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EFFECT OF SODIUM CITRATE ON GASTRIC JUICE PH. AN ANALYTICAL STUDY

Anesthesiology

Dr Preeti Goyal Professor, Dept. of Anaesthesia , Gajra Raja medical College Gwalior (MP)

Dr Suman Gupta* Associate Professor, Dept. of Anaesthesia , Gajra Raja medical College Gwalior (MP)*Corresponding Author

ABSTRACT

Introduction: Risk of aspiration is increased in patients undergoing elective or emergency surgery during general anaesthesia. The damage to the lung tissue is more when the aspirated content is more acidic in nature. Hence, it's always desirable to increase the pH of Gastric contents before induction of anesthesia.

Methods: Total of 50 patients were enrolled in the study. They were equally divided in two groups of 25 each. Group I received 30 ml of 0.3M sodium citrate solution while group II received 15 ml of Sodium citrate solution. The Gastric pH was measured at repeated intervals, at 0 minutes, 15minutes, 60 minutes and after 120 minutes of sodium citrate administration.

Results: The demographic parameters were comparable in both groups. There was significantly increased pH of Gastric Juice at sequential interval as compared from base line in both the groups. The gastric pH increased significantly from baseline mean ± SD value of 2.17±0.48 and 2.44±3.9 in group I and group II to mean ± SD value 5.3±0.56 and 4.5 ± 0.42 respectively. The effect of sodium citrate was seen till 2hrs duration.

Conclusion: Single dose of Sodium citrate 0.3 Molar solutions was effective in increasing gastric juice pH from the baseline. The 30 ml single dose of sodium citrate was more effective than a single dose of 15 ml.

KEYWORDS

Sodium Citrate, Gastric Juice, Aspiration, Anaesthesia

INTRODUCTION:

The risk of aspiration during general anaesthesia leading to death was recognized more than 100 years back. Regurgitation, vomiting and subsequent aspiration of vomitus into trachea and bronchial tree is a potential complication associated with general anaesthesia.

Medelson et al (1) in 1946 showed that associated exudative edema was a result of irritant effect of acidic aspiration. The role of gastric pH as a causative agent for development of aspiration pneumonitis was suggested by Teabout (2) in 1952 who along with Bannister et al (3) in 1962 suggested that critical pH for sever lung damage in human was 2.5. Robert and Shirley et al (4) in 1974 confirmed their views and added that a volume more than 0.4 ml/kg place the patient at risk of developing acid aspiration syndrome.

Over the years many prophylactic measures have been proposed to decrease the risk of aspiration during general anaesthesia. These includes preoperative starvation, use of anti-emetics and induced vomiting before induction, cuffed endotracheal tube, awake or rapid intubation and the administration of antacid or atropine to neutralize or decrease the gastric contents during anaestheisa . Despite this acid aspiration syndrome continues to pose a threat.

Various methods are used to increase the pH of gastric contents before the induction of anaestheisa other than antacids or pre-operative use of H2 receptor blockers which has become an accepted practice as prophylaxis against acid aspiration syndrome.

The introduction of H2 receptor antagonist like, Cimetidiene, Ranitidine, and Famotidine etc has allowed elevation of gastric pH by inhibition of gastric acid secretion. Proton pump inhibitors like Omeprazole, Pantoprazole etc are other drugs which inhibits the gastric acid production by selectively inhibiting the action of proton pump H+K+ATPase enzyme which exchange luminal potassium for cellular hydrogen ions. Most of the antacids like H2 receptor antagonist or proton pump inhibitors have delayed onset of action hence cannot be used for emergency situations. Particulate antacids if aspirated, can produce harmful physiological and histological changes in the respiratory tract.

Sodium citrate (0.3Molar solution) is a non particulate antacid which is known to increase Gastric pH rapidly without significant side effects even on accidental aspiration. The study was planned to see the effect of single preoperative dose of 0.3 Molar solution of Sodium citrate on gastric Juice pH on patients receiving general anaesthesia.

MATERIALAND METHODS:

This analytical study was carried out at a tertiary care centre in north India. It included total of 50 adult subjects (>18years of age) of either sex of ASA grade I or II, posted for elective or emergency surgical procedure under general anaesthesia. Patients were divided into 2 groups of 25 each. Group I received 30 ml of 0.3 M of sodium citrate by nasogastric tube 15 minutes before the induction of anaesthesia and Group II received 15 ml of0.3M sodium citrate by nasogastric tube 15 minutes before induction of anaesthesia. Patients with history of peptic ulcer disease or history of taking H2 blocker or proton pump inhibitor were excluded from the study.

