



A pregnant woman with Brugada Syndrome: the point of view of cardiologist, anaesthesiologist and perinatologist

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Abstract

Objective: The aim of this article was to present a point of view of cardiologist, anaesthesiologist and perinatologist on the case of Brugada Syndrome in pregnant woman.

Case(s): We present a case of a patient with an implantable cardioverter-defibrillator (ICD) due to diagnosed Brugada syndrome, who at the age of 41 years became pregnant for the fourth time. Below are the comments of doctors involved in the care of this patient. They all agree that the key to success in unusual clinical situations, such as the one described in this paper, is close cooperation, detailed knowledge of the clinical problem and good communication.

Conclusion: Summarizing the considerations of doctors from three different specialties, a common conclusion emerges (which is consistent with the literature data) that close cooperation is necessary for the broadly defined good of the patient. This seems to be the optimal approach to every medical problem we have to face, regardless of its scale, the level of understanding of underlying mechanisms and awareness of what is still unexplained.

Keywords: Pregnancy, Brugada Syndrome

Introduction

Brugada Syndrome (BrS) is a rare, genetically determined disease manifested by the occurrence of life-threatening ventricular arrhythmias in people without structural changes in the myocardium. The prevalence of BrS is 1/1000 to 1/10000 and is eight times higher in men than in women.^[1,2] The highest prevalence is observed among the inhabitants of Southeast Asia, where it is the main cause of sudden deaths in young healthy men.^[3,4] The first symptoms most often appear between the 3rd and 5th decade of life, but the disease may appear at any age. Brugada syndrome is inherited in an autosomal dominant manner with incomplete penetrance. More than 12 genes associated with BrS have been discovered, but mutations in only two of them (SCN5A) were confirmed in more than 5% of patients with the identified pathogenic genotype.^[5]

A pregnant patient with Brugada syndrome is a challenge for the anaesthesiologist due to the possibility of fatal ventricular arrhythmias. A patient in the perinatal period may potentially require anaesthesia, either in the form of labour analgesia, anaesthesia for a caesarean section, or anaesthesia for postpartum obstetric procedures. In order for the patient and the medical team to be best prepared for management during childbirth, good cooperation between the gynaecologist, anaesthesiologist and cardiologist is necessary during pregnancy and in the perinatal period.^[6,7] During pregnancy, the patient should be referred by the obstetrician who is supervising her pregnancy to a cardiologist to assess cardiac performance, identify potential problems during delivery, ensure that heart function is adequately controlled before delivery, determine the scope of necessary supervision during delivery

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and specialist monitoring during possible anaesthesia. Then, well in advance of the planned date of delivery, the patient should be referred to a consultation with an anaesthesiologist at the hospital where the delivery is to take place.

Case(s)

We present a case of a patient with an implantable cardioverter-defibrillator (ICD) due to diagnosed Brugada syndrome, who at the age of 41 years became pregnant for the fourth time. A perinatologist, an anaesthesiologist and a cardiologist were involved in the patient's care during pregnancy and delivery. Throughout this time, the patient, aware of the seriousness of the diagnosed heart disease, cooperated with the doctors and followed all recommendations. She also expressed her conscious desire to deliver the child vaginally due to the lower risk of complications, mainly related to anaesthesia, compared to a caesarean section. The patient's pregnancy, which occurred at the age of 41, was not planned. The obstetric history at that time included one missed miscarriage in the 12th week with subsequent diagnostic abrasion of the uterine cavity; two vaginal deliveries: the first delivery in the 40th week — a female newborn weighing 3730g, born in good condition; the second delivery in the 37th week due to developing hypertension and generalized oedema — a female newborn weighing 3590 g, born in good condition. Both pregnancies were complicated by pre-existing hypothyroidism in the course of Hashimoto disease, treated with L-thyroxine – in all pregnancies the patient's thyroid gland was euthyroid.

