Lactic Acidosis in Patients With Cancer

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Lactic acidosis is the most common metabolic acidosis in hospitalized patients—the result from an underlying pathogenic process. To successfully manage lactic acid production, its cause needs to be eliminated. Patients with cancer have many risk factors for developing lactic acidosis, including the cancer diagnosis itself. Patients with lactic acidosis are critically ill, requiring an intense level of nursing care with accompanying frequent cardiopulmonary and renal assessments. The mortality rate from lactic acidosis is high. Therefore, appropriate nursing interventions may include end-of-life and palliative care.

Lactic acidosis is the most common metabolic acidosis in hospitalized patients, and is defined as serum lactic acid (LA) levels greater than 5 mmol/L (normal: less than 2 mmol/L) with a serum pH of less than 7.3 (normal: 7.32–7.42) (Emmett, 2013; Martinez-Outschoorn et al., 2013). Two molecules of lactate produced by the Cori cycle are transported by the blood, primarily to the liver (60%–90%) and kidneys (30%), as well as heart and other tissues, where it is metabolized back to glucose (gluconeogenesis) (Brandis, n.d.; Emmett, 2013; Gunnerson, 2013; Nandwani, Saluja, Yats, & Mehta, 2010; Ruiz et al., 2011) (see Figure 1).

LA is constantly produced by the cytoplasm of almost all of the cells, particularly the red blood, brain, gut, and skin cells. LA is a normal end product of glucose metabolism via the anaerobic glycolytic pathway. When adequate oxygen and glucose reach the cells, they obtain their needed energy from the glycolytic production of pyruvate, the end product of glycolysis. The pyruvate is converted to acetyl coenzyme A (CoA) by the enzyme pyruvate dehydrogenase. Thiamine is needed as a cofactor for this reaction. The acetyl-CoA enters the cell’s mitochondria and joins in the tricarboxylic acid cycle (TAC), or Krebs cycle. Thirty-six molecules of adenosine triphosphate (ATP) are produced from one molecule of glucose. If no oxygen is available, the TAC cycle does not proceed, and energy is obtained by converting pyruvate to LA by the enzyme lactate dehydrogenase in the presence of nicotinic acid dehydrogenase (NADH). That anaerobic process only produces four molecules of ATP per molecule of glucose, which dramatically decreases the amount of ATP available to cells (Brandis, n.d.; Emmett, 2013; Gunnerson, 2013; Nandwani, Saluja, Yats, & Mehta, 2010; Ruiz et al., 2011) (see Figure 1).

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Other mechanisms that may contribute to the development of lactic acidosis in patients with cancer are decreased ability to clear lactate from the liver because of the presence of cancer, overproduction of lactate caused by thiamine and/or riboflavin deficiencies, and embolization of the microvasculature by malignant cells (Emmett, 2013; Ruiz et al., 2011). Patients with cancer also are prone to sepsis, which may cause impaired tissue perfusion with anaerobic metabolism (Blomklans, 2007; Dell, 2014).