STUDY OF METABOLIC ACIDOSIS IN PATIENTS UNDERGOING SURGERIES OF OPERATIVE TIME GREATER THAN 2 HOURS DURATION

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ABSTRACT

BACKGROUND

Metabolic acidosis is proven complication of major surgery, but very less significance is given to it. Metabolic acidosis has a significant effect in postoperative recovery and morbidity of patients undergoing major surgery. Metabolic acidosis has a say in proper functioning of cardiovascular, renal and pulmonary system, added to severe stress full condition related to postoperative period, it bring about major shift in the speedy recovery of patient. It becomes significantly important that metabolic acidosis in diagnosed as early as possible and corrective measures are taken immediately.

MATERIALS AND METHODS

Study design is a prospective observational study. 109 patients who underwent elective and emergency surgeries in the department of General Surgery, Govt. Medical College Kottayam was studied for a period of 3 months (2016). On arrival of the patient, a detailed history of the patient was taken, along with emphasis to the multiple factors in the history which could be contributory to postoperative metabolic acidosis such as diabetic status, drug history, history of respiratory, cardiac and renal status. Basic preoperative laboratory investigation was carried out and its values were recorded. A preoperative arterial blood gas analysis (ABG) of the patient was done before patient was taken for surgery, values of which were recorded and analysed to rule out existing acidotic status of patient, if the patient is already having metabolic acidosis he was excluded from the study. A second ABG was sent at 2 hours after induction of anaesthesia, values of which was recorded, along with the values of intraoperative fluids, preoperative Hb, duration of surgery, type of surgery, blood transfusion and colloid administration given during the time of anaesthesia. A third ABG was sent within six hours of completion of surgery and the values analysed, with due notes on postoperative care done and the days of ICU stay, for analysis and comparison.

RESULTS

Duration of surgery was significantly related to incidence of both intraoperative and postoperative metabolic acidosis. When cross tabulation was done between operative duration and intra operative metabolic acidosis, it was found that 14(87.5%) subjects of total 16 who had duration of greater than 5 hrs had metabolic acidosis. 10 (62.5%) individuals of operative duration 2.01-3 hrs., 28 (73.7%) individuals from 3.01-4 hrs. group and 37 (94.9%) individuals from 4.01-5 hrs duration group were found to have intra operative metabolic acidosis. On doing an independent t test for calculating the significance of each of total crystalloid, normal saline and ringer lactate it was found that ringer lactate had a significant relationship with postoperative metabolic acidosis, indicated by a p value of 0.030, while normal saline had no significant relation to postoperative metabolic acidosis. Still it is interesting to note that total crystalloid administered had a significant relation to postoperative metabolic acidosis indicated by a p value of 0.047. Colloid administration was also found to have a positive relationship with both intra operative (p value of 0.019) and postoperative metabolic acidosis (p value of 0.009).

CONCLUSION

This study stresses the need of doing an intra operative blood gas analysis, in all prolonged surgeries, to analyse and begin corrective measures intra operatively itself, such that patient has least postoperative morbidity and speedy recovery.

KEYWORDS

Colloid, Crystalloid, Duration, Intraoperative metabolic acidosis, Postoperative metabolic acidosis.

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BACKGROUND

Metabolic acidosis is an established complication of major surgeries. It has a direct effect on cardiovascular system resulting in bradycardia and peripheral vasodilatation, which in turn produces a vicious cycle which bring about an accelerated production of substrates such as lactic acid, which in turn increase the acidosis. Hence it should be identified and tackled immediately. Thus, early detection and corrective measures of metabolic acidosis are important steps in the perioperative management of major surgeries. Major cause of perioperative metabolic acidosis, is found to be perioperative fluid administration and lactic acidosis. Large amount of perioperative normal saline administration will produce postoperative dilutional metabolic acidosis. Lactic acidosis is produced by presence of perioperative sepsis or because of very severe blood loss causing lactic academia due to anaerobic respiration. These two being the major causes, certain other factors such as type of surgery, duration of surgery, blood transfusion, haemoglobin level, were also suspected to influence the blood gas value, which are studied here.

