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Blood Acidity and Metabolic Acidosis In Hyperglicemia Crisis Patients After Rehydration Therapy

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ABSTRACT

The increasing status of the body's hyperglycemia triggers hyperosmolarity and dehydration, dehydration will reduce perfusion to the kidneys, decreased perfusion will reduce the excretory function of ketone acids and increase lactic acidosis due to what occurs in patients with KAD (Diabetic KetoAsidosis). The purpose of this study was to analyze the differences in Arterial pH after 3000 ml of rehydration therapy in patients with KAD (Diabetic KetoAsidosis). This research is a comparative analytical research. Based on the analysis test using paired T test p value = 0.000 with the purpose that it can be concluded that there is a change in arterial pH before and after rehydration therapy as much as 3000 ml. The mean changes in arterial pH in this study were close to normal values (Normal Arterial pH = 7.35 -7.45). Changes in arterial pH close to normal can be interpreted by rehydration therapy as much as 3000 ml to improve blood perfusion to the kidneys, thereby increasing kidney function in reducing ketone acid, decreasing ketone acid will reduce the occurrence of acidosis. After rehydration therapy, it is expected that acids and ketones can be excreted, therefore, fluids have an important role in the management of diabetic ketoacidosis (KAD) patients.

Keywords: KAD, Arterial pH, Rehydration Therapy

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INTRODUCTION

Fluid deficit that occurs in patients with KAD is caused by an increase in blood glucose which causes osmotic diuretics which triggers fluid transfer in the body (English & Williams, 2004)(Kitabchi et al, 2009). In KAD, the presence of insulin deficiency stimulates counter-regulatory hormones to carry out lipolysis and releases glycerol and FFAs which cause the accumulation of ketoacids (Semarawima, 2017). Increased ketoacids will be excreted into the kidneys, which will cause ketonuria (English & Williams, 2004). Diuresis and osmotic ketonuria also increase the total body sodium deficit via urine (Kamel S. Kamel, 2015). The combination of the above problems will reduce perfusion in the kidneys, decreased perfusion to the kidneys will increase the risk of ketoacids in the body which triggers an increase in the incidence of acidosis (English & Williams, 2004) (Gosmanov, Gosmanova, & Dillard-Cannon, 2014).

The main priority in the management of KAD is fluid therapy (Lutfi, Fitriasnani, & Kauniyah, 2019) (Kitabchi, 2009). The severity of the fluid deficiency that occurs is influenced by the duration of hyperglycemia, kidney function, and the patient's fluid intake (Diacon, 2012). In patients with KAD the water deficit can reach 6 liters (Fayfman, Pasquel, & Umpierrez, 2017). Initial fluid therapy is aimed at expanding interstitial and intravascular volume and rebuilding adequate renal perfusion (Hidayatulloh, Supriyadi, & Sriningsih, 2016). The type of fluid given in accordance with the guidelines for managing emergency hyperglycemia is isotonic fluid (normal saline containing 0.9% NaCl) with a dose of 10-20 ml / kgBW / hour depending on body conditions (heart function, blood vessels and kidney function) (Lutfi, 2019). Fluid resuscitation should be carried out aggressively (Gosmanov et al., 2014). The target is 50% fluid replacement of the fluids shortage in the first 8-12 hours and the remainder in the next 12-16 hours (Umpierrez, 2018). Physiological fluids (NaCl 0.9%) are given at a rate of 15-20 ml / kg / hour or more during the first hour (± 1 - 1.5 liters) (Fayfman et al., 2017). The choice of the next fluid depends on hydration status, serum electrolyte levels, and urine output (Bar-or et al., 2019). In general, 0.45% NaCl solution is given if the serum sodium level is high (> 150 mEq / L). The success of fluid therapy is determined by hemodynamic monitoring (blood pressure improvement), measurement of fluid intake and discharge, and clinical examination (Lutfi, 2019)(English & Williams, 2004). In patients with kidney, heart or liver disorders especially the elderly, monitoring of serum osmolality and continuous assessment of cardiac, renal, and mental status during fluid resuscitation is necessary to avoid iatrogenic fluid overload (Gosmanov et al., 2014).

