Variants in SCN2A, encoding the voltage-gated sodium channel Nav1.2, are commonly associated with developmental and epileptic encephalopathy. Although animal studies demonstrated a role for Nav1.2 in intraventricular conduction, heart anomalies have been only occasionally described in patients with SCN2A variants. In this report we trace the prenatal and neonatal history of a fetus/newborn with a de novo pathogenic variant in the SCN2A gene identified by prenatal trio whole-exome sequencing (WES). In addition to more typically SCN2A-associated neurological manifestations, the patient showed sustained tachyarrhythmia, potentially expanding the phenotypic spectrum associated with SCN2A variants and raising the question of whether cardiological assessment and prompt pharmacological intervention in SCN2A channelopathies to avoid heart complications might be beneficial. To the best of our knowledge, this represents the first clinical description of a SCN2A phenotype in a prenatal setting, as well as the first SCN2A diagnosis achieved by prenatal trio-WES approach.