The course of the pregnancy described in this study was normal. The patient came for her first visit at 7.5 weeks. She was then assessed by a cardiologist who obtained information about the proper operation of the ICD – the device did not record any abnormal heart rhythms. Acetylsalicylic acid was used to prevent preeclampsia. The risk of genetic disorders was estimated to be low by prenatal testing; the patient did not want to extend the diagnostics to include invasive tests. After the 24th week of gestation, the patient was diagnosed with gestational diabetes, which was successfully controlled with diet. Blood

pressure was normal throughout the pregnancy and the patient did not require pharmacological treatment. Between the 31st and 35th week of gestation, a slight increase in amniotic fluid volume was observed (AFI 23 cm); no intervention was implemented for this reason. The course of intrauterine growth of the fetus was regularly assessed, but given the reassuring information from the cardiologist regarding the course of the underlying disease, no disturbances in maternal-fetal blood flow were expected and observed. Throughout the entire pregnancy, special attention was paid to the patient's proper hydration, maintaining electrolyte balance and adequate glycaemia, as well as urgent treatment of any febrile conditions. A patient was assessed by an anaesthesiologist in the third trimester of pregnancy. During the consultation, the anaesthesiologist reviewed the all medical records and laboratory test results, determined the BMI (which was >35), obtained all necessary information about the ICD implanted 2 years earlier and a certificate about the proper functioning of the device. The anaesthesiologist also reviewed the patient's medical history and information about the anaesthesia she received previously. Thanks to the anaesthesiologist's consultation carried out in advance, it was possible to complete the documentation, order some laboratory tests and collect additional data about the disease. The purpose of this planned consultation was also to discuss with the patient the possibility of anaesthesia during and after delivery. Before Brugada syndrome was diagnosed in our patient, she underwent spinal anaesthesia with 0.5% bupivacaine spinal heavy; bupivacaine was also used for epidural analgesia of her previous delivery, which was uneventful. Heart disease in the form of Brugada syndrome was diagnosed in our patient 2 years before her last pregnancy. The first manifestation of the disease was the patient's loss of consciousness for approximately 1–2 minutes, in a sitting position, without prodromal symptoms preceding syncope, without chest pain or heart palpitations, as well as without symptoms after fainting. On the 4th day after the event, the patient went to hospital, where due to family history of sudden cardiac deaths (father's mother, aged 46, and father's two brothers, aged 38 and

42) and comorbidities (Hashimoto's disease – the patient was receiving hormonal replacement; migraine headaches – on prophylactic treatment with duloxetine), a highly sensitive troponin T (hsTnT) test was performed, obtaining a result of 255 ng/mL (normal range: 0–3 ng/mL). The patient was admitted to the cardiology department for further diagnostics. The biochemical tests performed did not reveal any results beyond the normal range, and there was no increase in CK-MB and CPK. The echocardiographic examination revealed no abnormalities – no local contractility disorders were observed, the morphology and function of valves and large vessels remained normal, and there were no signs of pulmonary hypertension or changes in the pericardium. Holter monitoring performed several times did not register any arrhythmia or A-V or intraventricular conduction disturbances; QTc was normal and sinus rhythm was regular at 70 beats/min. No embolic material was observed in the pulmonary arteries in chest CT angiogram. Cardiac MRI was normal. Due to the high level of hsTnT and family history, coronary angiography was performed, which revealed no atherosclerotic changes in the coronary arteries. MRI of the head and EEG were also performed, which did not reveal any pathology. The neurological examination also did not show any abnormalities. Taking into account the high level hsTnT, the family history of sudden cardiac death and the lack of abnormalities in other tests, the ajmaline challenge was performed. During this provocation test, the patient reported dizziness and tongue tingling, and the ECG showed an elevation of the ST segment of 2 mm in V1-V2, typical of Brugada syndrome. Due to the fact that the cause of the syncope was not documented (probably arrhythmic), the patient was initially implanted with an event recorder, but after a thorough analysis after approximately 3 weeks, the event recorder was removed, and as a primary prevention of sudden cardiac death, a cardioverter-defibrillator was implanted with antibiotic prophylaxis. The patient remains under the care of the cardiac electrostimulation clinic. The patient was hospitalized as planned after the 38th week of gestation.

Labor spontaneously occurred after a week, in the late evening, starting with uterine muscle contractions. The obstetrician supervising the pregnancy and the anaesthesiologist who had previously consulted the patient came to the hospital. The cardiologist was on call. After analysing the options for possible anaesthesia, the consulting anaesthesiologist defined in writing the rules for monitoring and managing the patient after admission to the delivery room, including informing the anaesthesiologist on duty that the woman giving birth has Brugada syndrome. After the patient was admitted to the delivery room, the levels of potassium, magnesium, calcium, sodium and chlorine ions in the serum were determined. During delivery, the patient was hydrated by intravenous infusion of Ringer's solution, and the potassium level was maintained within the normal range by intravenous administration of 20 meqv of potassium in Ringer's solution. During delivery, the patient's superficial body temperature was checked several times; it was stable and remained within the range of 36.6–36.7 °C. On preparing, before delivery, we placed a defibrillator with cardioversion and external pacing function in the delivery room.