Insulin deficiency, increased insulin regulatory hormones (cortisol, glucagon, growth hormone, and catecholamines), and peripheral insulin resistance lead to hyperglycemia (Semarawima, 2017), dehydration, ketosis, and electrolyte imbalance, which underlie the pathophysiology of DKA (Fayfman et al., 2017). KAD is characterized by hyperglycemia, metabolic acidosis, and increased concentrations of circulating ketones (American Diabetes Association, 2004). Ketoacidosis is the result of a lack of insulin that occurs along with an increase in counterregulatory hormones (glucagon, epinephrine, norepinephrine, catecholamines, cortisol, and growth hormone) (Kitabchi et al, 2009). Both of these result in changes in glucose production and increase lipolysis (Lutfi, Andri Wihastuti, & Kristianto, 2017). With a decrease in insulin effectiveness, it will result in an increase in gluconeogenesis, glycogenolysis and impaired peripheral glucose use (Suharto, Lutfi, & Rahayu, 2019)(Thorén et al., 2017). Hepatic gluconeogenesis is the main mechanism of crisis hyperglycemia in

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ketoacidosis supported by renal gluconeogenesis (Haak, Santen, & Hoeven, 2015) (Kitabchi et al, 2009).

An increase in osmolarity supported by increasing acidosis and ketogenesis will cause metabolic acidosis (pH <7.3) (Kamel S. Kamel, 2015). This situation is also exacerbated by the increase of lactic acidosis due to poor tissue perfusion. Progressive dehydration, hyperosmolarness, acidosis, and electrolyte disturbances will further exacerbate hormonal imbalances and cause this state to continue forming a cycle. As a result, metabolic decompensation will be progressive (Kamel S. Kamel, 2015). In the management of KAD, the organ most expected to play an active role is the kidney (Ekoe, J.M., Goldenberg, R., Katz, 2018). In human body, one of the roles of the kidneys is to excrete the waste products of the body's metabolism in the form of urine (Hung, 2012). In contrast to KAD patients, even though the kidneys are healthy, they will not be able to excrete normally. Along with the occurrence of hyperosmolarity, the body will experience dehydration, coupled with a state of metabolic acidosis (increased fatty acids and ketones) in blood plasma (Kamel S. Kamel, 2015). The kidneys which are supposed to compensate for metabolic acidosis are inhibited by dehydration. Blood that should be perfused into the kidneys and filtered, will not performed it's job. Thus the renal glomerulus prioritizes the dehydration of the body over the excretion of metabolic waste. The goal of rehydration management is to increase blood perfusion to the kidneys in order to maximize the process of excretion of fatty acids and ketones from the body by the kidneys in the form of urine (English & Williams, 2004) . After rehydration therapy, it is expected that acids and ketones can be excreted so that the acidity of the blood decreases and can increase blood pH to a normal state (7,35-7,45)(Kamel S. Kamel, 2015). Therefore, fluids have an important role in the management of diabetic ketoacidosis (KAD) patients (Biff F. Palmer, 2015).

METHODS

This research is comparative analytical research with retrospective cohort approaches. This approach is used because researchers retrieve data after a given hydration program therapy. The limiting observation of rehydration therapy only up to 3000 ml in research is based on conditions in the field, in which rehydration therapy is only at 3000 ml in patients with KAD. Subsequently, fluid maintenance is given in accordance with the hemodynamic condition of the patient. This research has fulfilled the ethical principles of research based on a letter of ethics issued by dr. Iskak by number. 070/1862 / 407.206 / 2018. Data retrieval conducted during June-July 2018 at Dr. Iskak Tulungagung. The number of respondents in this study was 26 respondents of KAD since January – December in 2017 with characteristics of patients with KAD treatment in ICU both in the red zone room and yellow zone that gets rehydration therapy. Data collection of respondents using techniques of total sampling, undergoing isotonis NaCl fluid rehydration therapy, 9% as much as 10-20 ml/kgBW/hour. Analysis of data in This study uses wilcoxon test.

