# Studies on Shankha Bhasma - II Comparison of Antacid Activity of Marketed Shankha Bhasma with Chewable Antacid Tablet

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Received 20 May 1996
Accepted 21 March 1997

**In the present study, antacid activity of two marketed Shankha bhasma formulations was compared with a chewable antacid tablet. The activity was studied on the basis of acid neutralization capacity, Rosset-Rice test and Reheis test. All the products pass the Preliminary Antacid Test U.S.P. The acid neutralization capacity of the chewable tablet is significantly greater than the bhasmas. Bhasma (300 mg.) causes the initial pH to rise slightly above 5.0 and may cause acid rebound effect, whereas, the chewable tablet has slower rate of reaction confirmed by Reheis test. Bhasmas failed to raise pH to 3.0 in Reheis test. Thus the unitary dose for a marketed bhasma as an antacid is 300 mg was confirmed after its *in vitro* testing.**

SHANKHA bhasma is a widely used Ayurvedic formulation indicated for various conditions such as indigestion, loss of appetite and acidity, and its recommended dose is 300 mg[1]. Different formulations of Shankha bhasma are available in the market. Traditionally, preparation of Shankha Bhasma involves the complex procedures like *Shodhana* and *Marana*. Significance of *Marana* process was studied by Aphale *et al*[2]. The large number of process variables such as rate of heating and *Shodhana* treatment involved in these processes may cause significant variations in the properties of the product obtained from different manufacturers as well as different lots from the same manufacturer. The aim of the present study is to evaluate the antacid activity of marketed Shankha bhasma formulations and its comparison with a chewable antacid tablet. Different *in vitro* methods are reported for evaluation of antacid activity[3], but Rosset - Rice test[4] is widely accepted due to better simulation of *in vivo* conditions.

Two marketed Shankha Bhasma formulations (Product A and Product B) and a Chewable tablet (Product C), were directly purchased from the market. The instruments include magnetic stirrer (Remi India Ltd. Bombay) and pH meter (Toshniwal Instruments Mfg. Pvt. Ltd. 0.01 ± 1 digit) and reagents such as HCl, NaOH and all other chemicals were of analytical grade obtained commercially.

Antacid evaluation of the marketed bhasma was carried out in a similar manner as reported previously[2]. Similarly evaluation of antacid activity was carried out for chewable tablets.[4-7]

Product A and Product B have achieved pH 7.12 and 6.08 respectively, whereas, Product C has achieved pH 3.82 at the end of 10 min. during PAT, and hence these products can be labeled as an antacid and subjected to further evaluation.

The total mEq. of the acid neutralized by two bhasma samples were significantly lower than the chewable tablet (Table I). Rosset- Rice test is a widely accepted *in vitro* method which reflects the efficacy of the dose. Some studies report the rate of addition of acid as 2 ml/min[7]. However, normal acid secretion rate in the stomach is equal to about 4 ml of 0.1 N HCl/min[8], hence, in the present study the rate of addition of acid was kept as 4 ml/min.

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Table I: Comparative antacid activity of the products

| Product | A.N.C. | Rosset-Rice Test | | Reheis test |
|---|---|---|---|---|
| | Total mEq. | Tiem required to reach pH 3 | AUC of pH time plot | Time to reach pH 3 |
| A | 7.804 ± 0.3743 | 6 min | 21.89 | pH < 3.0 |
| B | 8.519 ± 0.0685 | 7 min | 22.43 | pH < 3.0 |
| C | 13.570 ± 1.1511 | 2 min | 06.29 | 13.55 min |

