ORIGINAL ARTICLE

Postmortem Identification of Hyperglycemia using Vitreous Fluid Analysis

TASNEEM MURAD[1], MUNAIR AHMAD BALOCH[2], FAISAL KHAN[3], JAHAN ZAIB[4], SIDRA MUMAL[5], ALIA SARFRAZ[6]
[1]*Assistant Professor of Forensic Medicine Islamic International Medical College Rawalpindi*
[2]*Associate Professor of ENT Bolan Medical College Quetta*
[3]*Associate Professor of Biochemistry Multan Medical & Dental College, Multan*
[4]*Associate Professor of Physiology Jhalwan Medical College Khuzdar*
[5]*Demonstrator Pharmacology Department Islamic International Medical College Rawalpindi*
[6]*Senior Lecturer Forensic Medicine Department, Islamabad Medical & Dental college Islamabad*
Correspondence to: Dr. Tasneem Murad, Email: tasneem.murad@riphah.edu.pk, Cell: 03005319963

ABSTRACT

Objective: To investigate the role of vitreous glucose and lactate levels in determining post-mortem hyperglycemia.

Methods: In this study, 150 autopsies for postmortem Identification of hyperglycemia using vitreous fluid analysis were also conducted in mortuary unit of DHQ Hospital Rawalpindi during Jan. 2020 to December 2020. The analysis was performed as soon as the body arrived in the mortuary. 0.2 ml of Vitreous fluid was obtained from the centre of eyes for each patient in a 1 mL syringe. Blood gas analyzer was used for potassium (K^+), glucose and lactate measurement, by directly connecting the sample to the analyzer. The final autopsy studies were conducted 2 to 3 days after the first sample and vitreous fluid as obtained again for further biochemical analysis.

Results: Mean age was 53 years with 70% comprising male population. We observed an initial steeper drop in vitreous glucose levels in early postmortem period time, however, in samples obtained after 2 to 3 days the glucose levels remained stable. In contrast, the lactate levels continued to rise with postmortem time as documented using vitreous potassium levels. In known diabetic patients we found glucose levels >10 mmol in all patients at first sample and therefore this value can be used as cutoff to labelled hyperglycemic state as the cause of death.

Conclusion: Vitreous glucose can be used as a bedside technique to determine hyperglycemia as the cause of death and can provide a valuable information regarding cause of death before autopsy studies.

Keywords: hyperglycemia, postmortem, vitreous biochemistry.

INTRODUCTION

In forensic labs, the biochemical analysis is routinely performed in deceased patients where patho-physiologic changes determined by morphologic methods are no sufficient to identify the cause of fatality, and to better understand the contributing factors, predisposing factors and pathologic processes before death.[1,2] This is especially important for medico-legal cases where identifying the fatality cause is of prime importance. Elevated levels of vitreous glucose and ketones from the autopsy biopsy specimens have been shown to be beneficial in determining the diagnosis of diabetes.[3-6]

In comparison to blood, the vitreous fluid has limited cell numbers and is therefore little affected by postmortem changes unlike the blood and serum which are severely affected by postmortem changes because of excretion of extra and intracellular contents which significantly hampers their use in autopsy studies. Therefore, biochemical analysis of vitreous fluids has been successfully used for diagnosis of various conditions in autopsy studies.

The principal advantage of using chemistry analysis in forensic sciences is to obtain analysis results as early as possible. In postmortem period, diagnosis of hyperglycemia can provide valuable information about the etiology of death. It is recommended to measure vitreous lactate along with blood glucose levels to determine the postmortem hyperglycemia. Studies have recommended to measure sum of lactate and glucose levels, because the sum of these provides most accurate information because it also provide information about the vitreous glucose that has been degraded from death to the time of diagnosis.[3, 7, 8] In this study we determined the vitreous hyperglycemia in postmortem patients.

METHODS

In this study, 150 autopsies for postmortem Identification of hyperglycemia using vitreous fluid analysis were also conducted in mortuary unit of DHQ Hospital Rawalpindi during Jan. 2020 to December 2020. The analysis was performed as soon as the body arrived in the mortuary. 0.2 ml of Vitreous fluid was obtained from the centre of eyes for each patient in a 1 mL syringe. Severely decomposed bodies and infants bodies were excluded. Approval for study was obtained from hospital IRB.

Blood gas analyzer was used for potassium (K^+), glucose and lactate measurement, by directly connecting the sample to the analyzer. The final autopsy studies were conducted 2 to 3 days after the first sample and vitreous fluid as obtained again for further biochemical analysis, however samples for toxicology investigations were obtained on the same day of examination.