The patient gave birth with her husband in a private delivery room equipped for this occasion with life-saving equipment. The location of the magnet, which was supposed to be used in the event of a heart rate >190/min, was known to all medical staff present at the birth. After preparing the patient for delivery and observing contractions for an hour, the attending physician decided to administer 5 units of oxytocin using an infusion pump. Then, when the cervix was dilated to 6 cm, it was decided to perform amniocentesis, intensifying contractions; clear amniotic fluid drained. During labour, the patient received a 50/50 mixture of nitrous oxide and oxygen inhaled for 2 hours 15 minutes, at her request. She did not express the need for epidural analgesia. Our patient did not experience any cardiac arrhythmias or tachycardia during labour, requiring the ICD to be temporarily turned off. Due to the fact that the patient should be able to move during labour and that our patient did not require labour anaesthesia,

continuous ECG monitoring was ultimately abandoned, leaving a pulse oximeter to continuously monitor cardiac function and oxygen saturation. The ECG monitor was ready for use in the delivery room near the patient all the time. Blood pressure was periodically checked and ranged from 125/70 mmHg to 140/80 mmHg. The first stage of labour lasted 3 hours and 5 minutes, the patient had normal contractions and after 10 minutes of the second stage she gave birth to a healthy female newborn, weighing 3280 g, in good condition. At the end of the second stage of labour, contrary to the expected intrapartum tachycardia, we observed a tendency to bradycardia reaching 46–48 bpm in our patient.

Discussion

The basis for the diagnosis of Brugada Syndrome is the presence of typical ECG changes (the so-called Brugada pattern) and clinical symptoms in the form of syncope caused by fast polymorphic ventricular tachycardia (VT). Brugada syndrome may also manifest itself as sudden cardiac arrest or death resulting from the transformation of ventricular tachycardia into ventricular fibrillation (VF). There are no symptoms between VT episodes. Approximately 15–30% of BrS patients may experience episodes of atrial fibrillation. Factors that may trigger tachycardia in the course of BrS include high fever, alcohol, a large meal, and some medications. Ventricular fibrillation in patients with Brugada syndrome most often occurs at night or during rest. Current European Society of Cardiology Guidelines for the management of patients with ventricular arrhythmias and the prevention of sudden cardiac death (2022) define the criteria for the diagnosis of Brugada syndrome as the presence of J point elevation of ≥ 2 mm with coved ST elevation in at least one right precordial ECG lead, V1 or V2, positioned in the 2nd, 3rd or 4th intercostal space, where ST elevation is followed by a T wave inversion. These changes may occur spontaneously or after the use of a sodium channel blocker, such as ajmaline, flecainide or procainamide (class of recommendations I, level of evidence C).^[8] Less specific for BrS is J point elevation of ≥ 2 mm

with a saddleback ST segment elevation of ≥ 1 mm and a positive or biphasic T wave (type 2) or J point elevation ≥ 2 mm with ST elevation < 1 mm (type 3). In these cases, Brugada syndrome is diagnosed when type 2 or 3 changes transform into a type 1 electrocardiographic pattern after the use of a class I antiarrhythmic drug. The only treatment that can reduce the risk of sudden cardiac death (SCD) is the cardioverter defibrillator (ICD), and therefore its implantation is recommended in patients with a documented episode of VF or VT (class of recommendation I, level of evidence C). Such treatment should also be used in patients with spontaneous type 1 ECG with a history of concomitant syncope (class of recommendations IIa, level of evidence C).^[9,10]

