washington State TB Controller

3:10-4:20 **Update from workgroup MDR/XDR:**
Costs of and potential impact on LHJs

4:20-4:30 **EVALUATION & CLOSURE**