International Journal of Cardiology 220 (2016) 131–136

Contents lists available at ScienceDirect

International Journal of Cardiology

journal homepage: www.elsevier.com/locate/ijcard

Ventricular tachyarrhythmia during pregnancy in women with heart disease: Data from the ROPAC, a registry from the European Society of Cardiology

Ebru Ertekin [a,1], Iris M. van Hagen [a,1], Amar M. Salam [b,1], Titia P.E. Ruys [a,1], Mark R. Johnson [c,1], Jana Popelova [d,1], William A. Parsonage [e,1], Zeinab Ashour [f,1], Avraham Shotan [g,1], José M. Oliver [h,1], Gruschen R. Veldtman [i,1], Roger Hall [j,1], Jolien W. Roos-Hesselink [a,k,*,1]

[a] Erasmus University Medical Center, Rotterdam, The Netherlands
[b] Hamad General Hospital, Hamad Medical Corporation, Doha, Qatar
[c] Imperial College School of Medicine, Chelsea and Westminster Hospital, London, United Kingdom
[d] Hospital Na Homolce & Pediatric Heart Center University Hospital Motol, Prague, Czech Republic
[e] Royal Brisbane & Women's Hospital, Herston, Australia
[f] Cairo University, Cairo, Egypt
[g] Hillel Yaffe Medical Center, Hadera, Israel
[h] Adult Congenital Heart Disease Unit, La Paz University Hospital, Madrid, Spain
[i] Adolescent and Adult Congenital Heart Program, Cincinnati Children's Hospital, Cincinnati,United States
[j] Norwich Medical School, University of East Anglia, Norwich, United Kingdom
[k] Fellow of the European Society of Cardiology, Sophia Antipolis Cedex, France

ARTICLE INFO

Article history:
Received 19 April 2016
Accepted 19 June 2016
Available online 23 June 2016

Keywords:
Ventricular tachyarrhythmia
Cardiovascular disease
Pregnancy
Maternal outcome
Fetal outcome

ABSTRACT

Objectives: To describe the incidence, onset, predictors and outcome of ventricular tachyarrhythmia (VTA) in pregnant women with heart disease.
Background: VTA during pregnancy will cause maternal morbidity and even mortality and will have impact on fetal outcome. Insufficient data exist on the incidence and outcome of VTA in pregnancy.
Methods and results: From January 2007 up to October 2013, 99 hospitals in 39 countries enrolled 2966 pregnancies in women with structural heart disease. Forty-two women (1.4%) developed clinically relevant VTA during pregnancy, which occurred mainly in the third trimester (48%). NYHA class >1 before pregnancy was an independent predictor for VTA. Heart failure during pregnancy was more common in women with VTA than in women without VTA (24% vs. 12%, p = 0.03) and maternal mortality was respectively 2.4% and 0.3% (p = 0.15). More women with VTA delivered by Cesarean section than women without VTA (68% vs. 47%, p = 0.01). Neonatal death, preterm birth (<37 weeks), low birthweight (<2500 g) and Apgar score <7 occurred more often in women with VTA (4.8% vs. 0.3%, p = 0.01; 36% vs. 16%, p = 0.001; 33% vs. 15%, p = 0.001 and 25% vs. 7.3%, p = 0.001, respectively).
Conclusions: VTA occurred in 1.4% of pregnant women with cardiovascular disease, mainly in the third trimester, and was associated with heart failure during pregnancy. NYHA class before pregnancy was predictive. VTA during pregnancy had clear impact on fetal outcome.

1. Introduction

Heart disease is an important cause of maternal death and, surprisingly, rates are increasing [1,2]. Interim analysis of the Registry Of Pregnancy and Cardiac disease (ROPAC) reported a 100 times higher maternal mortality rate in women with heart disease compared to the general pregnant population [3]. This raises serious safety concerns for women with structural heart disease and makes it imperative to identify those at greatest risk of complications during pregnancy.

Abbreviations: AOP, Aortic pathology; CHD, Congenital heart disease; CMP, Cardiomyopathy; IHD, Ischemic heart disease; NYHA, New York Heart Association; VHD, Valvular heart disease; VTA, Ventricular tachyarrhythmia.

* Corresponding author: Department of Cardiology, Office Ba 308, Erasmus University Medical Center, PO Box 2040, 3000 CA Rotterdam, The Netherlands.
E-mail address: j.roos@erasmusmc.nl (J.W. Roos-Hesselink).