More and more often in everyday medical practice we encounter pregnant women with specific health problems. Paradoxically, the fact that we have a sick patient, but with a diagnosis already made, seems to be good news, because we are not faced with a situation where the first symptoms of the disease appear during pregnancy, complicating its natural course. In the context of supervising pregnancy and physiological delivery in a woman with heart disease, one should take into account the physiological adaptive changes in the pregnant woman's circulatory system, which in the case of cardiac disease may lead to heart overload. In the period from approximately the 30th to the 32nd week of pregnancy, when the volume of the pregnant woman's plasma reaches its highest value, and during labour, especially its second stage, the patient requires vigilant medical attention and possibly intervention to protect against cardiac complications. In the pre-induction of labour, the use of mechanical methods leading to the desired changes in the cervix (Foley catheter) is preferred. Prostaglandins are considered potentially arrhythmogenic and likely to induce coronary vasospasm. Importantly, dinoprostone has a stronger effect on the increase in blood pressure than misoprostol, so it is contraindicated in pregnant women with cardiovascular disease.^[11] During labour, the fetal head should be allowed to reach the pelvic floor without additional pushing, thus avoiding the Valsalva effect,

which has an adverse effect on the circulatory system. As pointed out by Rodriguez-Mañero et al.^[12], serious events during pregnancy and childbirth do not occur more often in the group of patients with Brugada syndrome compared to the general population. The authors analysed a group of 104 women with this diagnosis and a total of 219 deliveries. There were 5 women who gave birth four times, 2 who gave birth 5 times and one pregnant woman who gave birth 6 times. In the analysed group, 13 patients underwent caesarean section, and the remaining deliveries took place vaginally. The authors emphasize the need to conduct further research to confirm or exclude the role of Brugada syndrome in the pathogenesis of miscarriages and sudden infant deaths – in the analysed group, one infant death within 3 months of delivery and 15 spontaneous miscarriages were reported. In 6 women, the course of pregnancy was complicated by syncope, in three women an ICD was implanted before pregnancy, but in none of them — like in our patient — cardiac arrhythmias were observed. The case of a pregnant woman with Brugada syndrome described by Giambanco et al. was consistent with our observations: the course of pregnancy was uncomplicated, as was the vaginal delivery. Nevertheless, the authors conclude that in such clinical situation, close cooperation between a cardiologist, anaesthesiologist and the obstetric team is extremely important.^[7,13]

The reason for the manifestation of symptoms in the patient's 42nd year of life remains unknown, although the age range between the 3rd and 4th decade of life is quite typical for the appearance of the first symptoms of Brugada syndrome. It is believed that the causes leading to symptom occurrence in patients with the SNC5A gene mutation (hereditary or recently acquired) include physical exercise, fever and pregnancy. Each of the above-mentioned situations may lead to the so-called electrical storm resulting in ventricular arrhythmias.^[7] When managing the pregnancy of a patient with broadly defined heart disease, one should remember that the symptoms may worsen or reappear during pregnancy, even if the patient was asymptomatic before. Physi-

ological adaptation processes of the pregnant woman's circulatory system, the role of which is to ensure the safe development of pregnancy and childbirth, include: increased circulating blood volume, acceleration of heart rate by approximately 10–30 bpm, chronic hypercoagulable state associated with, among others, increased production of vitamin K-dependent coagulation factors and decreased protein S level. On the other hand, the above changes in haemodynamics, the autonomic system and the hormonal changes caused by pregnancy may generate or intensify pre-existing cardiac arrhythmias or palpitations often described by patients.^[14] In the context of potentially fatal ventricular tachycardias or ventricular fibrillation, it is believed that pregnant women with a positive history of these events are at a significantly higher risk of their recurrence during pregnancy, reaching 27%, compared to the general population. Risk factors include: primary electrical heart disease (i.e. long QT syndrome, Brugada syndrome), hypertrophic cardiomyopathies, valvular heart diseases and others.^[14] ICD implantation is a procedure that protects patients with Brugada syndrome against sudden cardiac death; the causal treatment remains unknown, which applies to both pregnant and non-pregnant populations. A multicentre retrospective study of 44 pregnant women with an implanted ICD confirmed the safety of its use for the mother and fetus (no complications in 82% of respondents). However, 8 patients (18%) experienced complications related to the implanted device or the course of the disease itself, i.e. stroke, pulmonary embolism or congestive heart failure.^[15] On the other hand, a retrospective Japanese study conducted in 2012 assessing the safety of the ICD in pregnancy did not show any complications related to its presence – like in our patient, no discharges were observed, and the condition of the patient and the fetus remained stable throughout pregnancy, delivery and the postpartum period mentioned above, Brugada syndrome is much more common in men.^[16] The reasons for these differences have not yet been clearly explained. Postulated pathomechanisms include sex-rela-

