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META-ANALYSIS

Cardiac Troponin Levels after Percutaneous Atrial Septal Defect Closure: A Qualitative Systematic Review and Meta-Analysis

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ABSTRACT

Introduction: We conducted a systematic review and meta-analysis of published studies to determine the prevalence of troponin elevation after percutaneous atrial septal defect closure (pASDc) as well as to describe the association between troponin elevation and different anatomical risk factors for erosion. **Methods:** A qualitative systematic review and meta-analysis was undertaken. The selected studies included patients of any age receiving a pASDc; performed under transesophageal echocardiography monitoring; reporting troponin level measurement after the intervention; and indicating prevalence of troponin elevation and/or the association with risk factors for erosion. **Results:** Six studies were found which included 391 patients in total. The age of the patients ranged from 1 to 80 years and were mainly female (between 59 and 81%). The success rate of pASDc varied from 92–100%. The prevalence of myocardial injury varied between 16% and 100%. In the meta-analysis cohort including 347 patients with available data, the fixed effect model showed a prevalence of 41.8% CI (95%) 36.6% to 47.2%. Five studies found a relationship between the size of the implanted device and the presence of myocardial injury. The size of the defect, multiple defects within the interatrial septum, deficient posterior rim, lack of use of sizing balloon and longest duration of the procedure were also related to myocardial injury. **Conclusion:** The usefulness of troponin levels measurements after pASDc has been insufficiently studied. The routinely use in a standardized protocol would be useful to determine which patients need a closer follow-up.

KEYWORDS

Atrial septal defect; interatrial septum; percutaneous closure; troponin; erosion

1 Introduction

The *ostium secundum* type atrial septal defect (ASD) is a common form of congenital heart disease with an incidence of 6–10/10,000 live births [1]. The use of transcatheter device techniques has become widely accepted as an alternative strategy to surgery closure. About 80% of all *ostium secundum* type ASD patients are treated with percutaneous closure implanting a device [2]. Current studies have observed that this technique involves lower morbidity and lower costs than surgical treatment [3–6].

Percutaneous closure has several potential complications and, although rare, one of the most dangerous is the occurrence of erosion of adjacent structures caused by the implanted device. The most frequent location

for erosions are the roof of the atria and the aortic root [7]. It is estimated that this complication occurs between 4 and 30/10,000 implants, generally occurring early after the intervention [8], although a case-report including this complication occurring 8 years after implantation has been published [9].

Cardiac troponins I (TnI) and T (TnT) are components of the contractile apparatus of the myocardial cell and are almost exclusively expressed in the heart. Both biomarkers are very specific and sensitive markers hence they are useful for the evaluation and diagnosis of myocardial injury. Detection of a high value of any of the troponins (above the 99th percentile of reference) is associated to myocardial injury [10].

We hypothesized that a relationship between myocardial injury after pASDc implanting a device and different anatomical characteristics of the congenital defect are related to cardiac erosion.

We conducted a systematic review of published studies in order to determine the prevalence of troponin elevation after pASDc as well as to describe the association between troponin elevation and different anatomical risk factors for erosion.

2 Material and Methods

A search for relevant studies was carried out in the MEDLINE, BioMed Central, Cochrane Database of Systematic Reviews and Google Scholar Databases from 1990 until March 2020. The investigation was performed using the following words and their combination: “atrial septal defect”; “interatrial septum”; “closure”; “troponin”; and it was restricted to human studies and English language. Approval from the Hospital Ethics Committee was not required.

The selected studies had to meet the following inclusion criteria: 1) report patients of any age receiving a pASDc as the main population or subgroup; 2) procedure performed under transesophageal echocardiography monitoring; 3) troponin level measurement after the intervention; 4) report prevalence of troponin elevation according to the definition selected by the author and/or the association of troponin elevation with risk factors for erosion.

The criteria for excluding studies from the analysis were as follows: 1) studies without sufficient data on anatomical characteristics of the interatrial defect, 2) duplicate publications, 3) non-original articles such as reviews, letters to editor and case report.

In the case of multiple articles including the same population, the one with the most complete data was selected.

