



Phenotype and genotype report on homozygous and heterozygous patients with congenital factor X deficiency

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ABSTRACT

Factor X deficiency is a severe rare hemorrhagic condition inherited as an autosomal recessive trait. It is one of the most severe recessive inherited coagulation disorders. We analyzed the clinical manifestations, laboratory phenotype and genotype in 10 patients with severe Factor X deficiency and in their heterozygous relatives. The most frequent bleeding episodes were hematomas (70%) and gum bleeding (60%). Fifty percent of the homozygous patients required blood transfusion and one-third of heterozygotes required treatment after surgery or delivery. The genetic characterization revealed six different missense mutations, two of which were novel: p.Glu694Lys and p.Asp103His. Haplotype analysis, performed with intra- and extra- *FX* gene polymorphic markers in Indian, Iranian and Italian patients with the same mutations failed to establish identity by descent, despite the same Caucasian origin. In conclusion, factor X deficiency was confirmed to be one of the most serious among rare bleeding disorders and genetically heterogeneous in different populations.

Key words: bleeding disorders, bleeding symptoms, factor X deficiency, *F10*, phenotype-genotype.

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Introduction

Factor X (FX), a vitamin K dependent plasma glycoprotein, plays a pivotal role in the coagulation cascade being the first enzyme in the common pathway of thrombin formation. FX is synthesized by the liver and circulates in plasma at a concentration of 8-10 µg as a two-chain protein with a 17-kDa light chain linked to a 45-kDa heavy chain.¹ The light chain contains a γ-carboxyglutamic acid (Gla)-domain necessary for a Ca²⁺-dependent conformational change associated with phospholipid binding, and two epidermal growth factor (EGF) domains. The heavy chain contains the catalytic serine protease domain, structurally homologous to that of other coagulation serine proteases.² FX is activated (FXa) by both factor VIIIa/tissue factor and factor VIIIa/factor IXa. In turn, FXa, which forms the prothrombinase complex together with factor Va, catalyses thrombin formation.³ The *F10* gene (*F10*), located on chromosome 13q34, spans 27kb and contains eight exons, each of which encodes a specific protein domain.^{4,5} To date, approximately 95 variants, compris-

ing deletions, missense, frame shift and splice site mutations have been reported in *F10*.^{6,7} FX deficiency is a rare hemorrhagic disorder, inherited as an autosomal recessive trait with a reported incidence of approximately 1:10⁶ in the general population. It is more common in populations with a high rate of consanguineous marriages, with an 8 to 10 fold increase in frequency.⁸ The disorder is diagnosed by a concomitant prolongation of the prothrombin time (PT) and activated partial thromboplastin time (APTT), and by the low level of FX activity. Its clinical presentation makes it among the most severe of rare coagulation defects, and typically includes hemarthroses, muscle hematomas, umbilical cord bleeding, gastrointestinal and central nervous system (CNS) bleeding.^{9,10} In this study we evaluated the severity of bleeding symptoms and the relationship between genotype and laboratory phenotype in 10 patients from 7 unrelated Iranian families not included in previous studies. Mutation analysis identified six missense mutations: four previously described and two novel, one located in the (Gla)-domain and one in the EGF1 domain. A haplotype analysis was also

MK and MM contributed equally to this work.

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