Saint Luke's REGIONAL LABORATORIES

Clinical Laboratory News

November 2015

Lactate Update

Lactic acid is the product of anaerobic glycolysis and exists in the blood entirely as lactate ion. Lactic acidosis is defined as a plasma lactate level higher than 5 mmol/L with a pH of less than 7.35. Laboratory findings typically include decreased blood pH, decreased bicarbonate level, decreased pCO_2, and increased lactate. Anion gap is typically, but not always, increased.

Raised blood lactate concentrations may result from either increased production by tissues or decreased clearance by the liver. In addition, some drugs, toxins and inborn errors of metabolism can produce lactic acidosis.

Increases in lactate synthesis in the absence of tissue hypoxia may occur when the rate of glucose metabolism exceeds mitochondrial function. Blood lactate levels often increase slowly in patients with lymphoma, leukemia, neoplasms, and sepsis. Increases are usually modest, in the range of 1 to 2 mmol/L. Malignant cells have a higher rate of anaerobic glycolysis. Certain drugs and ethanol can also increase blood lactate levels. Phenformin, a biguanide, causes lactic acidosis by increasing both glucose utilization in peripheral tissues and conversion of pyruvate to lactate. Metformin, another biguanide, acts similarly to phenformin, but causes lactic acidosis much less frequently and usually only in patients with coexisting renal failure.

Lactic acidosis is seen in hypoxia associated with acute cardiac or respiratory failure, shock, and acute blood loss. Blood lactate levels rise very rapidly as oxygen delivery to the peripheral tissues decreases below a critical level. An increased blood lactate level is an earlier indicator of tissue hypoxia than pH. In patients with circulatory shock, mortality increases from 10 to 90% as lactate concentration increases from 2 to 8 mmol/L. A sudden increase in oxygen requirement occurs in patients with grand mal seizures and in patients with severe asthma. The increased work of breathing results in release of lactate from the muscles of respiration

Because lactate is normally metabolized in the liver, blood levels are highly dependent on hepatic perfusion and function. During circulatory failure, lactate clearance is slowed down, especially in the presence of liver dysfunction. Hepatic tumors or metastases impair the liver's ability to clear lactate.

Lactate is the most widely used marker of tissue hypoperfusion in patients with severe sepsis. In 2002, the Surviving Sepsis Campaign added lactate to their criteria for diagnosis of severe sepsis. If lactate is elevated above 2.2 mmol/L, resuscitation should be targeted to normalize lactate as rapidly as possible. If the initial lactate is elevated, it should be remeasured as part of the 6 hour resuscitation bundle.

As of October 22, 2015, Saint Luke's Health System Laboratories began cascading elevated lactates, so that lactate results >2.0 mmol/L generate a repeat sample to be collected 3 hours after the initial draw (metro campuses) or 2 hours after the initial draw (regional campuses).

Lactate can be measured on arterial or venous blood samples. The specimen requirement for arterial samples, or venous samples on inpatients, is a green top (heparin) tube or lithium heparin blood gas syringe. Samples should be transported to the laboratory on ice, and must arrive within one hour of collection. Reference range for arterial lactate is <1.3 mmol/L and for venous lactate is 0-2.0 mmol/L.

Cord Blood Gas Reference Range

Reference intervals have recently been revised for blood gases from cord blood samples to include ranges for pO2 and pCO2, and update pH ranges:

Cord Blood	New range	Old range
pH arterial	7.23-7.33	7.03-7.45
pCO2 arterial	41-57 mm Hg	
pO2 arterial	12-24 mm Hg	
pH venous	7.30-7.40	7.14-7.50
pCO2 venous	32-44 mm Hg	
pO2 venous	23-35 mm Hg	

Flu Season Approaches

As of November 14, influenza activity remains at low levels in most regions of the United States. Missouri is reporting sporadic influenza cases, while Kansas has reported no cases to date. Surveillance data collected thus far indicate that influenza A H3 is predominant, with no resistance to neuraminidase inhibitors detected. Since mid-October, Saint Luke's Laboratories have reported fewer than 10 positive influenza A tests.

During past seasons when influenza A H3N2 viruses have predominated, higher overall and age-specific hospitalization rates and increased mortality have been observed, especially among older people, very young children, and persons with certain chronic medical conditions compared with seasons during which influenza A H1N1 or influenza B viruses have predominated.

Saint Luke's Laboratories offer three different tests for detection of influenza. Rapid antigen testing is performed by all testing sites. It detects both influenza A and influenza B. The major disadvantage of rapid antigen testing is low sensitivity of 60-80%. Sensitivity of the rapid antigen tests is variable from season to season, depending on the predominant strain.

Specimens can also be sent to the central Microbiology laboratory at Saint Luke's Hospital for respiratory PCR panel testing. The panel detects influenza, influenza B, and differentiates A subtypes H1 & H3. Additional pathogens detected by the panel include coronavirus (not MERS co-V), human metapneumovirus, rhinovirus, parainfluenza, RSV, adenovirus, *Bordetella pertussis*, *Mycoplasma pneumoniae*, and *Chlamydophila pneumoniae*. Respiratory panel testing can be performed on bronchoscopy specimens in addition to nasal swabs or washes.

The Microbiology laboratory also performs PCR testing for influenza viruses only. In addition to influenza B, the flu A/B PCR detects influenza A H1 and H3 subtypes and differentiates 2009 H1N1. Sensitivity averages 90% with specificity near 100%. During seasons when rapid antigen sensitivity is low, influenza PCR testing is recommended for patients needing hospitalization for respiratory illness, at the time of admission. Nasopharyngeal or nasal swabs submitted in viral transport media or nasal washes are the only acceptable specimen types for flu A/B PCR.

Test Name	Detects	Specimen types	Transport
Rapid Flu A/B Antigen	Influenza A & B	NP/nasal swab Nasal wash	Flocked swab in VTM Flocked swab in saline Eswab in Amies
Flu A/B PCR	Influenza A & B (differentiates 2009 H1N1 subtype)	NP/nasal swab Nasal wash	Flocked swab in VTM
Respiratory panel PCR	7 respiratory viruses, (differentiates influenza subtypes) Bordetella, Mycoplasma, Chlamydophila	NP/nasal swab Nasal wash Bronchoscopy wash/lavage	Flocked swab in VTM