[1] This author takes responsibility for all aspects of the reliability and freedom from bias of the data presented and their discussed interpretation.

http://dx.doi.org/10.1016/j.ijcard.2016.06.061

Potentially life-threatening ventricular tachyarrhythmia (VTA) is rare during normal pregnancy (2 per 100,000 pregnancies) [4,5], but may be associated with maternal hemodynamic compromise causing adverse consequences for both mother and fetus [6]. This data, particularly in women with structural heart disease, however is scarce and is generally poorly reported in the literature. The incidence of VTA in pregnant women with heart disease has been documented ranging from 1.0% to 1.4% [1,7]. In patients with congenital heart disease, VTA occurs in up to 1.6% [1,8], whereas there are hardly any reports on the incidence of VTA in pregnant women with valvular heart disease, ischemic heart disease or cardiomyopathy. Also, insufficient data exist on the outcome of VTA during pregnancy. Previous studies mostly report on arrhythmias in general, but it is of major importance to distinguish potentially life-threatening VTA from the mostly benign supraventricular tachycardia. This study describes the incidence, onset, predictors and outcome of VTA during pregnancy in women with structural heart disease.

2. Methods

2.1. Study design

The Registry Of Pregnancy And Cardiac disease (ROPAC) is part of the EURObservational Research Programme (EORP of the European Society of Cardiology) and was initiated in 2007. From January 2008 pregnant women with heart disease were included prospectively. Patients from 2007 were included retrospectively, assuming that the complete data were available and reliable. All patients included up to October 2013 were included in the current interim analysis. In this period, 99 hospitals in 39 countries contributed to the registry and a total of 2966 pregnant women with congenital heart disease (CHD), valvular heart disease (VHD), cardiomyopathy (CMP), ischemic heart disease (IHD), aortic pathology (AOP) or pulmonary hypertension were enrolled. Non-structural heart disease, for example arrhythmia occurring in the context of a structurally normal heart, was excluded. The study protocol and first results of this registry were published in 2013 [3]. Informed consent was obtained from the patients and the study protocol conforms to the ethical guidelines of the 1975 Declaration of Helsinki as reflected in a priori approval by the institution's human research committee.

2.2. Data

VTA has been defined as three or more consecutive ventricular beats with a mean rate of more than 100 beats per minute; however, only clinically relevant VTA (when the patient had physical complaints, needed treatment for VTA or when the patient had more than 100 consecutive beats) was included. Onset of VTA, given in weeks of gestation, was calculated using the expected date of delivery. Data up to one week after delivery was available for all patients. Baseline characteristics before pregnancy were analyzed, including cardiac diagnosis, maternal age, parity, clinical signs of heart failure, hypertension, smoking status, medication use and New York Heart Association (NYHA) functional class. The following cardiac diagnoses were included: CHD, VHD, IHD, CMP, AOP and pulmonary hypertension. The type of cardiac lesions were divided into three categories for the univariable logistic regression analysis: right sided lesions (e.g. Ebstein anomaly, tetralogy of Fallot, pulmonary stenosis), left sided lesions (e.g. aortic valve disease, mitral valve disease and most cardiomyopathies) and shunt lesions (e.g. atrial septal defects and ventricular septal defects). Participating countries were classified as developed or developing according to the International Monetary Fund classification [9].

2.3. Statistical methods

Categorical data are presented as frequencies (numbers) and percentages. Normality of continuous data was checked by one-sample Kolmogorov–Smirnov tests and histograms. Continuous data are presented as mean values ± one standard deviation (SD) when normally distributed. The chi-squared test was used to compare differences in categorical data between independent patient groups. Fisher's exact tests were applied if any expected cell count was less than 5. The Student's t-test was used to compare differences in continuous data between independent patient groups. Mean birthweight was corrected for

Table 1
Baseline characteristics of cardiac patients with and without VTA[a].

	Total group (n = 2966)	Patients with VTA (n = 42)	Patients without VTA (n = 2924)	p-Value
Mean age in years (SD[b])	29.3 (5.6)	28.9 (5.7)	29.3 (5.6)	0.78
Nulliparity (%)	45	55	45	0.21
Clinical signs of heart failure (%)	10	21	10	0.02
Hypertension (%)	6.5	4.9	6.5	1.00
Current smoker (%)	4.3	5.1	4.3	0.68
Developing countries (%)	35	45	35	0.17
NYHA class[c]				0.002
NYHA class 1 (%)	73	50	73	
NYHA class 2 (%)	22	38	22	
NYHA class 3 (%)	2.9	11.9	2.8	
NYHA class 4 (%)	0.3	0	0.3	
Type of heart disease				
Congenital heart disease (%)	56	48	56	0.28
Valvular heart disease (%)	32	14	32	0.01
Ischemic heart disease (%)	1.6	2.4	1.6	0.68
Cardiomyopathy (%)	7	29	7	<0.001
Aortic pathology (%)	3.4	7.1	3.4	0.18
Pulmonary arterial hypertension (%)	0.4	0	0.4	1.00
Medication use before pregnancy				
Beta-blocker (%)	12	17	12	0.39
Other anti-arrhythmic drugs (%)	3.1	0	3.1	0.64
Diuretics (%)	5.8	9.5	5.7	0.30
ACE inhibitors (%)[d]	3.9	4.8	3.9	0.68
Calcium antagonists (%)	0.2	0	0.2	0.34

[a] VTA: ventricular tachyarrhythmia.
[b] SD = STANDARD deviation.
[c] NYHA class = New York Heart Association functional class.
[d] ACE inhibitors: Angiotensin-converting enzyme inhibitors.

