Editorial

Glucose Management in Acute Stroke: A Daunting Nursing Task?

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Continuous glucose monitoring can give information on glucose metabolism during the early phase of Acute Ischemic Stroke (AIS). This may contribute to more effective treatment and improved patient outcomes. Current guidelines in patients with AIS recommend maintaining plasma glucose levels at 140-180 mg/dl and close monitoring for the prevention of hypoglycaemia (<60 mg/dl). Moreover, post-AIS glucose levels have fluctuations that require careful monitoring and recording. Along these lines Allport et al, (2006) used a blinded continuous AIS glucose recording system and found that during induction, hyperglycaemia was observed in 81% of diabetic and 32% of non-diabetic patients. Non-diabetic patients, 8 hours after onset of symptoms had a 50% glucose increase.

However, continuous glucose recording offers many important parameters resulting both from measurements and from the processing of these by the software of each device. Standard measures of continuous glucose monitoring to be taken into account in research and clinical care are as follows:

- Number of days the CGM has been loaded.
- Percentage of CGM device activation time.
- Average glucose.
- Glycaemic variability target (%CV) ≤ 36%*.
- Time in the adjustable range, above and below this (Zhou et al., 2024).

From a statistical point of view, the standard deviation (SD) around a mean glucose value measured over a 24-hour period using the continuous glucose monitoring system is probably the most appropriate tool for assessing intra-day glycaemic variability. The method incorporates small and large variations, but does not allow them to be differentiated. The other methods developed for assessing intra-day glycaemic variability are more or less based on determining the differences between the maximum and minimum glucose levels (ElSayed et al., 2023).

International practice for the use of continuous glucose monitoring recommends CV as the preferred measure and considers SD as secondary. CV is calculated by dividing SD by the average glucose and multiplying by 100 to obtain a percentage. SD represents the spread in glucose measurements around the average. The target is the lowest possible SD, which will reflect a constant glucose level with minimal fluctuations. The preference for the use of CV over SD is due to the fact that SD is heavily influenced by the average glucose - patient with higher average glucose will have a higher SD. CV corrects and normalizes GV. Current recommendations target a CV of 36% or lower, it is considered a constant plasma glucose concentration indicator (American Diabetes Association, 2021).
The overall nursing management of the patient with an IAEA should include:
Protocols for nurses in the emergency department and sorting include the following:

- Neurological status assessment using scales.
- Hydration, avoiding glucose solutions.
- Appropriate patient placement (sedentary, semi-sedentary).
- Feeding and dysphagia control.

- Vital signs.
- Oxygen therapy.
- Glucose management.
- Monitoring of intestinal function and bladder.
    - Mobility
    - Depression / psycho-mental evaluation (Tauschmann et al, 2022).

Additionally, nurses should closely monitor the patient's blood pressure (BP) in the first 48 hours after the start of stroke. Given the various available data, the level of BP that should be maintained in patients with acute ischemic stroke to ensure optimal outcome remains unknown. However, an increase in systolic blood pressure of 9.1 mmHg and an increase in diastolic blood pressure of 2.3 mmHg were associated with poor outcome. Intensive reduction of BP in the acute phase after stroke (systolic BP <140 mm Hg) is also not recommended.

Hypotension and hypovolaemia should be corrected to maintain tissue perfusion levels. In addition, the nurse should perform the patient's continuous evaluation on an individual basis irrespective of whether the patient received reperfusion therapy. The frequency of all assessments and observations should be determined by the patient's condition (Tauschmann et al., 2022).

In order to prevent or detect complications in time nurses should regularly evaluate patients for a change in communication level (worsening of infarct, cerebral edema and hemorrhagic transformation). The avoidance of aspiration pneumonia, flooding and falls are also included in the context of the patient's personalized nursing management.