Aims and Objectives

To assess the proportion of metabolic acidosis in surgeries of operative time greater than 2 hours and to find out the intraoperative factors that cause it.

MATERIALS AND METHODS

Study design is a prospective observational study, 109 patients who underwent elective and emergency surgeries in the department of General Surgery, Govt. Medical College Kottayam, were studied for a period of 3 months (2016). On arrival of the patient a detailed history of the patient was taken, along with emphasis to the multiple factors in the history which could be contributory to postoperative metabolic acidosis such as diabetic status, drug history and history of respiratory, cardiac and renal status. Basic preoperative laboratory investigation was carried out and its values were recorded. A preoperative arterial blood gas analysis (ABG) of the patient was done before patient was taken for surgery, values of which were recorded and analysed to rule out existing acidotic status of patient, if the patient is already having metabolic acidosis he was excluded from the study.

A second ABG was sent at 2 hours after induction of anaesthesia, values of which was recorded, along with the intra operative fluids, preoperative haemoglobin (Hb), duration of surgery, type of surgery, blood transfusion, colloid administration which were given during the time of anaesthesia.

A third ABG was sent within six hours of completion of surgery and the values analysed, with due notes on postoperative care done and the days of ICU stay, for analysis and comparison.

Inclusion Criteria-

All surgeries of time period greater than 2 hours, in patients 13 years and above.

Exclusion Criteria-

Patients with preexisting acidosis- There were no funding agencies and all the data were collected personally.

Method of blood collection and the factors influencing it-

Blood was usually withdrawn from the radial artery as it is easy to palpate and has a good collateral supply. The patient's arm was placed palm-up on a flat surface, with the wrist dorsiflexed at 45°. A towel was placed under the wrist for support. The puncture site was disinfected with 5% povidone iodine, and a local anaesthetic agent (1% lignocaine) was infiltrated. The radial artery was palpated for a pulse, and a pre-heparinised syringe with a 23 or 25 gauge needle was inserted at an angle just distal to the palpated pulse. A small quantity of blood is sufficient. After the puncture, sterile gauze was placed firmly over the site and direct pressure applied for several minutes to obtain haemostasis. If repeated arterial blood gas analysis was required, it was advised to use a different site (other radial artery or femoral artery) or insert an arterial line. To ensure accuracy, it was important to deliver the sample for analysis promptly. If there was any delay in processing the sample, the blood was stored on ice for approximately 30 minutes with little effect on the accuracy of the results. Complications of arterial puncture were infrequent. They include prolonged bleeding, infection, thrombosis or arteriospasm.

A number of sampling and environmental factors may affect the result of the analysis. Delayed processing of the sample may yield a falsely low PaO_2, as the delay allows leucocytes to consume oxygen. This can be avoided by prompt transport of the sample on ice. Air bubbles introduced when performing the arterial puncture can also cause a falsely high PaO_2 and a falsely low $PaCO_2$. This was avoided by gently removing air bubbles within the specimen immediately after collection without agitating the sample. Body temperature can also affect arterial blood gas tensions. This is relevant in febrile or hypothermic patients.

Factors evaluated in the study

Each of the factors such as anaemia, intensive care unit (ICU) stay, intravenous fluid administration, colloid administration, packed red cell(PRC) and fresh frozen plasma(FFP) transfusion, age, sex, operation type, duration of surgery, were looked for significance with intraoperative or postoperative metabolic acidosis by means of Chi-square tests. Intravenous fluids were evaluated by independent t test for identifying their significance to intraoperative and postoperative metabolic acidosis.

RESULTS

Total of 109 study subjects were included, in whom, the incidence of postoperative and intraoperative metabolic acidosis was evaluated. We also tried to find out if there was any significant relationship between intraoperative factors such as duration of surgery, type of surgery, ICU stay, intravenous fluid administration, colloid administration, PRC and FFP administration and incidence of acidosis.