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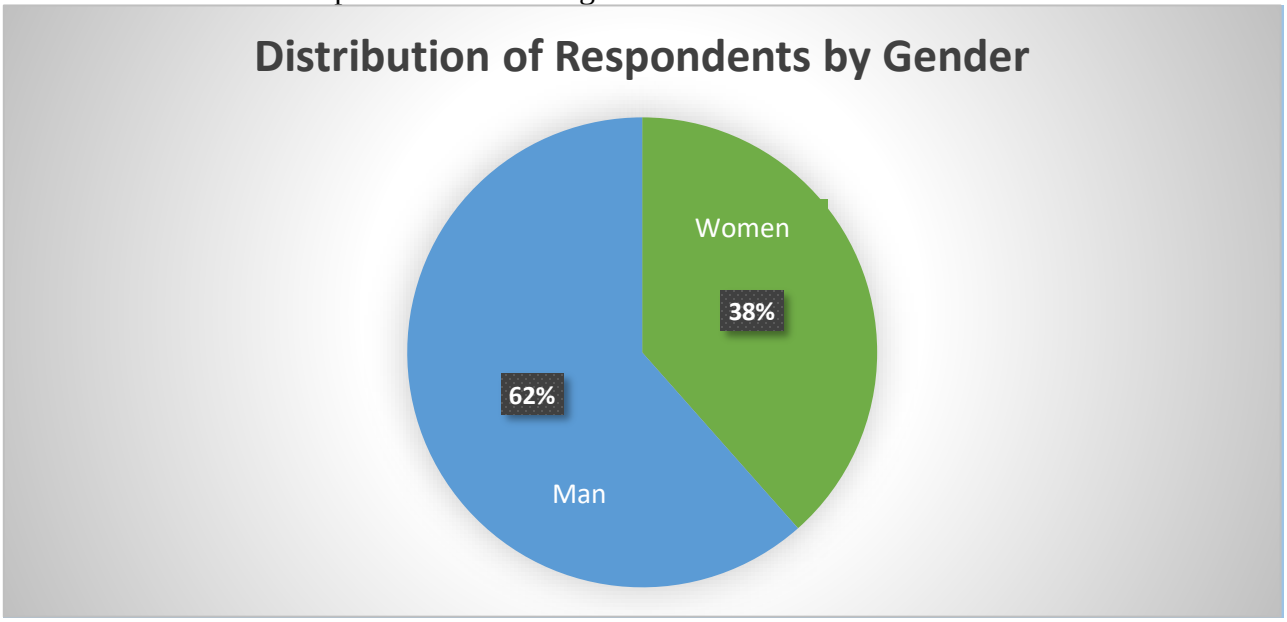
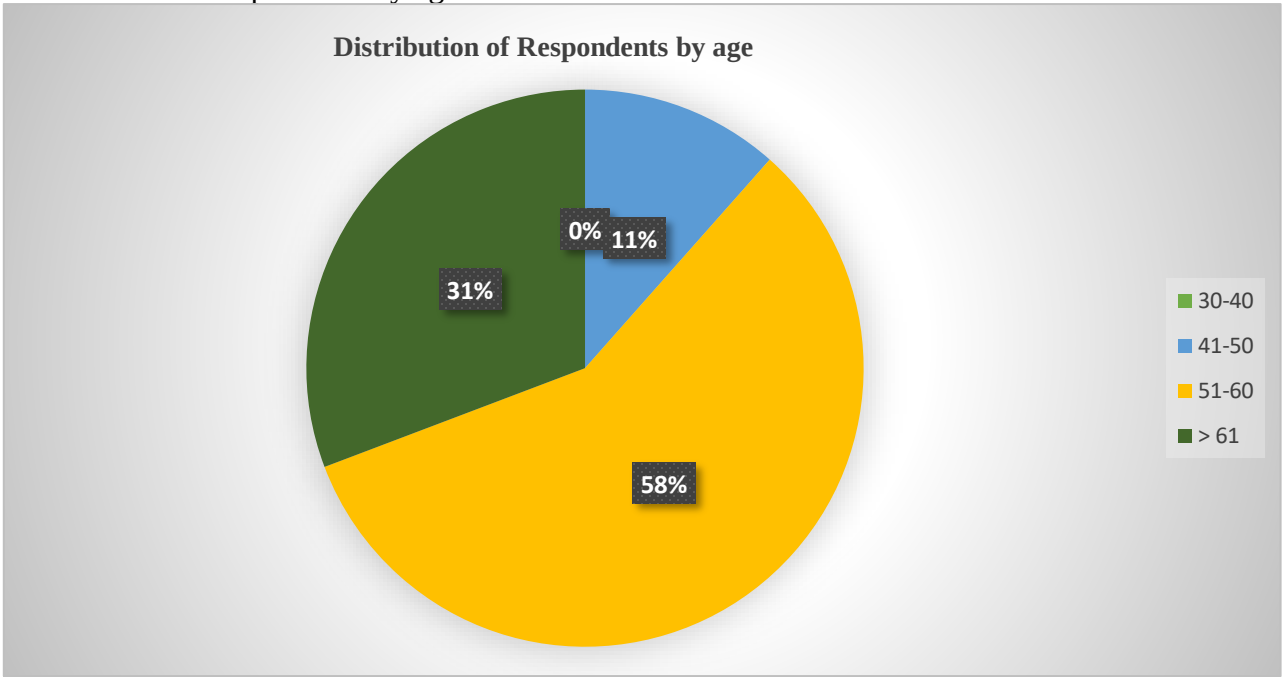
RESULT

