



# Saint Luke's Regional Laboratories Clinical Laboratory Letter



January 2011

## Transfusion-Transmitted HIV Infection from Missouri Blood Donor

The current risk for acquiring HIV infection from a blood transfusion is estimated at 1 in 1.5 million. Screening for HIV infected blood products includes enzyme immunoassay for HIV 1/2 antibody, as well as HIV nucleic acid testing (NAT) performed on a sample from every donation, prior to any products' release for transfusion.

Recently, CDC described the first U.S. case of transfusion-transmitted HIV infection reported since 2002 (MMWR October 22, 2010 / 59(41);1335-1339). In November 2008, a donation at a Missouri blood center tested positive for HIV infection. Through a lookback process, the same donor was found to have donated in June 2008. Products from the June 2008 donation were transfused to two different recipients. One of the recipients died 2 days post-transfusion due to cardiac surgery complications. The other recipient received fresh frozen plasma during a kidney transplant. This recipient was subsequently found to be HIV-infected. Sequencing of the donor & recipient's HIV strain by the CDC confirmed them to be identical viruses.

Repeat testing of specimens from the June 2008 donation confirmed the absence of detectable HIV infection. NAT testing can detect HIV infection as early as 10-15 days after infection, therefore the donor is presumed to have been in the interval between acute infection and having detectable HIV RNA at the time of donation. Further questioning of the donor revealed he incorrectly answered pre-donation questions which would have categorized him at high risk for HIV infection & excluded him from donation.

This case is a reminder that even though the blood supply is extremely safe, a small percentage of patients continue to experience serious adverse transfusion reactions. The best way to minimize risk is to order a transfusion only when absolutely necessary.

## Shiga Toxin

Community acquired diarrhea outbreaks are most often caused by enteric pathogens such as *Salmonella*, *Shigella*, *Campylobacter* and Shiga toxin-producing *E. coli* (STEC). Although most strains of *E. coli* are harmless, *E. coli* O157:H7 has integrated the gene that produces Shiga toxin, possibly by means of a virus which transferred the gene from *Shigella* to *E. coli*. Most STEC infections are caused by *E. coli* O157:H7, which was first recognized as a pathogen in 1982. *E. coli* are serotyped on basis of their somatic (O) and flagellar (H) antigens. *E. coli* O157:H7 is so named because it possesses the 157<sup>th</sup> somatic antigen ever identified and the 7<sup>th</sup> flagellar antigen.

Although *E. coli* O157:H7 is the major cause of large Shiga toxin related outbreaks in the U.S., non-O157:H7 outbreaks have been documented. Colitis due to many other serotypes has been reported in other parts of the world since 1984. Non-O157 *E. coli* are generally associated with less severe disease than O157:H7. Non-O157 serogroups include O26, O45, O103, O111, O121 and O145. At least two reports of human disease caused by Shiga toxin producing *Citrobacter freundii* and *Enterobacter cloacae* have been reported.

STEC infections and HUS occur in patients of all ages, but the incidence of STEC infection and HUS is highest in children aged <5 years. STEC infections can occur throughout the year, but are most common during summer months. Each year in the U.S., STEC cause approximately 100,000 illnesses per year, 3000 hospitalizations, and 90 deaths. They are the leading cause of hemolytic uremic syndrome (HUS), the most common cause of acute renal failure in children. Endothelial cells in the intestine, kidney and brain are the major targets of the toxin.

STEC transmission occurs through consumption of a wide variety of contaminated water and foods, including undercooked ground beef, unpasteurized

juice, raw milk, and raw produce such as lettuce. Outbreaks are due to fecal contamination from ruminants, such as cattle. Transmission can also occur by person to person contact in settings with close contact and substandard hygiene, such as child care and institutionalized care settings. Both STEC and *Shigella* can cause infections after ingestion of only 10 to 100 organisms.

The onset of illness usually occurs between 3 and 7 days after exposure. STEC infection causes abdominal pain and acute, often bloody, diarrhea secondary to hemorrhagic colitis. Approximately 8% of persons with STEC infection develop hemolytic uremic syndrome (HUS), which is a life-threatening condition characterized by thrombocytopenia, hemolytic anemia and renal failure. The signs and symptoms of HUS are very similar to TTP. When TTP is diagnosed after a diarrheal illness, the condition is usually caused by infection with 0157 STEC and classified as HUS.

Currently there are no clear guidelines for who should be tested for Shiga toxin producing *E. coli* infection. Saint Luke's Regional Laboratories tests all stools submitted for culture for *Salmonella*, *Shigella*, *Campylobacter* and STEC. Prompt and accurate diagnosis is important because appropriate treatment with parenteral volume expansion early in the course of the illness can decrease renal damage and improve outcome. Antibiotics may exacerbate disease by increasing toxin production and release. The laboratory detects STEC with bacterial culture on sorbitol-containing MaConkey selective media and toxin detection by enzyme immunoassay (EIA). Most 0157 *E. coli* are sorbitol nonfermenters and produce colorless colonies. Non-0157 strains are detected by Shiga toxin EIA. Positive cultures and toxin tests are sent to the state public health laboratory for confirmation and serotyping. STEC are difficult to detect in stool after the first week of illness, so samples should be submitted as early as possible after onset of infection.

Toxins produced by STEC were named based on their similarity to Shiga toxins produced by *Shigella dysenteriae* type 1. STEC that cause hemorrhagic colitis produce 1 of 2 types of Shiga toxin (Stx),

spinach and alfalfa sprouts. Most of these STx1 and STx2. These toxins are encoded by separate genes, *stx*<sub>1</sub> and *stx*<sub>2</sub>, one or both of which may be carried by *E. coli* organisms, independent of each other. STEC strains that produce only Stx2 toxin are more often associated with HUS than strains that only produce STx1 or that produce both toxins. EIA detects both types of toxin STx1 and STx2, produced by both *E. coli* 0157:H7 and non-0157 strains. EIA requires overnight broth cultures of fecal specimens for maximum sensitivity. Whether a STEC illness progresses to HUS depends on strain virulence and host factors.

### **New Urine Specimen Collection Device**

Improper transport of urine specimens impacts quality of results for both urinalysis and urine culture. Prolonged length of time between specimen collection and analysis can result in false-positive urine nitrites and false-negative glucoses. Likewise, bacterial overgrowth in non-preserved, non-refrigerated specimens leads to false-positive or contaminated urine culture results. Beginning in January, Saint Luke's Regional Laboratories will introduce a closed urine transport system with integrated transfer tubes that contain preservative. This system decreases potential for bacterial contamination and allows for urine culture set-up up to 48 hours after collection and urinalysis up to 72 hours after collection. All urine specimens collected with this system include a preservative tube that can be used for add-on urine culture orders. The new collection device will be used for inpatients at all SLHS hospitals first, followed by other SLRL clients.

### **Update to von Willebrand Profile**

Effective January 26<sup>th</sup>, Mayo Medical Laboratories will no longer offer Ristocetin Cofactor as a stand alone test, nor will they include it in their von Willebrand Profile. Von Willebrand Factor Activity will replace Ristocetin Cofactor.

Saint Luke's Regional Laboratory's von Willebrand profile has included VWF activity for more than 2 years. There will be no change to our profile. We will continue to reflex to the Ristocetin Cofactor, available at ARUP Laboratories, when an abnormal VWF activity is obtained.