Thorough pre anaesthetic checkup of these patients were done. All patients were assessed clinically by meticulous general and systemic examination. All patient were pre-medicated with injection atropine 0.02 mg/kg intramuscularly 30 minutes before the induction of anaesthesia. A 16 F nasogastric tube was passed in the stomach. The position of nasogastric tube was confirmed and gastric contents aspirated with the help of 5 ml disposable syringe just before administration of sodium citrate. These cases were induced with sleep dose of 2.5% thiopentone sodium (4-7 ml/kg) followed by apneic dose of suxamethonium IV (11.5 mg/kg body weight). Intermittent positive pressure ventilation with 100% oxygen for two minutes was done. Larynx and trachea were sprayed with 4% lignocaine and trachea was intubated with well lubricated cuffed endotracheal tube. The anaesthesia was maintained by O2 (33%) N2O (67%) , pentazocine and non-depolarizing muscle relaxant with controlled ventilation. All patients were reversed with injection atropine in a dose of 0.02 mg/kg with neostigmine injection in a dose of 0.04-0.05mg/kg intravenously. Gastric samples were aspirated with help of 5 ml disposable syringe at different time intervals and the pH was measured using Toshniwal pH meter before sodium citrate administration, after induction of anaesthesia at 15 minutes and thereafter 60 and 120 minutes later of sodium citrate administration. Vitals including pulse, blood pressure and ECG were monitored throughout the course of anaesthesia. All the observations were recorded in a pre-designed proforma. Appropriate statistical test (paired student t test and paired Annova) were used for the analysis.

RESULTS

Total of 50 subjects were enrolled in the study. Mean ± SD age of patients in group I was 29±10 years and that in the group II was 31±12 years. There is preponderance of female patients in both the groups. (Table 1).

TABLE 1: Demographic profile of the patients.

Variables	Group I(n=25)	Group II (n=25)
Age years	29± 10	31±12
Male	8(32%)	3(12%)
Female	17(68%)	22 (88%)

In group I and II the average pH at 0 minutes, 15 minutes, 60 minutes and 120 minutes are shown in Table 2. The pH in both the groups increased significantly (p value <0.01) after the base line. The difference between the two groups is shown in Table 3. The pH was higher in group I at 15 minutes, 60 minutes and 120 minutes as compared to group II and the difference was highly significant. (Table 3)

TABLE 2: Comparison of Gastric pH at sequential time intervals in both the groups.

Variables	0 minutes	15 minutes after induction	At 60 minutes	At 120 minutes	t value	P value
Group I (n=30)						
Mean Ph	2.17	5.32	5.45	5.19	-24.4	<0.01
SD	0.48	0.56	0.63	0.76		
SE	0.09	0.08	0.13	0.15		
Group II (n= 30)						
Mean Ph	2.44	4.53	4.7	3.86	-21.9	<0.01
SD	0.39	0.42	0.53	0.47		
SE	0.03	0.08	0.10	0.09		

TABLE 3: Comparison of gastric pH in both groups

Variables	Group I Mean ± SD	Group II Mean ± SD	t value	P value
pH at 0 minutes	2.17±0.48	2.44±3.9	2.2	0.57
pH at 15 minutes	5.3±0.56	4.5 ± 0.42	5.6	<0.01
pH 60 minutes	5.4±0.63	4.7 ± 0.53	4.5	<0.01
pH 120 minutes	5.1±0.76	3.8 ± 0.48	7.4	<0.01

DISCUSSION:

The present study showed that administration of 0.3M sodium citrate increases the pH of gastric content effectively which was more so for 30 ml as compared to the 15 ml solution at different time intervals.

Schmidt et al (5) compared the gastric juice Ph after sodium citrate solution administration (group I), water administration (Group II) and sodium citrate and water administration (Group III). They found no statistically significant difference of pH and aspirated volume between the group receiving water and the group receiving sodium citrate.

Atanassoff et al (6) did a study comparing gastric pH in patient receiving variable dose of ranitidine and sodium citrate solution. In group one patient received 300 mg ranitidine + sodium citrate, group II received 150 mg Ranitidine + sodium citrate and third group received only sodium citrate alone. They found that two minutes after administration of ranitidine 300mg and Ranitidine 150mg , a mean (range) gastric pH of 6.8 (5.8-7.5), and 5.6 (1.2-7.0), respectively, was reached, and remained above 2.5 for 14 hr (P = NS). Plain Sodium citrate increased the gastric pH within 2 minutes to a mean (range) of 6.8 (6.7-7.0), and maintained it above 2.5 for 6 hours

Viegas et al (7) measured gastric fluid pH following induction of anesthesia and placement of an endotracheal tube in 30 patients undergoing elective surgery. Fifteen patients who received 15 ml of sodium citrate solution 15 - 20 minutes before induction of anesthesia had a mean pH of 6.2 +/- 0.8. The control group (n= 15) which didn't received sodium citrate, had a mean pH of 2.1 +/- 1.4. Hence they also showed increase in gastric pH noted following sodium citrate similar to our study. The increased pH following sodium citrate administration would result in reduced pulmonary reaction should aspiration occurs.

In a study done by Maltby et al (8), the effect of ranitidine (oral) alone was compared with sequentially administered drugs like ranitidine, metoclopramide, and sodium citrate on gastric fluid volume and pH. The study included 196 healthy, elective surgical inpatients, each of whom was randomly assigned to one of four groups. In all groups mean pH was > 5.8, mean gastric fluid volumes were significantly higher in patients who received citrate than in those who did not. Their study concluded that single drug ranitidine was far more superior to giving triple prophylaxis containing ranitidine, sodium citrate and metoclopramide. We didn't use the ranitidine in our study.

CONCLUSION:

In patients undergoing elective or emergency surgery sodium citrate is effective in increasing the pH of gastric juice. The single dose of 30 ml 0.3M sodium citrate solution was more effective in increasing the gastric juice pH as compared to 15 ml volume. Effects was rapid in onset and was well sustained till 2hrs after the administration.

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