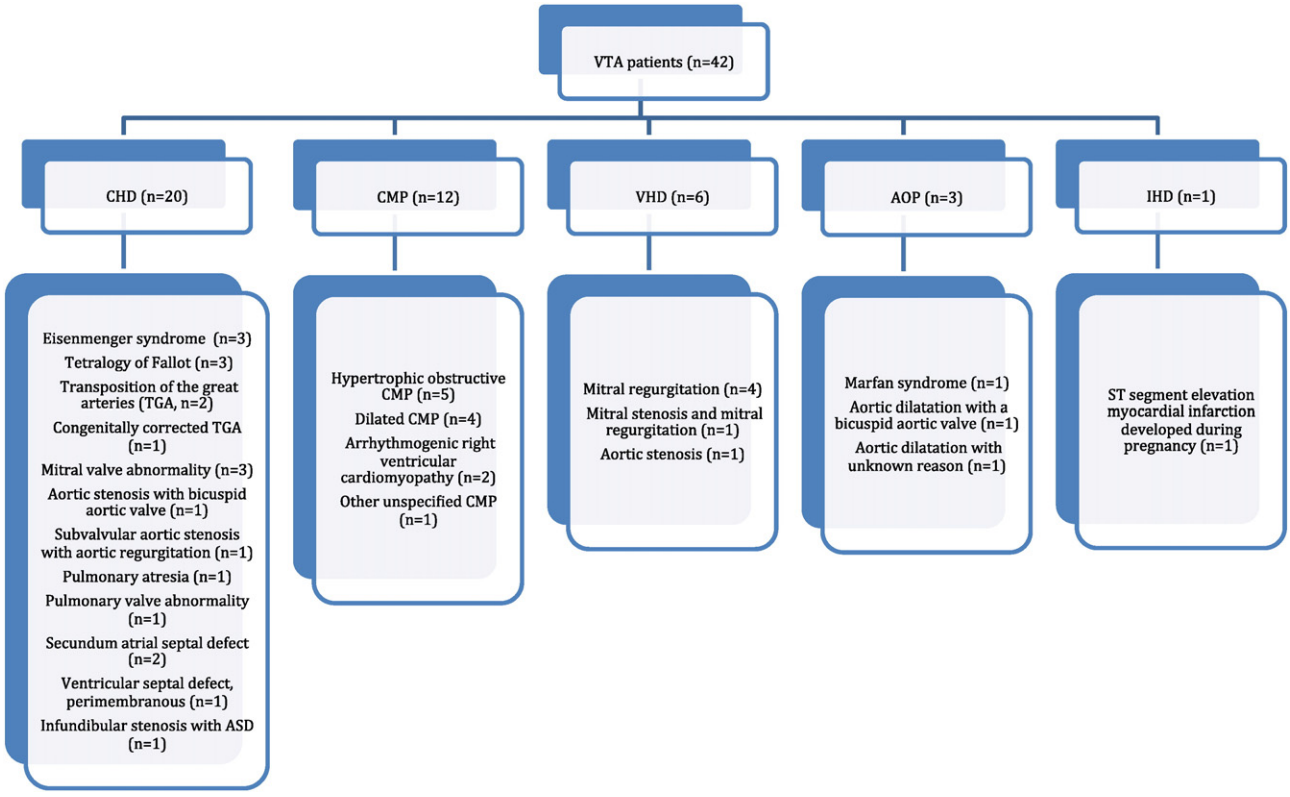

Fig. 1. Diagnoses per cardiac disease category. CHD = congenital heart disease, VHD = valvular heart disease, CMP = cardiomyopathy, AOP = aortic pathology.

gestational age, fetal sex, maternal age and diabetes, using linear regression. The birthweight was not normally distributed; therefore both the median birthweight and the corrected mean birthweight are shown in this article. Baseline patient characteristics associated with VTA were identified with univariable logistic regression analysis. Available echocardiographic data (moderate or severely impaired systemic ventricular function) were also analyzed in the univariable logistic regression analysis. The multivariable analysis consists of variables that were associated with an increased incidence of the studied endpoint ($p < 0.15$). For each 10 cases/patients, one of the most significant univariable predictors was included in the multivariable analysis. A p-value <0.05 (2-sided test) was considered statistically significant. All statistical analyses were performed using SPSS 21.0 (SPSS Inc., Chicago).