Most number of subjects belonged to age group of 61-70 yrs., 25.7%, and least one belonged to group 10-20 yrs., 1.8%. 72 were males (66.1%), and rest 37(33.9%) were females. But relationship of age to both postoperative and intra operative metabolic acidosis was found to be not significant.

Operation types were analysed by dividing into 4 groups; head and neck surgery, chest and thoracic wall surgery, abdominal surgery and extremity surgery. 89% (97) belonged to abdominal surgery group, 3.7% (4) to extremity surgery, 5.5% (6) to head and neck surgery and 1.8% (2)thoracic wall surgery .On cross tabulation of type of surgery and intraoperative and postoperative metabolic acidosis it was found that 6(100%) subjects who underwent head and neck surgery, 2(100%) patient who underwent chest wall surgery 78(80.4%) patients who had abdominal surgery and 3(75%) patients with extremity surgery had intraoperative metabolic acidosis whereas 5(83.5%) subjects who underwent head and neck surgery, I (50%) patient who underwent chest wall surgery, 72 (74.2%) patients who had abdominal surgery and 3 (75%) patients with extremity surgery had postoperative metabolic acidosis. But no significant relation could be derived between types of surgery and incidence of intraoperative and postoperative metabolic acidosis.

Duration of surgery was noted as one beginning from the induction of anaesthesia, to when patient is out anaesthesia, and it was recorded beginning from 2 hours after the induction. Study group was divided into 4 groups each belonging to Group 1- 2.01-3 hrs., Group 2- 3.01-4 hrs., Group 3- 4.01-5 hrs., Group 4- >5 hrs. [Fig. 1] Maximum number of patients 39(35.58%) belonged to group 3 which had an operative duration of 4.01-5 hrs., followed by the group 2 which had 38 (34.9%) patients, while group 1 and 4 had 14.7% subjects in each category. [Table 1] Of the total subjects who underwent surgery of operative time more than 2 hrs 81 showed metabolic acidosis, which was about 74.3% of total population, while 28(25.7%) subjects showed no metabolic acidosis.

When cross tabulations were done between operative duration and intraoperative and postoperative metabolic acidosis, it was found that 14(87.5%) subjects of total 16 who had duration of greater than 5 hrs, 10 (62.5%) individuals from 2.01-3 hrs. group, 28(73.7%) individuals from 3.01-4 hrs group and 37(94.9%) individuals from 4.01-5 hrs group had metabolic acidosis. In the case of postoperative metabolic acidosis, 15(93.8%) subjects of total 16 who had duration of greater than 5 hrs, 10(62.5%) individuals of operative duration 2.01-3 hrs, 23(60.5%) individuals from 3.01-4 hrs. group and 33(84.6%) individuals from 4.01-5 hrs duration group had postoperative metabolic acidosis. It was found that relation between operative duration and incidence of intraoperative and postoperative metabolic acidosis was significant (p values of 0.015 and 0.016 respectively).

It was found that 78(71.6%) patients, had an ICU stay of less than 3 days, and 26(23.9%) had ICU stay for 4-6 days, while only 5(4.6%) had ICU stay of greater than 7 days. When ICU stay was cross tabulated with intraoperative and postoperative metabolic acidosis they showed no significant relationship with either intraoperative or postoperative metabolic acidosis.

Subjects were divided into 2, on basis of level of haemoglobin in blood as anaemic and no anaemic. The cut off point for anaemia was kept as 10.9 mg%, which is the WHO defined cut off value for Moderate Anaemia. Of the total 11 patients with postoperative anaemia only 8(72.7%) showed intraoperative metabolic acidosis, and 3(27.3%) showed normal blood gas reports. A total of 11 subjects were found to have postoperative anaemia, of which 7(63.6%) showed postoperative metabolic acidosis and 4 (36.4%) were of normal acidotic status postoperatively. To check the relation of preoperative anaemia, as a causative factor for metabolic acidosis, they were cross tabulated. But no significant relationship between pre-anaemic status or post-anaemic status with incidence of intraoperative or postoperative metabolic acidosis could be derived.

