Tuesday, July 14, 1981

Poster Presentations

Coagulation – VII

Prothrombin, Vitamin K-dependent Factors

11:00–12:30 h

Simcoe Room Boards 149–160

0361

IS WARFARIN SODIUM ABSOLUTELY CONTRAINDICATED DURING LACTATION? R. McKenna, E.R. Cole and U.S. Vasan. Sections of Haematology and Neonatology, Rush Presbyterian-St.Luke's Medical Center, Chicago, IL., U.S.A.

There is widespread concern regarding the secretion of a variety of drugs and toxins into human milk. A survey of the practices of obstetricians and neonatologists at 2 teaching medical centers regarding the appropriateness of allowing a mother on warfarin sodium to breast feed her infant revealed that almost all advised such mothers against breast feeding. This study reports the data on two mother-infant pairs who were evaluated with Prothrombin time activity (Protime), Factor II (FII) and Factor VII/X (FVII) levels simultaneously while the mothers were on warfarin sodium and the infants were exclusively breast fed. Both mothers had extensive serious venous thromboembolic disease necessitating the administration of anticoagulants; warfarin sodium was first started in both mothers in the post-partum period.

The prothrombin time activities on the first mother-infant pair, on blood samples drawn simultaneously are shown below.

Infant's age in days	4	7	28	56
Baby's Protime	Ø	100%	120%	120%
Mother's Protime	46%	20%	30%	18%

Breast milk extracted from this mother on one occasion did not reveal any warfarin sodium by a spectrophotometric assay. Data on the second mother-infant pair on blood samples obtained simultaneously were as follows.

Infant's age in days	6	13	33	131
Infant's Protime	>150%	140%	>150%	>150%
Mother's Protime	30%	27%	27%	32%
Infant's FII	105%	95%	95%	150%
Mother's FII	80%	18%	16%	20%
Infant's FVII	130%	120%	145%	145%
Mother's FVII	34%	12%	23%	18%

These data demonstrate conclusively that there is no inhibition of the coagulation mechanism in normal full term infants when they are breast fed by mothers who are therapeutically anticoagulated with warfarin sodium.

0362

SYNTHESIS AND CATABOLISM OF HUMAN PROTHROMBIN AND PIVKA-II. H.Tsukada, M.Takada, H.Tanaka, T.Takeuchi, H.Gonmori, N.Kobayashi and T.Maekawa. Third Department of Internal Medicine, Gunma University School of Medicine, Maebashi, Japan.

The synthesis and catabolism of both prothrombin and PIVKA-II were studied in patients with stable stage of various thromboembolic disorders. Laboratory examinations revealed that their hemostatic and fibrinolytic activities were within normal range. Thirty mg of warfarin was administered orally to patients, which was followed by daily 4 to 8 mg of warfarin to maintain one stage prothrombin time in therapeutic range. Fifty mg of vitamin K was administered intravenously to these anticoagulated patients. PIVKA-II was determined as prothrombin related antigen in the $BaSO_4$ adsorbed plasma by the method of Ganrot using anti-human prothrombin antibody.

After the administration of 30 mg of warfarin, prothrombin clotting activity(FII:C) decreased exponentialy with the half life(T 1/2) of 74 hrs on an average. Rapid increase of PIVKA-II was observed in the early phase of warfarin administration, though in the later phase an increasing tendency slowed down. The changes of PIVKA-II in the early phase could be analyzed easier by following up the changes in the value of (100 - PIVKA-II activity)%. As the progress of time after the administration of warfarin, their values decreased exponentialy with the T 1/2 of 64 hrs on an average. After the administration of vitamin K, FII:C increased rapidly in the initial phase, then slowly returned to normal level. Similarly to the case of PIVKA-II after the administration of warfarin , values of (100 - FII:C activity)% decreased exponentialy with the T 1/2 of 45 hrs on an average. PIVKA-II decreased rapidly after the administration of vitamin K, describing exponential curve with the T 1/2 of 40 hrs on an average.

From the results described above, it is suggested that the catabolism of PIVKA-II is much more faster than that of FII:C.