3. Results

3.1. Baseline characteristics

Of the 2966 patients included in the registry, 42 (1.4%) patients developed clinically relevant VTA during pregnancy. One patient had ventricular fibrillation (VF), the other patients had ventricular tachycardia. Baseline characteristics of pregnant patients with and without VTA are shown in Table 1. The incidence of VTA was 1.2% in CHD patients, 0.6% in VHD patients, 5.9% in CMP patients, 2.1% in IHD patients and 3.0% in AOP patients. VTA was not observed in patients with pulmonary hypertension. The diagnoses per cardiac disease category are shown in Fig. 1. Before pregnancy, three patients had pacemaker-dependent rhythm. All others were in sinus rhythm.

3.2. Onset of VTA

Fig. 2 shows that VTA mainly occurred in the third trimester (48%). Fig. 3 shows the onset of VTA per cardiac disease category.

3.3. Predictors

The results of the univariable and multivariable logistic regression are shown in Table 2.

3.4. Medication before and during pregnancy

Of the 42 patients with VTA, 31% used cardiac medication before pregnancy. The medication used before pregnancy is shown in Table 1. During pregnancy, 74% of the VTA patients used medication: 57% used beta-blockers, 12% used other anti-arrhythmic drugs and 12% used diuretics. Amiodarone was used in 3 of the 42 patients with VTA.

3.5. Maternal outcome

Pregnancy outcome until one week after delivery is presented in Table 3. Heart failure was diagnosed in 10 VTA patients (24%), occurring

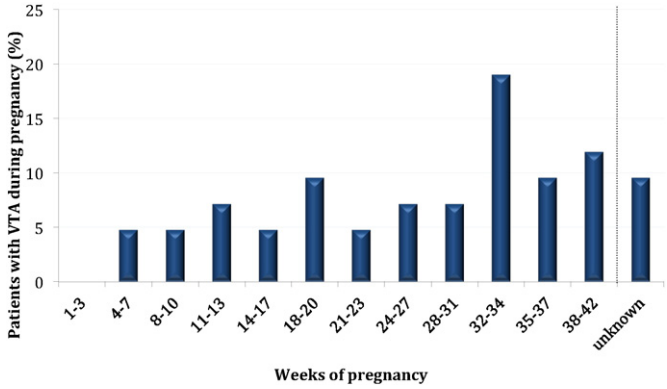

Fig. 2. The occurrence (onset) of VTA during pregnancy in women with heart disease. VTA = ventricular tachyarrhythmia.

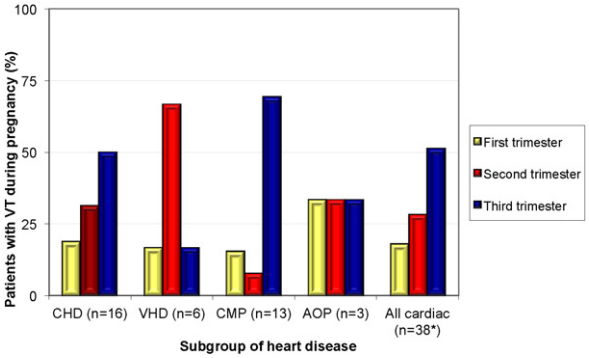

Fig. 3. The occurrence of VTA during pregnancy per cardiac disease category. The number of patients per category is shown between brackets. CHD = congenital heart disease, VHD = valvular heart disease, CMP = cardiomyopathy, AOP = aortic pathology, All cardiac = CHD + VHD + CMP + AOP. *The time of onset of VTA was missing in four CHD patients.

after VTA in three patients, before in three and at the same time in one and in the remaining three patients the temporal relationship was unclear. Maternal death occurred in one patient (2.4%). This was a 36-year-old woman with pulmonary atresia, who had a bioprosthetic pulmonary valve replacement before pregnancy. She was taking beta-blockers because of aortic dilatation and used LMWH throughout pregnancy. At 17 weeks of pregnancy, she had an out of hospital cardiac arrest due to VF. She survived resuscitation, but was left in a persistent vegetative state. She was ventilated until delivery at 32 weeks and died one week postpartum.

3.6. Delivery and fetal outcome

Fetal outcome is shown in Table 3. Mode of delivery in patients with VTA was by emergency Cesarean section in 20%, elective Cesarean section in 48% and vaginally in 32%. No fetal death occurred in VTA patients (Table 3), but there were two neonatal deaths. A 28-year-old patient with non-obstructive CMP developed VTA in week 6 of pregnancy. She underwent Cesarean section at 29 weeks for cardiac reasons, followed by an unexplained neonatal death. The second neonatal death was due to acute respiratory distress syndrome (ARDS) one week postpartum. The mother of this child had subaortic stenosis and a history of heart failure before pregnancy.

Table 2
Pre-pregnancy predictors for VTA.