Of the 28 individuals given colloid during their intraoperative period, 27(96.4%) was found to have intraoperative metabolic acidosis, nevertheless 62 (76.5%) individuals who were not given colloids were also found to have intraoperative metabolic acidosis whereas 26(92.9%) was found to have postoperative metabolic acidosis. But 55(67.9%) individuals who were not given colloids were also found to have postoperative metabolic acidosis. Moreover, in all the study subjects who were given colloid, it was noted that volume of colloid was restricted to 500 ml. It was also noted that a significant number of patients who were given colloids had severe degree of metabolic acidosis, than the subjects who were not given the same. Thus colloid administration was found to have a positive relationship with both intraoperative and postoperative metabolic acidosis (p values 0.019 & 0.009 respectively).

PRC transfusion was given to a total of 37 subjects, and 31 of them showed postoperative metabolic acidosis, while 6 subjects showed normal blood gas values in spite of giving intra operative blood transfusion. A significant relation could be derived between PRC administration and intraoperative metabolic acidosis, but such a relation was not found with postoperative metabolic acidosis, which could indicate that blood transfusion can produce metabolic acidosis as immediate effect, which could get corrected by body after some time.

Intraoperative FFP transfusion was cross tabbed with postoperative metabolic acidosis, which showed 19(86.4%) of total 22 subjects who were given FFP intraoperatively had postoperative metabolic acidosis, while 3(13.6%) were without acidosis postoperatively. A definite significant relation was not obtained between FFP transfusion and intraoperative or postoperative metabolic acidosis. But FFP transfusion showed a definite linear trend with intraoperative metabolic acidosis.

On doing an independent t test for calculating the significance of each of the total crystalloids, normal saline and ringer lactate it was found that ringer lactate had a significant relationship with postoperative metabolic acidosis, indicated by a p value of 0.030, while normal saline had no significant relation to postoperative metabolic acidosis. Still it is interesting to note that total crystalloid administered had a significant relation to postoperative metabolic acidosis indicated by a p value of 0.047. [Table 2] It was also found that ringer lactate had a significant relationship with intraoperative acidosis, indicated by a p-value of 0.023, while normal saline and total crystalloid administered had no significant relation to intraoperative metabolic acidosis. [Table. 3]

The mean amount of total crystalloid administered to intraoperative acidotic patients, intraoperatively was 2538 ml, with a standard deviation (SD) of 583 ml, while in non-acidotic individuals it was 2369 ml with SD of 474 ml. Mean normal saline administered was 1836 ml with a SD of 540 ml in intra operative acidotic subjects, while in normal subjects it was about 1809 ml with a SD of 409 ml. Ringer lactate in intra operative acidotic patients was 691 ml administered with SD of 421 ml, while normal subjects were given 560 ml with a SD of 153 ml.

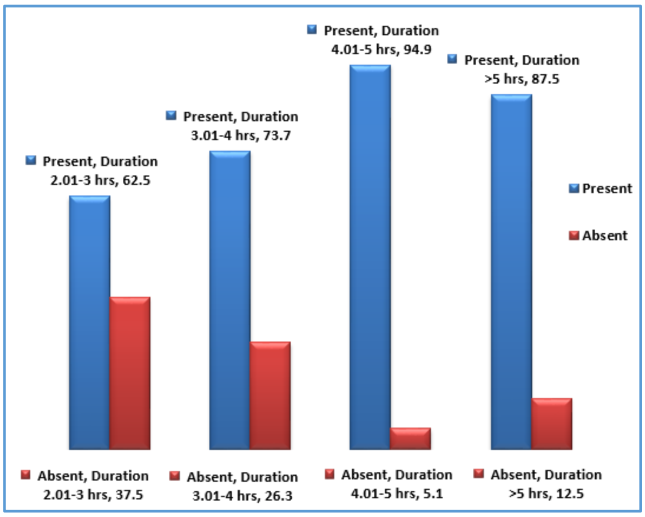

Figure 1. Duration and Intra Operative Metabolic Acidosis

Figure 1- When cross tabulation was done between operative duration and intra operative metabolic acidosis, it was found that 14(87.5%) subjects of total 16 who had duration of greater than 5 hrs had metabolic acidosis. 10(62.5%) individuals of operative duration 2.01-3 hrs., 28(73.7%) individuals from 3.01-4 hrs group and 37(94.9%) individuals from 4.01-5 hrs duration group were found to have intra operative metabolic acidosis. The relation between operative duration and intraoperative metabolic acidosis was found to be significant with p value of 0.015, on doing Chi square test.