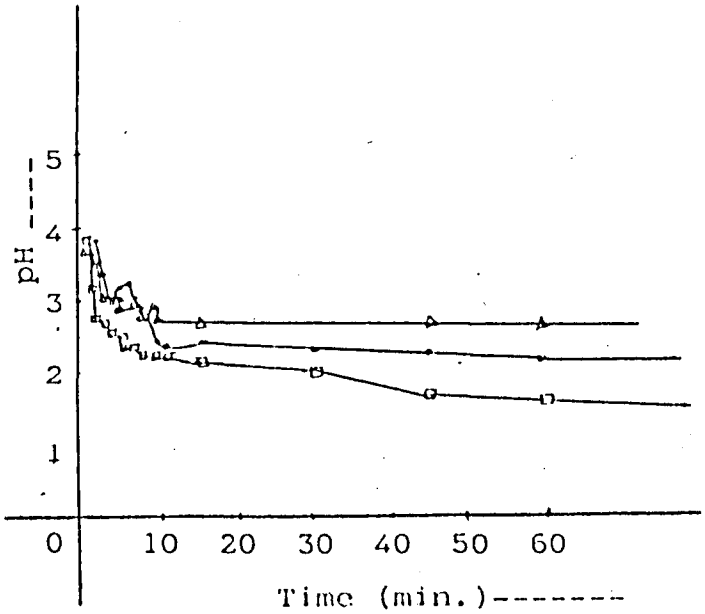


**Fig. 1 Rosset - Rice Test for Product A (•—•)**
**Product B ( Δ — Δ )**
**Product C ( ❑ — ❑ )**

During the test, the pH reached to 3.0 within 10 min. and maintained at that level upto one hour i.e. Rosset-Rice time (time for which pH remains above 3.0) for all the products is greater than one hour. The time required to reach the pH 3.0 is significantly greater for Product A and Product B as compared to Product C. Thus, Product A and Product B have lower acid neutralization capacity as well as rate of neutralization as compared to Product C.

A good antacid should not raise the initial pH above 5.0, which could cause acid rebound effect[7]. In the present study for Shankha Bhasma, 300 mg, which is generally recommended dose was considered as unitary dose for its antacid evaluation. It was observed that, Product A and Product B promptly raised the initial pH slightly greater than 5.0, whereas, Product C shows slow rise in pH. Thus, Product A and Product B have faster onset of action with some risk of acid rebound and Product C has late onset of action. The area under pH time profile (AUC upto pH 3.0) has been considered as a parameter to reflect overall efficiency of an antacid[9]. AUC for bhasmas is significantly higher than the chewable tablet.

Reheis test, which is a reaction velocity test indicates the time required to raise the pH to 3.0 and thereby shows the speed of neutralization of the acid by the drug. Singh et al[6] suggested that the weaker is the neutralizing capacity of an antacid more is the Reheis time. Product C has shown slow rise in pH which reached the level of 3.0 after 13.55 min. whereas, Product A and Product B can not reach the pH 3.0, and have very low acid neutralizing capacity.

Thus, it has been concluded that Product A and Product B have very low antacid activity as compared to Product C. The marketed bhasma samples have shown very low antacid activity as compared to the activity reported for the bhasma prepared by traditional method[2]. Thus the *in vitro* method adopted in the present study may serve as a tool for quality control of Shankha bhasma.

## ACKNOWLEDGEMENTS

The authors wish to thank the University Grants Commission, New Delhi, for sanctioning a minor research project to Kadam S.S.

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# Studies on the Protective Properties of Garlic oil against Acetaminophen-induced hepatotoxicity in the rat

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Received 3 June 1996
Accepted 5 July 1997

**Pretreatment of rats with garlic oil for six days followed by a single intraperitoneal dose of acetaminophen (125 mg/kg) protected against Acetaminophen-induced rise in liver alkaline phosphatase, glutamate oxaloacetate and glutamate pyruvate transaminase (GOT, GPT) activities. It also prevented the formation of thiobarbituric acid reactive substance (TBARS) and depletion of reduced glutathione (GSH) in the livers of acetaminophen treated rats.**

ACETAMINOPHEN is the most commonly used analgesic and antipyretic drug. Higher doses of this drug have been shown to cause hepatic toxicity.[1,2] The hepatotoxicity of aceaminophen is thought to be due to the cytochrome P450IIEI catalysed formation of N- acetyl-p-benzoquinoneimine[3] (NAPQI). The detoxication of this metabolite is brought about by conjugation with reduced glutathione, however, with excess dose of acetaminophen the hepatic GSH stores get depleted with consequent accumulation of free toxic metabolite which binds to tissue macromolecules[4]. Moreover, reduced oxygen species responsible for membrane lipid peroxidation have also been implicated in acetaminophen-induced hepatotoxicity[5].

A synthetic compound, cysteamine in combination with N- acetylcysteine[6] and deferoxamine, an

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