Univariable	Odds ratio	95% CI	p-Value
Congenital heart disease	0.72	(0.39–1.32)	0.28
Valvular heart disease	0.35	(0.15–0.84)	0.02
Cardiomyopathy	5.76	(2.90–11.40)	<0.001
Right sided lesion	0.72	(0.32–1.62)	0.43
Left sided lesion	1.71	(0.91–3.20)	0.09
Shunt lesion	0.61	(0.26–1.46)	0.27
NYHA class > 1	2.98	(1.62–5.49)	<0.001
Nulliparity	1.48	(0.80–2.72)	0.21
Hypertension	0.74	(0.18–3.08)	0.68
Clinical signs of pre-pregnancy heart failure	2.59	(1.23–5.47)	0.01
Developing countries	1.53	(0.83–2.82)	0.17
Any medication use before pregnancy	1.16	(0.60–2.24)	0.66
Beta-blocker use before pregnancy	1.43	(0.63–3.24)	0.39
Echo prior to pregnancy			
Systemic ventricular dysfunction moderate/severely impaired	4.59	(1.92–10.96)	0.001
Multivariable			
Cardiomyopathy	2.70	(0.95–7.69)	0.06
NYHA class > 1	2.64	(1.12–6.20)	0.03
Clinical signs of pre-pregnancy heart failure	0.88	(0.29–2.64)	0.82
Systemic ventricular dysfunction	2.25	(0.78–6.48)	0.13

4. Discussion

This is the first detailed study of pregnancy outcome after VTA in patients with cardiovascular disease. In this large prospective international registry of 2966 pregnancies with heart disease, the incidence of VTA during pregnancy was 1.4% and occurred mainly in the third trimester. NYHA class >1 before pregnancy was an independent predictor of VTA. VTA was associated with a marked increase in the neonatal death rate, preterm birth rate, low birthweight rate and poor Apgar score.

4.1. Incidence of VTA

Existing data on the incidence of VTA during pregnancy in patients with heart disease is scarce. Siu et al. studied pregnancy outcome in two cohorts of women with heart disease. VTA occurred in 4 cases in the cohort of 276 pregnancies and in 6 patients in the cohort of 599 pregnancies [1,7]. The incidence of VTA in our study (1.4%) was comparable to the incidence of Siu et al.'s retrospective (1.4%) and prospective (1.0%) studies.

4.1.1. Congenital heart disease

VTA during pregnancy in women with CHD is rare. We observed VTA in 1.2% of CHD patients, in keeping with the reported incidence range from 0.4 to 1.6% [1,8]. Patients with repaired CHD in the study of Niwa et al. [10] had a significantly higher incidence of VTA than healthy pregnant women. In their study, the prevalence of non-sustained VTA was 14% with the highest incidence in patients with previous surgical correction of Tetralogy of Fallot (TOF) [8,10,11]. In our study, VTA occurred in 2.5% of TOF patients (3 out of 119 patients), however, we only included patients with symptomatic VTA.

4.1.2. Cardiomyopathy

VTA occurred remarkably often in patients with CMP (7.4%) in our study. In the study of Grewal et al. [12], VTA occurred in 3% of dilated CMP patients. In patients with hypertrophic CMP, the incidence of arrhythmias (including VTA) was not increased during pregnancy [13]. However, in hypertrophic CMP patients with an Implantable Cardioverter Defibrillator (ICD), VTA is a common complication with an observed incidence of 22% during pregnancy [14]. Some case reports of VTA in patients with peripartum cardiomyopathy (PPCM) have been published previously [15–17]. In our study VTA did not occur in the 33 PPCM patients.

4.1.3. Valvular heart disease

We found that 0.7% of the patients with VHD developed VTA during pregnancy and the majority had mitral valve disease. In the literature, ventricular arrhythmia, not further defined, has been described in 1.5% of pregnant women with VHD [18] and occurred in 1.2% in Siu et al.'s prospective study.

4.1.4. Ischemic heart disease

Although seldom encountered during pregnancy, IHD is a major and increasing cause of maternal death [19]. In the existing literature, cases of VF, but not ventricular tachycardia, have been reported during pregnancy in patients with a myocardial infarction [20,21]. In our registry, VTA occurred in only one patient with IHD (2.1%).

4.2. Predictors for VTA

Women with a limited exercise tolerance are at higher risk of developing complications during pregnancy [4], and consequently, we were not surprised to find that NYHA class >1 before pregnancy is an independent predictor of VTA. Any disease process that affects the

Table 3
Pregnancy outcome in cardiac patients with and without VTA
Outcome until one week after pregnancy.

