venogram revealed no evidence of thrombosis of the subclavian vein or superior vena cava.

Further history revealed that the patient was a student of Kung Fu. Approximately 5 weeks previously, he had learned a new form of blocking that involves a forceful forward thrust of the right forearm while adducting the upper arm in close approximation to the chest wall. He had spent up to 3 hours a day practicing this maneuver for several days prior to the onset of his symptoms. He was treated with ibuprofen and experienced gradual symptomatic improvement over the next several weeks.

Approximately 500 cases of Mondor's disease have been reported in the world literature, but only a small percentage have occurred in men [1,3]. Mondor's disease may occur in association with breast carcinoma or mild trauma, although in some instances no inciting activity or injury can be identified [1,3,4]. Spontaneous resolution without recurrence is the usual outcome [5]. This case reemphasizes the benign nature of Mondor's disease and the importance of a thorough history in determining the etiology of the symptoms.

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Inherited Deficiency of Multiple Vitamin K-Dependent *Coagulation Factors and Coagulation Inhibitors* Presenting as Hemorrhagic Diathesis, Mental Retardation, and Growth Retardation

To the Editor: A 4-year-old male child of asymptomatic parents from a nonconsanguineous marriage in a Hindu Gujrati family presented with repeated spontaneous bruises and bleeding from different sites on different occasions. The patient was a full-term normal delivery with dysmorphic facies. He developed left cerebral hemorrhage at $1\frac{1}{2}$ months and a left thalamic hemorrhage at the age of 6 months. Cerebral hemorrhage was managed conservatively, but he was operated on for the thalamic hemorrhage under high-dose vitamin K and fresh frozen plasma cover. His developmental milestones were retarded and he was totally dependent on his mother for daily activities. There is no family history of excessive bleeding. He has normal liver function, malabsorption tests, fundus examination, and thyroid function; routine biochemical tests were normal. Coagulation tests were done when he was on vitamin K 5 mg orally daily with following results: activated partial thromboplastin time >120 sec (normal, 35 sec); prothrombin time 52 sec (normal, 14 sec); thrombin time 16 sec (normal, 15 sec); factor IXC 3.2%; factor VIII C 105%; factor VII 4.5%; factor X < 1%; factor V 115%; fibrinogen 240 mg/dl; and prothrombin 1.3%. All of these factors were determined by one-stage assay using standard technique. Prothrombin by two-stage assay was 12%. Screening for coagulation inhibitors was negative. Protein C antigen [74% (normal, 70–140 by enzyme-linked immunosorbent assay], protein C [46% by chromogenic assay (normal, 70–140%)], and total protein S [24% (normal 70–140% by enzyme-linked immunosorbent assay)] were found to be low. Levels of these coagulation factors did not change following stoppage of vitamin K for 1 month, and these levels were repeatedly confirmed. Platelet count and function were normal, and other members of the family had absolutely normal coagulation study.

Only seven cases have been reported in the English literature [1–5] with inherited deficiency of multiple vitamin K-dependent coagulation factors, but in only four cases were proteins C or S or both measured [5]. Like the previous studies, our case also demonstrates that reduced levels of proteins C and S do not protect against spontaneous bleeding or serious intracerebral bleeding if multiple clotting factors are substantially reduced. Varying responses to the effects of vitamin K administration have been seen among the reported patients. Most have responded partially and some completely to this therapeutic trial; unfortunately, our patient showed no response.

Mental retardation in the present case could be the result of recurrent cerebral hemorrhage but may also be the result of hypocarboxylation of some important carboxy glutamic acid-containing proteins in the central nervous system during the crucial period of fetal development. Our patient had dysmorphic facies like warfarin embryopathy, but his skeletal survey was not characteristic. In warfarin embryopathy teratogenesis could be particularly due to the warfarin molecule itself, or to consequences of vitamin K deficiency at the cellular level [4]. In our case the problem is pure vitamin K deficiency at the cellular level.

The cause of multiple vitamin K-dependent coagulation factor deficiency in the present case is conjectural. Deficiency of vitamin K epoxide reductase, abnormal vitamin K-dependent carboxylase, or abnormal uptake of vitamin K by liver cells are all possible mechanisms. However, the fact that continuous high-dose vitamin K both orally and parenterally has persistently failed to improve the coagulation factor levels suggests abnormality of vitamin K-dependent carboxylase as the most plausible mechanism of deficiency.

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