ted differences in cellular ion currents. A possible explanation may also be the different effects of male and female sex hormones. Experimental studies conducted by Di Diego et al. proved that the intracellular Ito potassium current is stronger in men, which may predispose them to a more frequent occurrence of clinical symptoms of BrS.^[17] In addition, Matsuo et al. observed the disappearance of typical ECG changes in patients with Brugada syndrome after surgical castration due to prostate cancer. There are also studies showing that testosterone levels are higher in patients with BrS than in the control group.^[18,19] Sex hormones can also increase their impact by modifying ion currents – oestrogens reduce the expression of the Kv4.3 gene, leading to a decrease in the intracellular Ito current, whereas testosterone increases the extracellular IKr, Iks, IK1 currents and reduces the intracellular Ica-L current.^[19-21] Hormonal changes that occur during pregnancy make this a special situation for women with Brugada syndrome. Sharif-Kazemi et al. presented the case of a pregnant woman with BrS, the first manifestation of which was an electrical storm, proving that hormonal changes were the factor triggering the arrhythmia.^[22] Rodriguez-Mañero et al. conducted a retrospective analysis of a population of 104 women diagnosed with Brugada syndrome.^[12] The characteristics of the study group are as follows: over a 17-year period, there were 219 deliveries, 10 women experienced 15 spontaneous miscarriages, 59 women remained asymptomatic at the time of diagnosis, 24 had a history of syncope, and in 4 women the first symptom was sudden cardiac arrest with successful resuscitation. Sixty-six women had a positive family history of Brugada syndrome, and 27 women, like our patient, had an ICD implanted. Among these patients, no cardioverter intervention was recorded during pregnancy. Follow-up data reveal that adequate ICD shocks occurred in 2 patients, non-sustained ventricular tachycardia was recorded in the device's memory in 1 patient, and in another 7 patients, inadequate interventions occurred due to lead damage or atrial fibrillation with rapid ventricular rate. The course of pregnancy in patients with an

implanted ICD was also described by Natale et al.^[23] The authors did not find an increased incidence of complications related to therapy or device during pregnancy, and the number of ICD interventions remained at a level similar to that in the preconception period. In the study cited above by Rodriguez-Mañero et al., 6 patients experienced recurrent syncope episodes during pregnancy. However, the perinatal period in these patients remained asymptomatic. During an average follow-up of 3 years, 97.6% of patients remained symptom-free. In patients with BrS, syncope is one of the most important risk factors. However, it should be noted that this is a highly non-specific symptom. The causes of loss of consciousness include: changes in the tone of the autonomic nervous system, sinus bradycardia, atrioventricular conduction disorders, ventricular and supraventricular arrhythmias, orthostatic hypotension, and, finally, quite common situational syncope. During pregnancy, fainting, apart from dizziness, palpitations and pre-syncope, are one of the most frequently reported ailments.^[24] It remains an open question whether, and if so, when to perform genetic testing in children born to mothers with BrS. In the group analysed by Spanish authors, one infant born to a mother with BrS died suddenly at the age of 3 months.^[12] There are also case reports of even two-day-old newborns with episodes of tachycardia resulting from BrS, as well as sudden infant death syndrome (SIDS) in infants who were, as it later turned out, heterozygous carriers of the SCN5A mutation.^[25,26] As in adult patients, clinical diagnosis in children, including newborns, is made based on characteristic findings in electrocardiography. A major difficulty when recording ECG in this group of patients is the correct placement of the right precordial leads due to the different shape of the chest of the growing organism. In the population of the youngest patients, fever remains the main factor predisposing to the occurrence of symptoms. The authors of a study analysing a group of children with BrS recommend the use of antipyretic drugs preventively after vaccinations and in the case of diseases that may involve high body temperature.^[25]

