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increased production of interleukin 1β that have a proconvulsivant effect in patients with FS[3]. Also, the high prevalence of family history of FS and occurrence of FS in homozygous twins suggest that genetic factors may play an important role in

a major proportion of patients in the pediatric group[5]. Unfortunately, individuals with epilepsy are at a higher risk of death than those from the general population and each year, about 1:500 to 1:1000 patients with chronic epilepsy will die sud-

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Mutation in potassium channel genes (KCNQ1, KCNH2, KCNE1 and KCNE2) and sodium channel genes (SCN5A) has been related to neonatal seizures and long QT syndrome[24] and over one third of referred cases of SUDEP were found to harbor a genetic arrhythmia-susceptibility mutation[20,25]. Physiologic changes of mutated channels causing seizures or cardiac arrhythmias are similar[25]. Also, FS is currently observed in patients with epileptic syndromes associated with genetic mutations in

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