Data regarding patient's personal information such as gender, age, cause of fatality, diabetes mellitus, and other medical records were obtained from the hospital. Patients having no previous medical records were also excluded from analysis.

Data was entered in SPSS v25 software. Simple descriptive statistics were calculated for study variables. Moreover, mean values of lactate and glucose were also noted in patients with different K^+ concentrations.

RESULTS

Mean age was 53 years with 70% comprising male population. Many of the patients with elevated glucose levels does not find to have history of diabetes. We observed glucose levels against different concentrations of potassium and did not observed a significant decrease in glucose levels when the potassium concentration increased to >10 mmol/L. This confirms that the glucose levels remain stable for a considerable time after death. However, in patients with small interval from death to sampling with K^+ levels ≤10 mmol/L the glucose concentration was high in comparison to other samples, which indicates that there is a steeper decrease in vitreous glucose immediately after death and after that the levels become stable. However, we observed that as the glucose concentration decreased the lactate levels got increased in these patients with increase in K^+ levels. This pattern may because of anaerobic metabolism continuing in the vitreous cells and possible in inner retinal layer (Table 1).

To test the assumption that sum of lactate and glucose is more reliable for determining hyperglycemic status, we also calculated the sum of lactate and glucose and determined its mean value in patients with different potassium levels. We found that instead of glucose the sum corresponds more to K^+ levels, as after 24 hours the glucose levels became zero in all most all the patients, but the sum of values continued to increase with increasing K^+ values (Table 1).

In bodies with diabetes mellitus history, the vitreous glucose levels were >10 mmol in all patients and the levels continued to decrease until the vitreous potassium increased to >10 mmol/L (estimating the death duration of 24 hours). After that the glucose levels remained stable for 2 to 3 days after death.

Table 1. Vitreous Glucose and Lactate Levels.

	Total	K^+ ≤10 mmol/L	K^+ 10.1-20 mmol/L	K^+ >20 mmol/L
Lactate	19.7±6.1	14.1±0.32	19.8±2.32	28.3±
Glucose	0.76±0.4	0.69±0.3	0.01±0.07	0.0±0.12
(Sum of lactate+Glucose)/2	10.3±3.2	7.8±0.7	10.4±0.6	15.2±`1.67

DISCUSSION

In present study, we used the vireous glucose, lactate and sum of these to determine the hyperglycemic states of diseased people to confirm the glycemic state as the cause of death. In routine urine glucose test is used to determine hyperglycemia and can help to label cases with higher likelihood of presence of hyperglycemia and to warrant further investigations. However, urine test is not an accurate predictor and cannot accurately exclude hyperglycemia.

We used 10 mmol/L as cut off value to define hyperglycemia state, we took this value on the value on the basis of assumption that the vitreous glucose drop about 3 mmol/L in early postmortem period and the concentration of glucose in vitreous fluid is half than the blood.[9] Therefore, this 10 mmol/L corresponds to 26 mmol of glucose in blood and is therefore ideal to determine the diabetic coma as the cause of death, if the sample is obtained within 24 hours after demise.

We observed that the vitreous glucose in postmortem period is decreased rapidly in early period of death and after that it becomes stables. This reduction in glucose levels were observed by Coe et al. 40 years before but they did not determined the time frame of reduction and stabilization of glucose levels.[10] The mechanism of this possible may be that initially the glucose is consumed by surviving inner retinal cells and hyalocytes and after their death the levels becomes stable.[11]

As lactate levels continue to increase after death, therefore vitreous lactate levels alone or sum of lactate and glucose levels cannot be used as reliable tool for determining hyperglycemia especially after 1 day of mortality. Palmiere et al. in their study recommended to use vitreous glucose and blood HbA1c levels for confirming hyperglycemia and they did not included lactate in their analysis.[12] Karlovsek et al. in another analysis concluded that vitreous glucose >13 mmol/L or mean sum of lactate plus glucose 23.7 mmol/L can be used as cutoff value to determine hyperglycemic coma as cause of mortality.[13] Similar to our study, Zilg et al. measured vitreous lactate & glucose levels in deceased medicolegal bodies. They observed an initial reduction in glucose levels in vitreous fluid in early death period, after that changes in glucose levels became stable. However, the lactate concetrations continued to rise despite stabilization of glucose levels. So the authors reported that increase in lactate levels is not only due to glucose breakdown but some other sources are responsible for increase in lactate levels. Therefore, mean sum of lactate plus glucose cannot be used as a reliable predictor of diabetic coma as the cause of mortality and only vitreous glucose should to be used for confirmation.[14]

CONCLUSION

Vitreous glucose can be used as a bedside technique to determine hyperglycemia as the cause of death and can provide a valuable information regarding cause of death before autopsy studies.

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