IS – 23
The investigation and approach to management of the poisoned patient with lactic acidosis of unknown cause

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Introduction: Lactic acidosis usually indicates a severe clinical situation. Etiologies of lactic acidosis are multiple and classified as A-type, if in relation with tissue hypoxia and as B-type, including all cases related to underlying primary diseases, poisonings and iatrogenic events as well as inborn errors of metabolism. The objectives of this presentation are 1)- to review mechanisms of drug-induced lactic acidosis and 2)- to report specific management when required.

Methods: Review of medical literature and recommendations of scientific societies.

Results: Increase in blood lactate concentration results from unbalance between lactate production (stimulation of anaerobic glycolysis or dysfunction of mitochondria respiratory chain) and clearance. Drug exposure represents the first direct cause of lactic acidosis including poisonings with metformin, cyanide, valproic acid, nucleoside/tide reverse transcriptase inhibitors, and adrenergic stimulants. However, drug-induced circulatory failure is probably the most typical situation associated with lactic acidosis. All cardiotoxicants including calcium-channel blockers, sodium-channel blockers and vasodilatators may induce hyperlactatemia. With beta-blockers, elevation in blood lactate is weak due to the blockage of its production and thus lactatemia does not correspond to the severity of the hemodynamic dysfunction and tissue ischemia. In the presence of hospital-acquired lactic acidosis, iatrogenic hypotheses should be suggested like propofol infusion syndrome, propylene glycol intoxication, as well as beta-adrenergic catecholamine- and linezolid-related side-effect. Other etiologies, sometimes obvious and sometimes more difficult to assess, are responsible for increase in blood lactate in the poisoned patient including liver insufficiency and seizures or status epilepticus especially if tonic or clonic. More rarely, lactic acidosis leads to the identification of an underlying solid cancer or blood malignancy. In selected patients at risk, vitamin deficiencies like thiamine and biotin and inborn errors of sugar metabolism should be suspected. In all lactic acid cases as no specific treatment of increased blood lactate exist, patient management should rely on the treatment of the underlying etiology and be prompt once the diagnosis is assessed, since this severe condition is usually associated with a bad final outcome. Adequate antidotes and hemodialysis when indicated should be provided.

Conclusions: It is mandatory to adequately understand the cause and mechanism of drug-induced or poisoning-associated increase in blood lactate to allow optimized management and improvement in patient's prognosis.