Genetic testing of family members of a BrS patient with an identified mutation is performed regardless of age. This allows for the implementation of preventive measures to reduce the risk of symptoms caused by fever, alcohol consumption and taking medications that may cause arrhythmia (www.brugadadrugs.org). Mutation carriers should be subject to special care and risk stratification based on clinical assessment. It should be noted, however, that due to incomplete penetrance, the occurrence of mutations does not always have to be associated with the clinical manifestation of the disease. Moreover, if a family member with a mutation associated with BrS is not a carrier of a given specific mutation, this does not exclude the risk of BrS associated with a different mutation. Baruteau et al. conducted a 15-year multicentre study including 442 newborns and children with mutations in the SCN5A gene causing Brugada syndrome type 1 (8 patients; 1.8%), long QT syndrome type 3 (47 patients; 10.6%), progressive conduction disorders (113 patients; 25.6%) and sick sinus syndrome (6 patients; 1.4%).^[27] Phenotypic overlap syndrome was diagnosed in 15.6% (69 patients), and 44.3% of the tested mutation carriers (196 people) did not present any changes in the electrocardiographic recording. After an average follow-up of a little over 8 years, 3 patients with BrS had an ICD implanted due to syncope (2 patients) or documented VT. In one of the treated patients, fever-induced VF occurred, resulting in ICD intervention. The remaining patients did not present clinical symptoms and did not require treatment. During follow-up, 5% of asymptomatic SCN5A mutation carriers presented changes characteristic of BrS. However, it is currently believed that the results of genetic screening tests have no significance for assessing prognosis or influencing treatment.^[8]

Throughout pregnancy, childbirth, and the pre- and postpartum period, a woman with Brugada syndrome requires special care and a multidisciplinary team including not only an obstetrician, but also a cardiologist, a neonatologist and an anaesthesiologist.^[7] It should also be emphasized that further research is necessary to develop guidelines for the

management of patients with Brugada syndrome in this special period of their lives, taking into account the safety of both the woman and the newborn. Childbirth is a factor contributing to cardiac arrhythmias; therefore, the patient should have her heart function monitored continuously.^[28] If anaesthesia is necessary for a patient with Brugada syndrome, Carey and Hocking^[29] and other authors recommend a 5-lead ECG with ST segment analysis. Dash and Pragathe^[30,31] emphasized that in patients with ICD undergoing anaesthesia and surgery, the cardioverter defibrillator should be turned off with a magnet for the duration of the surgery to prevent abnormal discharges. If tachycardia or changes in T wave morphology occur during delivery, there is a risk of too frequent ICD shocks^[9], as is the case during surgery. Therefore, there should be a ready-to-use magnet in the delivery room to temporarily turn off the ICD if there is a risk of ICD malfunction. When the ICD is turned off, there may be a need to immediately use an external defibrillator.^[22, 28] The main problem for an anaesthesiologist is the lack of reliable guidelines for the group of patients with diagnosed Brugada Syndrome, based on large studies, regarding drugs that can be used safely in regional anaesthesia and propofol in general anaesthesia.^[6,32] Decisions about the choice of anaesthesia method must be individualized and based on weak evidence. This raises controversy and uncertainty. There is a need for more reliable research on safe doses of local anaesthetics and propofol, as data from the literature are conflicting.^[29,33] According to many available literature reports, propofol is one of the drugs contraindicated in Brugada syndrome. However, our patient received propofol twice in intravenous boluses for short procedures performed before pregnancy. Similarly, other authors did not observe any complications after propofol administered in boluses.^[33,34] General anaesthesia using thiopental, succinylcholine, fentanyl, sevoflurane and nitrous oxide is considered safe.^[34] Bupivacaine is considered contraindicated for epidural and spinal anaesthesia.^[32] Despite this, there are reports of the safe use of bupivacaine both subarachnoidally and epidurally in this group

of patients.^[29,30,34] The consulting anaesthesiologist presented the patient with the option of epidural analgesia of labour using ropivacaine 0.1% and fentanyl. Ropivacaine has less cardiotoxicity, but there are no clear rules regarding its use in patients with Brugada syndrome. Van der Knijff et al. used low doses of ropivacaine for epidural analgesia of labour without any complications.^[35] It is important to administer local anaesthetics in low doses.^[29,36] A safe option for relieving labour pain is the inhalation of a 50% mixture of nitrous oxide and oxygen during delivery. Some authors emphasize that care should be taken to maintain serum electrolyte levels within normal limits in order to reduce the risk of cardiac arrhythmias caused by these abnormalities. Another problem that should be considered in such patients is hyperthermia. It may trigger cardiac arrhythmias in this group of patients; therefore it is necessary to maintain normothermia.^[29,30]

Conclusion

Summarizing the above considerations of doctors from three different specialties, a common conclusion emerges (which is consistent with the literature data) that close cooperation is necessary for the broadly defined good of the patient. This seems to be the optimal approach to every medical problem we have to face, regardless of its scale, the level of understanding of underlying mechanisms and awareness of what is still unexplained.

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