Based on the research results, the following data were obtained:

1. Distribution of respondents based on gender



2. Distributions of respondents by age



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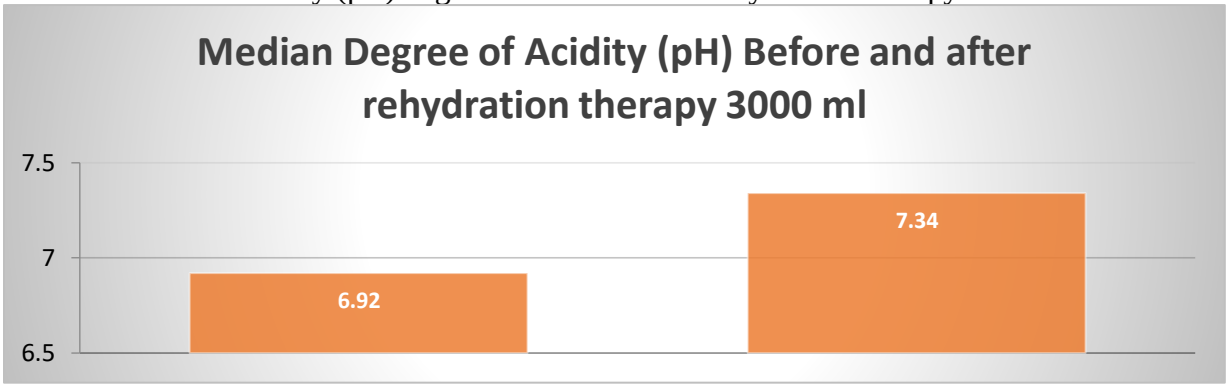
3. Median of acidity (pH) degree before and after rehydration therapy is 3000 ml



4. Analysis overview changes in blood acidity levels in KAD patients who have received rehydration therapy as much as 3000 ml

Table 1.1 Analysis of changes in blood acidity before and after 3000 ml of rehydration therapy in patients with KAD

Variable Type	Treatment	N	Median	Min	Max	P Value
Blood Acidity (pH)	Before rehydration therapy	26	6,92	6,44	7,19	0,000
	After rehydration therapy 3000 ml	26	7,34	7,06	7,48	

