A comparison of sodium citrate and sodium citrate/ranitidine combination for acid aspiration prophylaxis

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Summary

The effectiveness of sodium citrate and sodium citrate/ranitidine were compared in two randomised groups of elective caesarian patients during the various phases of anaesthesia. The mean pH values (3.5, 3.3, 3.6) were lower in the citrate group compared to the citrate/ranitidine group (6.1, 6.3, 5.9). The percentage of patients with pH values less than 2.5 was 40% in the citrate group compared to 7% in the citrate/ranitidine group. Sodium citrate alone is less effective than sodium citrate/ranitidine for acid aspiration prophylaxis.

Key words: acid aspiration prophylaxis; sodium citrate; oral ranitidine.

Introduction

Aspiration of gastric contents¹ still form a formidable cause of maternal death during anaesthesia. The damage to the lungs² is related to the pH of the aspirate. At the present moment, all our obstetric patients for caesarian sections are given 30 mls sodium citrate before general anaesthesia. There has been controversy regarding the effectiveness of sodium citrate alone^{3,4,5} and a growing trend to use a combination of a nonparticulate antacid and a H₂ receptor blocker. We therefore decided to compare the effectiveness of the 2 regimen i.e. 30 mls sodium citrate and sodium citrate combined with ranitidine orally.

Method

The study was approved by the Ethics Committee of University Hospital, Kuala Lumpur. Two series of patients for elective caesarean sections under general anaesthesia were studied. The first series consisted of patients from maternity ward 1 who were given 30 mls of sodium citrate before being sent to the operating theatre.

The second series consisted of patients from maternity ward 2 who were given 150 mg oral ranitidine the night before and 150 mg oral ranitidine on the morning of the procedure together with 30 mls sodium citrate when called to the operating theatre. Admissions to the two wards were random and not biased and hence admissions to the two series were randomised.

After induction and intubation, a size 14/16 nasogastric tube was inserted. 5 - 10 mls of stomach contents were aspirated at 3 different times when the patient was most likely to aspirate, that is:

- (a) around the time of induction and intubation
- (b) around the time of extubation
- (c) in the recovery before full consciousness is regained

The pH of the samples aspirate were determined by a digital pH meter (Jenway model 3070).

The time of drug administration in relation to induction of anaesthesia were recorded.

All the patients in the series were in ASA Class I with no other medication. We excluded all patients with history of gastrointestinal problems.

The following statistical test had been used: t-test; 1-factor analysis of variance (ANOVA) and the x^2 .

Results

The racial distribution were similar in the two series (Table I). The age distribution though significantly different was not considered important as the mean age difference between the 2 groups was 2 years (Table II).

Table I Racial distribution

	Sodium Citrate	Sodium Citrate / Ranitidine
Chinese	4	1
Indian	18	11
Malay	26	30
Total	48	42

 $X^2 = 3.39, p > 0.18$

Table II
Age distribution (in years)

	Sodium Citrate	Sodium Citrate / Ranitidine
Mean	30.4 ± 4.5	32.4 ± 4.9
Range	18 – 38	23 – 43

t = 1.997, p < 0.05

Sodium citrate preoperatively gave a lower mean gastric pH compared to a combination of sodium citrate and ranitidine for the various phases of anaesthesia (Table III).

The pH in any one series did not vary significantly within the group over the period of anaesthesia (Sodium citrate group: F = 0.36, p > 0.05; sodium citrate/ranitidine group: F = 0.56, p > 0.05)

There was significantly a greater proportion of patients with pH lower than 2.5 in the sodium citrate group compared to the sodium citrate/ranitidine group (Table IV)

The proportion of patients with no aspirate of gastric juice was significantly higher in the combination group compared with the sodium citrate group (Table V)

Table III

Mean values of gastric samples during various phase of a general anaesthesia following administration of two different regimen

	Sodium Citrate	Sodium Citrate / Ranitidine	p value
pH ₁ (induction)	3.5 ± 1.9	6.1 ± 1.5	p < 0.05
pH ₂ (extubation)	3.3 ± 1.7	6.3 ± 1.6	p < 0.05
pH ₃ (recovery)	3.6 ± 1.7	5.9 ± 1.3	p < 0.05

Table IV
Percentage of patients with pH values less than 2.5 during the various phases of anaesthesia

	Sodium Citrate	Sodium Citrate / Ranitidine	x²; p value
pH ₁ (induction)	39.0	7.4	6.42; p < 0.05
pH ₂ (extubation)	41.9	6.9	8.72; p < 0.05
pH ₃ (recovery)	37.0	3.0	9.35; p < 0.05

Table V
Percentage of dry aspirate during the various phases of anaesthesia

	Sodium Citrate	Sodium Citrate / Ranitidine	X ² ; p value
pH ₁ (induction)	14.6	35.7	4.91; p < 0.05
pH ₂ (extubation)	10.4	31.0	5.69; p < 0.05
pH ₃ (recovery)	4.2	21.4	5.12; p < 0.05

Discussion

Both gastric pH values and volumes are studied in determining effectiveness of any drug regimen for gastric acid prophylaxis. The usual technique of determining gastric volume is by blind aspiration of stomach contents and this may yield volumes significantly different from true total gastric volumes⁶. As such we decided not to determine the volume of stomach contents in our study but to concentrate our attention on gastric pH values only.

30 mls of sodium citrate is definitely inadequate. About 40% of our citrate group still had pH values less than 2.5. This is consistent with the proportion obtained in Hester's³ study where 37% of his patients on citrate prophylaxis recorded a pH less than 2.5. In his study however he gave only 15 mls of sodium citrate and this has been claimed by Gibbs et al⁵ as responsible for failure to achieve effective prophylaxis. They did a similar study using 30 mls and was able to obtain pH values greater than 2.5 in nearly all their patients.

Ranitidine, a H₂ receptor blocker reduces acidity and volume. In our study, a group has been given a combination of ranitidine and citrate. This has resulted in producing a consistently higher pH with a mean of 6.0 and a lower percentage of patients (7%) with pH less than 2.5. The decreased volume produced by ranitidine is also reflected in our study by the higher percentage of dry aspirate in the combination group compared to the sodium citrate group.

H₂ receptor blockers cannot neutralise acids already found in the stomach but if they are also given the night before (as in our study) is the acidity in the stomach high enough to require neutralisation by antacids? This we probably would have been able to answer had we additionally studied a series of patients given ranitidine alone.

The conclusion from our paper is that sodium citrate alone is inadequate and the addition of ranitidine to the regimen significantly increases the effectiveness of sodium citrate as an agent for acid aspiration prophylaxis.

Acknowledgement

I would like to thank all the staff in my department who have participated in extracting the gastric contents for the study, the staff of the department of Obstetric and Gynaecology who has helped to organise the regimen in the two wards and who patiently allowed the insertion of the naso gastric tubes. A special thanks also to Mr Ong Leong Huat for helping with the statistics and Glaxo for providing the pH meter.

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