Group		**Postoperative Acidosis**		**Total**
		Present	**Absent**	
Duration	2.01-3 hrs	10 62.5%	6 37.5%	16 100%
	3.01-4 hrs	23 60.5%	15 39.5%	38 100%
	4.01-5 hrs	33 84.6%	6 15.4%	39 100%
	>5 hrs	15 93.8%	1 6.2%	16 100%
Total		**81 74.3%**	**28 24.7%**	**109 100%**

Table 1. Duration and Postoperative Acidosis

Table 1- When cross tabulation was done between operative duration and postoperative metabolic acidosis, it was found that 15(93.8%) subjects of total 16 who had duration of greater than 5 hrs had metabolic acidosis. 10(62.5%) individuals of operative duration 2.01-3 hrs, 23(60.5%) individuals from 3.01-4 hrs group and 33(84.6%) individuals from 4.01-5 hrs. duration group were found to have postoperative metabolic acidosis. The relation between operative duration and postoperative metabolic acidosis was found to be significant with p value of 0.016, on doing chi square test.

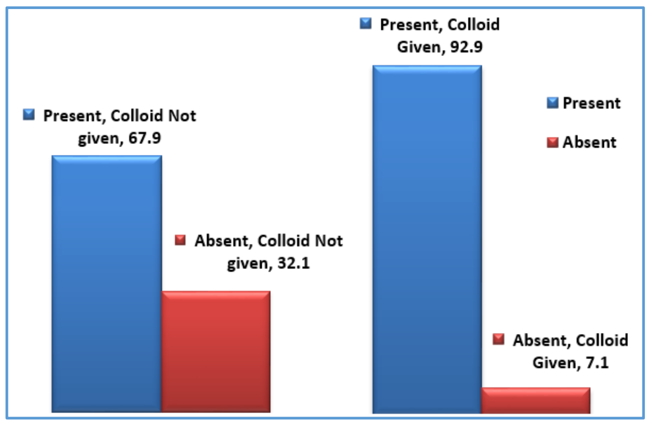

Figure 2. Colloid use and Postoperative Metabolic Acidosis

Figure 2- Of the 28 individuals given colloid during their intraoperative period, 26(92.9%) was found to have postoperative metabolic acidosis, nevertheless 55(67.9%) individuals who were not given colloids were also found to have postoperative metabolic acidosis.

	Levene's Test for Equality of Variances		**t-test for Equality of Means**						
	F	**Sig**	**t**	**df**	**Sig 2**	**Mean difference**	**Std. error**	**95% Confidence Interval of the Difference**	
								Lower	**Upper**
Total Crystalloid	1.063	.305	1.208	107	.230	169.03933	139.99050	-108.47551	446.55416
Normal Saline	2.089	.151	.216	107	.830	27.742	128.517	-227.028	282.512

Ringer Lactate			2.321	85.265	.023	130.624	56.276	18.737	242.510

Table 2. Fluid Administered and Intraoperative Metabolic Acidosis - Independent t test

Table 2- On doing an independent t test for calculating the significance of each of total crystalloid, normal saline and ringer lactate it was found that ringer lactate had a significant relationship, indicated by a p value of 0.023, while normal saline and total crystalloid administered had no significant relation to intra operative metabolic acidosis.