DISCUSSION

Changes in blood acid levels of patients who received 3000 ml rehydration therapy were 0.4 with a median blood acid level of 6.92 before therapy and 7.34 after therapy. Acidosis (pH <7.3) is characterized by a decrease in the level of bicarbonate (HCO3-ion) followed by a decrease in the partial pressure of CO2 in the arteries. Under normal conditions, the HCO3-ion content is 24 mEq / L and the PCO2 content is 40 mmHg with an H-ion content of 40 nanomol / L. A decrease in HCO3 levels of 1 mEq / L will certainly be followed by a decrease in PCO2 of 1.2 mmHg (Biff F. Palmer, 2015) (Siperstein, 2018). In the body, HCO3 acts as a buffer system, namely as a buffer when there is an acidosis. HCO3 starts from the hydration of CO2 which dissolves in water and forms carbonic acid (H2CO3). H2CO3 will then dissociate into H-ions and HCO3-ions. In the case of diabetic ketoacidosis (KAD), where there is a significant increase in acid (H-ion) and ketones, the role of HCO3 as a buffer becomes less effective given the acid content is higher than HCO3. This condition results in PCO2 compensation resulting in hyperventilation in the lungs which is characterized by rapid and deep breathing (kussmaul) (English & Williams, 2004)(Kamel S. Kamel, 2015). Apart from the lungs, the kidneys also have an important role to play in compensating for metabolic acidosis (English & Williams, 2004) (Lutfi, 2019). In a state of metabolic acidosis, the body's compensation through the kidneys is by increasing secretion and excretion. In the kidney glomerulus, a filtration process will occur which will excrete acid (H-ions) in the form of urine. The H-ion that is still needed by the body will be reabsorbed back in the proximal tubule and then will combine with the HCO3-ion which is filtered by the glomerulus. With the help of the enzyme carbonic anhydrase, the combined H and HCO3 ions will dissociate into H2O and CO2. Passively, CO2 and H2O will enter the proximal tubule then react with H2O to form HCO3-ions, subsequently enter the

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blood circulation to work again as a buffer system. This situation will run optimally if supported by effective perfusion (Biff F. Palmer, 2015). The presence of a large deficit in KAD patients requires a rapid rehydration effort to improve body fluid balance (Umpierrez, 2018). The main goal of rehydration is to increase blood perfusion to the kidneys in order to maximize the process of excretion of fatty acids from the body by the kidneys in the form of urine (Biff F. Palmer, 2015) (Umpierrez, 2018). Hence after rehydration therapy, it is expected that the acidity of the blood will decrease and blood pH will increase to normal conditions (Haak et al., 2015). Therefore it can be seen that fluids have an important role in the management of diabetic ketoacidosis (KAD) patients (Gosmanov et al., 2014)

In the management of KAD, the organ most expected to play an active role is the kidney (Semarawima, 2017). In human body, one of the roles of the kidneys is to excrete the waste products of the body's metabolism in the form of urine (Diacon, 2012). In contrast to KAD patients, even though the kidneys are healthy they will not be able to excrete normally (Gosmanov et al., 2014). Along with the occurrence of hyperosmolarity, the body will experience dehydration, coupled with a state of metabolic acidosis (increased fatty acids and ketones) in blood plasma (Umpierrez, 2018). The kidneys which are supposed to compensate for metabolic acidosis are obstructed by dehydration (Ekoe, J.M., Goldenberg, R., Katz, 2018). Blood, which is supposed to be perfused into the kidneys and filtered, will not performed it's job. Thus the renal glomerulus prioritizes the dehydration of the body over the excretion of metabolic waste (Gosmanov et al., 2014)

CONCLUSION)

Fluid deficiency conditions found in KAD patients will exacerbate metabolic acidosis conditions due to renal hypoperfusion. Fluid therapy in the acute phase of KAD patients, besides being used as body rehydration therapy, will also provide benefits in the form of increased blood perfusion to the kidneys, which will maximize the process of fatty acid excretion from the body by the kidneys in the form of urine. In consequence, after rehydration therapy, it is expected that the acidity of the blood will decrease and blood pH will increase to normal conditions. Therefore, fluids have an important role in the management of diabetic ketoacidosis patients.

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