	Total group (n = 2966)	Patients with VTA (n = 42)	Patients without VTA (n = 2924)	p-Value
Maternal mortality (%)	0.4	2.4	0.3	0.15
Cardiac				
Heart failure (%)	13	24	12	0.03
Thromboembolic events (%)	0.8	0	0.8	1.00
Endocarditis (%)	0.2	0	0.2	1.00
Bleeding during pregnancy (%)	6.2	4.8	6.2	1.00
Obstetric				
Intra-uterine growth retardation (%)	4.6	4.8	4.6	0.72
Pregnancy induced hypertension (%)	2.3	0	2.4	0.63
(Pre)eclampsia (%)	2.4	0	2.5	0.63
Cesarean section (%)	48	68	47	0.01
Fetal outcome				
Miscarriage (<24 weeks; %)	2.7	0	2.7	0.26
Late fetal death (≥24 weeks; %)	0.7	0	0.7	1.00
Neonatal death (%)	0.3	4.8	0.3	0.01
Median pregnancy duration (weeks)	38.2	37.4	39.0	<0.001
Apgar score < 7 (%)	7.6	25	7.3	0.001
Preterm birth (<37 weeks; %)	16	36	16	0.001
Low birthweight (<2500 g; %)	15	33	15	0.001
Median birthweight (g)	–	2730	3020	0.006
Corrected mean birthweight (g)[a]	–	3283	3289	0.94

[a] Birthweight corrected for: gestational age, fetal sex, maternal age, and diabetes.

ventricular myocardium causing hypertrophy, infiltration or scarring may disrupt the electrical integrity of the myocardium and induce VTA [11]. CMP in general is known to be associated with ventricular arrhythmias as it is often associated with diminished ventricular function [22]. In accordance with this, we found that a diagnosis of CMP showed a borderline significance (0.06) in the multivariable analysis.

4.3. Onset during pregnancy

Nakagawa et al. [23] studied 11 pregnant women who experienced a new onset of ventricular tachycardia during pregnancy. In their study, the onset of ventricular tachycardia was distributed equally over the three trimesters and did not occur in the postpartum period. Overall, VTA occurred throughout pregnancy but more in the third trimester in our study, with CHD patients experiencing VTA more in the second and third trimester, while CMP patients typically present with VTA at the end of pregnancy (≥32 weeks), perhaps reflecting a different threshold for VTA. These findings raise a number of hypotheses regarding the mechanism of VTA during pregnancy. Cardiac output, induced by a decline in systemic vascular resistance, rises rapidly through the first and second trimesters reaching a peak at around 26 weeks and remaining at this level until the end of pregnancy [24]. The increase in cardiac output is due to an increase in heart rate and stroke volume. The greater filling, resulting in an increase in cardiac end diastolic volumes, will result in myocardial stretch which has been shown to induce VTA [25]. In addition to these physiological changes during normal pregnancy, women with structural heart disease have limited ability to adjust to the hemodynamic requirement of pregnancy, and this may become manifest as systolic and diastolic cardiac dysfunction during pregnancy [24], which again may increase the risk of VTA. Finally, the increased sympathetic activity observed during pregnancy has also been suggested to be responsible for the increased incidence of arrhythmias and it is well known that high plasma catecholamine concentrations and adrenergic receptor sensitivity may trigger VTA [26]. In all patients, the hemodynamic overload may well have contributed to the development of VTA, while the increased sympathetic activity may be more important in specific groups such as cardiomyopathy patients, which may be more susceptible.

4.4. Maternal outcome

VTA, outside of pregnancy, is associated with sudden cardiac death, especially in the presence of cardiomyopathy [22,27]. Previous publications on VTA during pregnancy were either case reports or studies which have only reported on its incidence. Therefore, it is not well known whether VTA during pregnancy carries an extra high risk for mortality. We did not detect a significant increase in maternal mortality, but it must be noted that, although our series is by far the largest, our mortality numbers are still small and we have analyzed outcome only until one week after pregnancy. The timing of heart failure and VTA seemed to be associated in our study. However, a clear cut pattern was not found as some patients developed heart failure shortly after VTA, while others developed heart failure prior to VTA.

4.5. Delivery and fetal outcome

Women with VTA during pregnancy had higher rates of neonatal death, preterm birth and poor Apgar score. Certainly, low birthweight was more common in patients with VTA than in patients without VTA and the median birthweight was significantly lower in VTA patients. However, the corrected mean birthweight did not significantly differ between the two groups, suggesting that the difference in birthweight is primarily due to the higher preterm birth rate. Physicians often opt for preterm delivery in women with VTA, partly to shorten the period of hemodynamic compromise and also to be able to institute a more aggressive therapy for VTA, which might have an adverse effect on the fetus. Equally, the decision for early delivery may have a negative impact on neonatal development [28].