	Levene's Test for Equality of Variances		**t-test for Equality of Means**						
	F	**Sig**	**t**	**df**	**Sig 2**	**Mean difference**	**Std. Error**	**95% Confidence Interval of the Difference**	
								Lower	**Upper**
Total Crystalloid	.801	.373	2.008	107	.047	246.09788	122.57323	3.11075	489.08502
Normal Saline	1.319	.253	.442	107	.659	50.282	113.775	-175.264	275.828
Ringer Lactate			3.190	105.023	.002	184.087	57.707	69.666	298.508

Table 3. Fluid Administered and Postoperative Metabolic Acidosis - Independent t test

Table 3- On doing an independent t test for calculating the significance of each of total crystalloid, normal saline and ringer lactate it was found that ringer lactate had a significant relationship, indicated by a p value of 0.002, while normal saline had no significant relation to postoperative metabolic acidosis. Still it is interesting to note that total crystalloid administered had a significant relation to postoperative metabolic acidosis indicated by a p value of 0.047.

In 1907 the remarkable ability of blood to neutralize large amounts of acid led Lawrence J. Henderson (1878-1942), then an instructor in biochemistry at Harvard University, to investigate the relationship of bicarbonate to dissolved carbon dioxide gas, and how they acted as buffers of fixed acids.[1] Following this lead, in 1917 Hasselbalch adapted Henderson's mass law for carbonic acid to the logarithmic form known as the Henderson-Hasselbalch equation, a staple of contemporary clinical acid-base analysis: pH= pK+log $[HCO_3^-/PCO_2]$.[2] The credit for introducing the term pH goes to S.P.L. Sorensen (1868-1939), who apparently tired of writing seven zeros in a paper on enzyme activity and wanted a simpler designation. While the credit of discovery of H^+ ions and their electromagnetic effects where credited to Van't Hoff (1901), Arrhenius (1903), Ostwald (1909), Nernst (1920), and Heyrovsky (1959), who won Nobel prize for the same in orders of years as given.[3, 4, 5, 6] The first blood glass pH electrode specifically designed to keep carbon dioxide in solution was constructed by Phyllis T. Kerridge (1902-40) in London in 1929.

Finally it was the outbreak of Poliomyelitis during early 1952 in Copenhagen, that paved way for large scale use of ABG analysis, pioneered by work of Dr Bjorn Ibsen, Danish anaesthesiologist, who established that ABG analysis was integral part of critical care, and paved way for positive pressure ventilation as the gold standard of respiratory support, in a failing lung, and ABG analysis helped to decrease mortality from 90% to 25% by the end of epidemic.

D Filipescu, I Raileanu, M Luchian, M Andrei and D Tulbure at Institute of Cardiovascular Diseases, Bucharest, Romania studied one hundred patients who underwent cardiac surgery with cardiopulmonary bypass (CPB)in March 2006. All patients received crystalloid (0.9% saline or Ringer's) and colloid solutions (gelatin), sampling of arterial blood for gas, acid-base parameters and serum electrolytes were performed at four-time points and got the results that sixty-six patients (66%) presented a simple normal-anion gap hyperchloraemic acidosis. Twenty-seven patients had no acidosis.[7]

Waters JH, Miller LR, Clack S, Kim JV at University - affiliated Veteran's Affairs Medical Center studied, Twelve patients undergoing prolonged surgical procedures expected to last >or= 4, the reason of dilutional acidosis as major cause of intra operative metabolic acidosis, than lactic acidosis itself. The largest source of chloride is usually normal saline. The common treatment of administering more fluid for intra operative acidosis may be inappropriate, may have caused the acidosis, and may further exacerbate the acidosis.[8]

Park CM, Chun HK, Jeon K, Suh GY, Choi DW, Kim S conducted a study to evaluate the factors related to post-operative metabolic acidosis and to attempt to identify the clinical effect of metabolic acidosis following major abdominal surgery. Their result showed total infused saline and lactate level were independent factors related to metabolic acidosis. ICU and hospital length of stay were significantly longer in the acidosis group.[9]

Mark NH, Leung JM, Arieff AI, Mangano DT of Veterans Affairs Medical Center (a teaching hospital of the University of California) conducted a prospective, double-blind, randomized trial, to determine the safety and physiologic effects of low-dose sodium bicarbonate in humans who developed intra operative metabolic acidosis in the absence of hypoxemia and they concluded that Administration of sodium bicarbonate to well-oxygenated patients with mild metabolic acidosis resulted in a correction of the acidosis, without significant changes in cardiac output.[10]

