PYRIDOXIN AS AN ANTI-LACTATING AGENT

by

M. Y. Raval,* M.D., D.G.O.
S. K. Shah,** M.D., D.G.O., F.C.P.S.
U. O. Shah,*** M.D., D.G.O., D.F.P.
Vijay Ambiye,† M.D., D.G.O.
and
J. S. Chaddha,†† M.D., D.G.O.

Introduction

The mammary glands are restrained from producing milk during pregnancy by the presence of large amount of oestrogen in the circulation. After parturition, this hormone level falls and this allows lactogenic hormone to act on mammary alveolar epithelium. Lactation also depends on a supply of milk precursors, on stimulas provided by suckling and on the maternal emotional status. But prolactin provides the direct stimulus and is effective only after oestrogen levels have fallen. Prolactin is essential for induction of lactation, but marked personal variation in its level has been found.

Since Kamberi *et al* (1970) observed that prolactin, the hormone primarily responsible for milk secretion is markedly inhibited by increased hypothalamic concentration of dopamine and since Wurtman (1970) showed in animals that brain concentration of dopamine can be increased by administration of its precursor L-dopa, it has been suggested that similar effect might be obtained in human beings. In order to avoid the risk of side effects of this drug, it has been suggested that an increase in hypothalamic content of dopamine could be achieved in lactating women by promoting the natural conversion of DOPA to dopamine through the administration of Pyridoxin.

Material and Methods

A 5 mg. tablet of stilbesterol and a 40 mg. tablet of Pyridoxin was given in a dose of one tablet three times a day to alternate patients who required suppression of breast milk. The tablets were given from 2nd day of delivery so that patients with failure of or deficient milk secretion were excluded. Thus 125 patients received pyridoxine and 125 patients received stilbesterol and a comparative study was made.

There were 2495 deliveries during the trial period and 268 mothers required breast milk suppression. For convenience, only 250 cases are included in the study.

Results

It was found that pyridoxine in a dose of 40 mg. three times a day for 7 days was quite effective in suppressing breast milk secretion. At the same time, toxicity and side effects of stilbesterol were not seen with pyridoxine.

*Professor of Obst. & Gynaec., T.N.M. College, Bombay 400 008.
**Hon. Assoc. Professor, Obst. & Gynaec., T.N.M. College, Bombay 400 008.
***Registrar, Obst. & Gynaec., T.N.M. College, Bombay 400 008.
†Lecturer, L.T.M.C., Bombay 400 022.
††Lecturer, Dept. of Obst. & Gynaec., T.N.M. College, Bombay 400 008.
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TABLE I
Indication

Stillbirths	136
Neonatal death	107
2nd trimester abortion	7
Total	250

TABLE II
Type of Drug Used and Response

Drug used	No. of patients	Response after 7 days		
		Good	Fair	Poor
Pyridoxine	125	112	8	5
Still besterol	125	76	32	17

Good: Patient comfortable, breasts soft, no secretion.
Fair: Patient comfortable, breasts soft, minimal secretion +.
Poor: Patient uncomfortable, breasts nodular, secretion +.

TABLE III

Drug used	No. of patients	Cessation of Laction		
		1 Week	2 Weeks	3 Weeks
Pyridoxine	125	112	125	—
Still-besterol	125	76	45	4

TABLE IV
Side Effect of Drug Used

Drug used	Toxic manifestation				
	Nausea vomiting	Headache	Withdrawal bleeding	Rebound breast filling	Delay in return of monses
Pyridoxine	—	3	—	—	—
Still besterol	8	5	10	26	9

Discussion

Pyridoxine in the form of pyridoxal phosphate serves as a co-enzyme of DOPA—decarboxylase. It may promote the conversion of intraneuronally preformed DOPA to dopamine. Pyridoxine is taken by the hypothalamus probably by diffusion. A rise of pyridoxine may therefore increase the formation of dopamine in neurones and their dandrities. Such an increase in dopamine content in hypothalamus might lead to inhibition of prolaction through stimulation of prolactin inhibiting factor.

Foukas (1973) used pyridoxine as anti-lactating agent and found 100% suppression of milk in first week. But the dose of pyridoxine used was much higher i.e. 200 mg. three times a day.

Summary

Pyridoxine in a dose of 40 mg. three times a day is an effective and economical anti-lactating agent.

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