4.6. Management of VTA during pregnancy

In general, the management of VTA in pregnancy is similar to that outside of pregnancy. For VTA with hemodynamic compromise, immediate cardioversion, which is reported to be safe in all phases of pregnancy, is recommended [4]. When the patient is not hemodynamically compromised, medication should be considered. The concerns with anti-arrhythmic drug use during pregnancy relate to their effects on fetal growth and development. Beta-blockers are the drug of choice

for hemodynamically well-tolerated VTA. The use of beta-blockers during pregnancy is generally well tolerated by both mother and fetus, even though it has been associated with a decrease in fetal heart rate, low blood glucose and reduced birthweight [22,29]. In our study, most patients with VTA (57%) were treated with a beta-blocker, other antiarrhythmic drugs were prescribed in 12% of the patients. Amiodarone was used in 3 of the 42 patients with VTA in our study and may have had a deleterious effect on one fetus, perhaps causing growth retardation requiring earlier delivery [22]. A multidisciplinary approach with close monitoring of mother and baby with timely intervention for both is required to optimize maternal and fetal outcomes.

4.7. Implications on clinical practice

The significance of VTA during pregnancy in women with heart disease lies in the fact that it carries a higher preterm birth rate and, consequently, lower birthweight and poor Apgar score. Cardiac patients in NYHA class >1 and patients with CMP are at particular risk and should be counseled about the risks before pregnancy and followed closely during pregnancy.

4.8. Study limitations

This dataset, like in other registry collected datapoints, was limited by the availability of information on the past history. In our registry missing data concerning the current pregnancy ranged from 0% to 4% for all parameters except two: smoking (14%) and left ventricular dysfunction (36%). Although VTA is an important complication especially in women with underlying heart disease, its incidence is low, reflected in the number of cases. This makes it difficult to draw any firm conclusions. The severity of the VTAs was not reported in detail.

Funding sources

The ROPAC is a registry within the EURObservational Research Programme (EORP) of the European Society of Cardiology. Since the start of EORP, the following companies have supported the program: Abbott Vascular Int. (2011–2014), Amgen (2012–2015), AstraZeneca (2014–2017), Bayer Pharma AG (2013–2015), Boehringer Ingelheim (2013–2016), Boston Scientific (2010–2012), The Bristol Myers Squibb and Pfizer Alliance (2014–2016), The Alliance Daiichi Sankyo Europe GmbH and Eli Lilly and Company (2014–2017), Gedeon Richter Plc. (2014–2017), Menarini Int. Op. (2010–2012), MSD-Merck & Co. (2011–2014), Novartis Pharma AG (2014–2017), ResMed (2014–2016), Sanofi (2010–2011), and SERVIER (2012–2015). The companies that support EORP were not involved in any part of the study or this report. The authors have no personal disclosures with regard to this article.

Conflict of interest

The authors report no relationships that could be construed as a conflict of interest.

Acknowledgments

We would like to thank all ROPAC investigators, who are listed in the Supplemental Material online. Our gratitude goes to the EORP team for their contribution, in particular: Elin Folkesson-Lefrancq, Viviane Missiamenou, Gérard Gracia and Myriam Lafay. To join the Registry: http://www.escardio.org/guidelines-surveys/eorp/surveys/pregnancy/Pages/welcome.aspx.

Appendix A. Supplementary data

Supplementary data to this article can be found online at http://dx.doi.org/10.1016/j.ijcard.2016.06.061.