Nursing vigilance, therefore, includes not only the prevention of hyperglycaemia, but more importantly the intensified monitoring to avoid hypoglycemic attacks. Hypoglycemia is defined as clinical syndrome characterized by low serum glucose, usually <70 mg/dL210. It is common in diabetic patients receiving drug therapy. Disturbance in the mechanisms of glucose homeostasis leads to the onset of symptoms that can be summarized by adrenergics such as tremor, sweating, anxiety, palpitations and tachycardia, due to the antiretroviral mobilization of the sympathetic nervous system and neuroglycogenics such as headache, weakness in concentration, blurred vision, confusion, amnesia and behavioral changes, due to the decrease in the glucose supply to the brain (Edelman et al., 2018).

Effective nursing intervention and communication - cooperation between doctors and nurses is considered essential to prevent iatrogenic hypoglycemia. The Committee of the Regions believes that the Commission should develop a European Health and Safety Agency (EMEA) to monitor and control the use of SIGINT. It is also worth noting that the stricter the glycemic target, the greater the likelihood of hypoglycemia.

In the context of hypoglycemia, intensified glycemic control of the patient becomes more important as hypoglycemia may persist without the onset of clinical symptoms; it may also mimic or be under-shadowed by symptoms of existing brain damage, or even lead to misdiagnosis.

Asymptomatic hypoglycemia (PAH) is characterized by the sudden onset of neuroglycosenic symptoms without awareness of warning symptoms within the autonomic nervous system. It is an important restriction on achieving a narrow glycemic goal and a particular risk factor in stroke patients.

Although the etiology of AI is multifactorial, possible mechanisms include chronic exposure to low blood glucose, pre-existing hypoglycemia, recurrent severe hypoglycemia, and regulatory hormone failure.

Prior hypoglycemia leads to an easing of the catecholamine response in a subsequent episode of hypoglycemia. Ramanathan & Cryer (2011), they argued that intravenous infusion of adrenergic blockers in a hypoglycemic attack prevents counter-regulatory failure at the next seizure. Also, patients with type 1 EDs, while asleep, have significantly reduced response to epinephrine and hypoglycemia, as well as reduced ability to wake up during hypoglycemia. Thus,

deaths are explained at night of healthy young people with type 1.

The distinction of hypoglycemic symptoms in AIS patients may be difficult in some cases. Glucose metabolism disorders are one of the common metabolic abnormalities that occur as a stroke. Focal neurological defects occur in up to 2% of those who experience hypoglycemia. Neuroglycopaenia leads to vasospasm which is responsible for asymmetric blood flow leading to ischemia. Rapid correction of hypoglycemia usually improves symptoms.

The immediate availability of glucose monitoring and the availability of an easily interpretable hypoglycemic protocol can ensure effective care for hypoglycemic patients. In these cases, glycaemic control should be significantly reduced when compared to alternative treatment options.

In a hypoglycemic episode, evaluation should include examining the patient's level of consciousness, respiratory and circulatory support, the existence of intravenous access, monitoring of time and quantity of insulin doses, the likely state of non-food administration, or the last food and drink intake. If the patient can be safely treated with oral carbohydrates, appropriate fluids or glucose tablets should be administered. If the patient fails to respond, then intravenous dextrose or intramuscular glucagon injection are the preferred treatment methods. Attempt to treat by increasing the intravenous glucose infusion rate quickly puts patients at risk of fluid overload, because 100 cc of dextrose solution 5% offers only 5g of water.

Factors that lead to glucose deregulation include post-SNE dysphagia. Oropharyngeal dysphagia is a symptom recognized among 39% - 78% of patients with SNEs. Dysphagia is one of the leading causes of post-SNE mortality because of its association with complications such as malnutrition or aspiration pneumonia.

In patients on a full intestinal diet, hyperglycaemia has been reported at 30% - 47%, with half of them not diagnosed with diabetes.

Conclusions

The immediate recognition and treatment of hyperglycaemia and hypoglycemia are of utmost importance in any case, not least in patients after SNEs. Both the deregulation of diabetic patients and the hyperglycaemia of stress have in the past created the reasonable question of whether they are innocent manifestations of the severity of the disease or affect independently the ischemic area, worsening the consequences of SNEs. In the latter case, it is imperative to carefully monitor the patient whatever the cost of this intensive care.

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