Rutherford EJ1, Morris JA Jr, Reed GW, Hall KS tried to determine the association of base deficit with mortality and other factors affecting mortality in consecutive samples of 3791 trauma patients admitted with an arterial blood gas sample taken in the first 24 hours and concluded that the base deficit is an expedient and sensitive measure of both the degree and the duration of inadequate perfusion. It is useful as a clinical tool and enhances the predictive ability of both the Revised Trauma Score and TRISS.[11]

Martin M1, Murray J, Berne T, Demetriades D, Belzberg H conducted a study to determine conventional measures such as anion gap and base deficit can be inadequate for defining and managing complex acid-base derangements and got the result that unmeasured anions are the most common component of metabolic acidosis in trauma intensive care unit patients.[12]

Davis, James W, Kaups, Krista L et al did a study to evaluate the differences in base deficit (BD) clearance, pH normalization, and the occurrence of complications between survivors and non-survivors after trauma. They concluded that Base deficit reveals differences in metabolic acidosis between survivors and nonsurvivors not shown by pH determinations and is clearly a better marker of acidosis clearance after shock.[13]

In our study patients were evaluated for intraoperative and postoperative metabolic acidosis, along with intraoperative factors which were suspected to produce metabolic acidosis. 72 male subjects and 37 female subjects were included in the study. Age, Gender, Type of operation were found to have no influence in metabolic acidotic status of patient. Haemoglobin level both preoperatively and postoperatively also had no relation to incidence of metabolic acidosis. A positive linear correlation was able to be deduced from ICU stay and metabolic acidosis, which means that prolonged ICU stay was closely related to metabolic acidosis of patient. Packed red cell administration was found to produce metabolic acidosis as immediate effect, which could get corrected by body after some time. A definite significant relation was not obtained between FFP transfusion and postoperative and intraoperative metabolic acidosis. But FFP transfusion showed a definite linear trend with intraoperative metabolic acidosis.

Duration of surgery was significantly related to incidence of both intraoperative and postoperative metabolic acidosis. Colloid administration was found to be closely related to incidence of metabolic acidosis. It was noted that colloid was given for patients who had severe metabolic acidosis, such as in trauma. On doing an independent t test for calculating the significance of each of the total crystalloids, normal saline and ringer lactate, it was found that ringer lactate had a significant relationship with intraoperative acidosis, indicated by a p-value of 0.023, while normal saline and total crystalloid administered had no significant relation to intraoperative metabolic acidosis. It was also found that ringer lactate had a significant relationship with postoperative metabolic acidosis, indicated by a p value of 0.002, while normal saline had no significant relation to postoperative metabolic acidosis. Still it is interesting to note that total crystalloid administered had a significant relation to postoperative metabolic acidosis indicated by a p value of 0.047. Thus, ringer lactate was found to have significant relation to both intraoperative and postoperative metabolic acidosis while normal saline was found not related to metabolic acidosis at all. Certain differences were detected in the study from western literature as, normal saline infusion if given injudiciously will produce dilutional metabolic acidosis, which was not found in this study.[8]

Limitation of this study is that the total amount of preoperative fluid administered, and type of fluid given was not quantified as, many of the patients of emergency department would be referred from local hospitals, whose fluid administration data was impossible to obtain. In addition, contributory factors such as hypothermia and anaesthesia drugs were not taken into consideration.

CONCLUSION

This study stresses the need of doing an intraoperative arterial blood gas analysis, in all prolonged surgeries, to analyse and begin corrective measures for the possible metabolic acidosis intra operatively itself, such that patients have the least postoperative morbidity so that they can have a speedy recovery. It becomes significantly important that metabolic acidosis in diagnosed as early as possible and corrective measures are taken immediately. This subject also requires further studies in all the other factors, which can cause metabolic acidosis and their degree of influence in the peri-operative blood gas status.

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