References

[1] S.C. Siu, M. Sermer, J.M. Colman, et al., Prospective multicenter study of pregnancy outcomes in women with heart disease, Circulation 104 (2001) 515–521.
[2] N. Jastrow, P. Meyer, P. Khairy, et al., Prediction of complications in pregnant women with cardiac diseases referred to a tertiary center, Int. J. Cardiol. 151 (2011) 209–213.
[3] J.W. Roos-Hesselink, T.P. Ruys, J.I. Stein, et al., Outcome of pregnancy in patients with structural or ischaemic heart disease: results of a registry of the European Society of Cardiology, Eur. Heart J. 34 (2013) 657–665.
[4] European Society of G, Association for European Paediatric C, German Society for Gender M, et al., ESC guidelines on the management of cardiovascular diseases during pregnancy: the task force on the management of cardiovascular diseases during pregnancy of the European Society of Cardiology (ESC), Eur. Heart J. 32 (2011) 3147–3197.
[5] J.M. Li, C. Nguyen, J.A. Joglar, M.H. Hamdan, R.L. Page, Frequency and outcome of arrhythmias complicating admission during pregnancy: experience from a high-volume and ethnically-diverse obstetric service, Clin. Cardiol. 31 (2008) 538–541.
[6] H.L. Tan, K.I. Lie, Treatment of tachyarrhythmias during pregnancy and lactation, Eur. Heart J. 22 (2001) 458–464.
[7] S.C. Siu, M. Sermer, D.A. Harrison, et al., Risk and predictors for pregnancy-related complications in women with heart disease, Circulation 96 (1997) 2789–2794.
[8] S. Tateno, K. Niwa, M. Nakazawa, et al., Arrhythmia and conduction disturbances in patients with congenital heart disease during pregnancy: multicenter study, Circ. J. 67 (2003) 992–997.
[9] (WEO) WEO, Growth Resuming, Dangers Remain, 2012 177–180.
[10] K. Niwa, S. Tateno, T. Akagi, et al., Arrhythmia and reduced heart rate variability during pregnancy in women with congenital heart disease and previous reparative surgery, Int. J. Cardiol. 122 (2007) 143–148.
[11] D.L. Adamson, C. Nelson-Piercy, Managing palpitations and arrhythmias during pregnancy, Heart 93 (2007) 1630–1636.
[12] J. Grewal, S.C. Siu, H.J. Ross, et al., Pregnancy outcomes in women with dilated cardiomyopathy, J. Am. Coll. Cardiol. 55 (2009) 45–52.
[13] R. Thaman, A. Varnava, M.S. Hamid, et al., Pregnancy related complications in women with hypertrophic cardiomyopathy, Heart 89 (2003) 752–756.
[14] P.K. Schuler, A. Herrey, A. Wade, et al., Pregnancy outcome and management of women with an implantable cardioverter defibrillator: a single centre experience, Europace 14 (2012) 1740–1745.
[15] N. Yahagi, K. Kumon, T. Nakatani, et al., Peripartum cardiomyopathy and tachycardia followed by multiple organ failure, Anesth. Analg. 79 (1994) 581–582.
[16] G. Gemici, H. Tezcan, A.S. Fak, A. Oktay, Peripartum cardiomyopathy presenting with repetitive monomorphic ventricular tachycardia, Pacing Clin. Electrophysiol. 27 (2004) 557–558.
[17] A. Puri, R. Sethi, B. Singh, et al., Peripartum cardiomyopathy presenting with ventricular tachycardia: a rare presentation, Indian Pacing Electrophysiol. J. 9 (2009) 186–189.
[18] A. Lesniak-Sobelga, W. Tracz, M. KostKiewicz, P. Podolec, M. Pasowicz, Clinical and echocardiographic assessment of pregnant women with valvular heart diseases—maternal and fetal outcome, Int. J. Cardiol. 94 (2004) 15–23.
[19] R. Cantwell, T. Clutton-Brock, G. Cooper, et al., Saving Mothers' lives: reviewing maternal deaths to make motherhood safer: 2006–2008. The eighth report of the confidential enquiries into maternal deaths in the United Kingdom, BJOG (118 Suppl. 1) (2011) 1–203.
[20] J.J. Curry, F.J. Quintana, Myocardial infarction with ventricular fibrillation during pregnancy treated by direct current defibrillation with fetal survival, Chest 58 (1970) 82–84.
[21] M. O'Donnell, J. Meecham, S.R. Tosson, S. Ward, Ventricular fibrillation and reinfarction in pregnancy, Postgrad. Med. J. 63 (1987) 1095–1096.
[22] European Heart Rhythm A, Heart Rhythm S, D.P. Zipes, et al., ACC/AHA/ESC 2006 guidelines for management of patients with ventricular arrhythmias and the prevention of sudden cardiac death: a report of the American College of Cardiology/American Heart Association task force and the European Society of Cardiology committee for practice guidelines (writing committee to develop guidelines for management of patients with ventricular arrhythmias and the prevention of sudden cardiac death), J. Am. Coll. Cardiol. 48 (2006) e247–e346.
[23] M. Nakagawa, S. Katou, M. Ichinose, et al., Characteristics of new-onset ventricular arrhythmias in pregnancy, J. Electrocardiol. 37 (2004) 47–53.
[24] J. Cornette, T.P. Ruys, A. Rossi, et al., Hemodynamic adaptation to pregnancy in women with structural heart disease, Int. J. Cardiol. 168 (2013) 825–831.
[25] S. Hunter, S.C. Robson, Adaptation of the maternal heart in pregnancy, Br. Heart J. 68 (1992) 540–543.
[26] H.J. Trappe, M. Tchirikov, Cardiac arrhythmias in the pregnant woman and the fetus (Herzrhythmusstorungen bei der Schwangeren und beim Fetus), Internist (Berl) 49 (2008) 788–798.
[27] S.H. Hohnloser, T. Klingenheben, D. Bloomfield, O. Dabbous, R.J. Cohen, Usefulness of microvolt T-wave alternans for prediction of ventricular tachyarrhythmic events in patients with dilated cardiomyopathy: results from a prospective observational study, J. Am. Coll. Cardiol. 41 (2003) 2220–2224.
[28] A.T. Gibson, S. Carney, E. Cavazzoni, J.K. Wales, Neonatal and post-natal growth, Horm. Res. 53 (Suppl. 1) (2000) 42–49.
[29] K. Meidahl Petersen, E. Jimenez-Solem, J.T. Andersen, et al., Beta-blocker treatment during pregnancy and adverse pregnancy outcomes: a nationwide population-based cohort study, BMJ Open (2012) 2.