Glycated hemoglobin

Glycated hemoglobin (hemoglobin A1c, HbA1c, A1C, or Hb1c; sometimes also referred to as being Hb1c or HGBA1C) is a form of hemoglobin that is measured primarily to identify the three-month average plasma glucose concentration. The test is limited to a three-month average because the lifespan of a red blood cell is four months (120 days). However, since RBCs do not all undergo lysis at the same time, HbA1C is taken as a limited measure of 3 months. It is formed in a non-enzymatic glycation pathway by hemoglobin's exposure to plasma glucose. HbA1c is a measure of the beta-N-1-deoxy fructosyl component of hemoglobin.[1] The origin of the naming derives from Hemoglobin type A being separated on cation exchange chromatography. The first fraction to separate, probably considered to be pure Hemoglobin A, was designated HbA0, the following fractions were designated HbA1a, HbA1b, and HbA1c, respective of their order of elution. There have subsequently been many more sub fractions as separation techniques have improved.[2] Normal levels of glucose produce a normal amount of glycated hemoglobin. As the average amount of plasma glucose increases, the fraction of glycated hemoglobin increases in a predictable way. This serves as a marker for average blood glucose levels over the previous three months before the measurement as this is the lifespan of red blood cells.

Elevated blood glucose is frequently detected early after aneurysmal subarachnoid hemorrhage (aSAH) and is considered a risk factor for poor neurological outcome. However it remains unclear whether hyperglycemia is caused by the SAH ictus or reflects a pre-existing hyperglycemic metabolism. In a prospective register Beseoglu and Steiger analysed glycated hemoglobin levels (HbA1c) in patients with aSAH and its influence on outcome.

Between July 2012 and July 2014, 87 patients with confirmed aSAH were included (NCT02081820). Within 72h HbA1c levels were assessed as a measure for hyperglycemic metabolism preceding aSAH. Blood glucose levels were recorded upon admission. Patient outcome was recorded after 6 months using modified Rankin scale (mRS).

HbA1c levels did not correlate with initial neurological status (p=0.338, r=0.104). On the contrary, initial blood glucose levels correlated significantly with neurological status at admission (p=0.001, r=0.341). Additionally, HbA1c levels failed to show a significant influence on the occurrence of delayed cerebral ischemia (DCI) (p=0.400) or outcome after 6 months (p=0.790).

A pre-existing hyperglycemic metabolism does not contribute to the severity of aSAH or influences the quality of neurological recovery. Hyperglycemia after aSAH correlates with initial neurological status and patient outcome and is potentially attributable to the metabolic changes induced by the brain injury after the hemorrhage [1)].

1)
Beseoglu K, Steiger HJ. Elevated glycated hemoglobin level and hyperglycemia after aneurysmal subarachnoid hemorrhage. Clin Neurol Neurosurg. 2017 Oct 31;163:128-132. doi: 10.1016/j.clineuro.2017.10.037. [Epub ahead of print] PubMed PMID: 29101862.