All authors reviewed the titles of the manuscripts as well as the abstracts. Once selected, the full articles were obtained for data examination and extraction. The main variables obtained were age, gender, ASD dimension, sizing-balloon use to measure the defect, dimension of the implanted device and erosion risk factors. Troponin type measured was also reported as well as the normal upper limit which define myocardial injury. The time-frame from sample extraction after ASD occlusion was also obtained. The quality of the articles was assessed using STROBE checklist for observational studies [11]. When there were inconsistencies between authors’ qualifications, these were resolved through discussion and subsequent agreement between the 3 authors.

2.1 Statistical Analysis

Due to the heterogeneity of the studies, we conducted a qualitative systematic review. The proportion of patients with troponin measurement higher than the upper limit proposed by each researcher troponin from each study and the association between troponin elevation and erosion risk factors were reported. The proportions were expressed as a percentage. The association between troponin levels and erosion risk factors was reported according to how it was analyzed in each study.

Meta-analysis was performed adopting a specific MedCalc module, designed to perform meta-analyses of proportions in MedCalc. Overall estimates were calculated with random effects mode and a test for heterogeneity was applied using chi-square and the I^2 statistics. The random effects meta-analysis model was chosen to take account of between study heterogeneity of the included studies, so the summary effect

was a mean estimate of all effects. Confidence intervals were calculated when not reported in the publication. The following formula was used to calculate the 95% Cis, assuming a Poisson distribution of the phenomenon: $\pi \pm 1,96 \sqrt{\pi (1 - \pi)/n}$, where π was the prevalence and n the number of participants [12,13]. MedCalc Software version 14.8.1 was used.

3 Results

Initially, 20 studies had been preselected, of which 6, were finally selected (Fig. 1). All manuscripts were observational and had been published between years 2003 and 2018 [14–19]. A summary of the results of this search is listed in Tab. 1. Only 2 studies, Chung et al. [17] and Saran et al. [19], included patients exclusively with ASD, the remaining 4 studies included patients with patent foramen ovale (PFO) closure and one of them, introduces a comparative group either with surgical ASD treatment or a diagnostic catheterization.

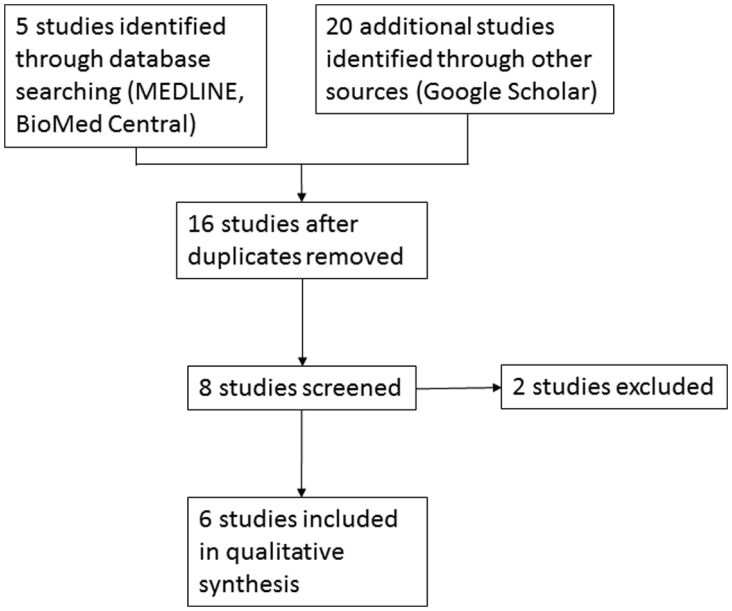

Figure 1: Flow chart of selected studies

Table 1: Patients characteristics

First author, year of publication, [# ref]	Number of patients with ASD	Age/Other	Gender	Devices
Pees et al. [14]	24 patients	Mean 43, 5 years (10–77 years)	79% female	Amplatzer (St Jude Medical)
Tarnok et al. [15]	44 patients (in 2 groups)	Mean 8, 3 years (3–15 years)/Weight 29, 8 ± 15,1 Kg Mean 44 years (18–67 years)/Weight 73, 4 ± 17, 4 Kg	59% female	Amplatzer (St Jude Medical)
Vydt et al. [16]	14 patients	Mean 57 years (17–75 years)	64% female	Cardia (Cardia Inc) Starflex (NMT Medical) Amplatzer (AGA Medical Corp) Cardiastar (Cardia Inc)
Chung et al. [17]	73 patients	Median 4, 5 years (1,1–18 years)/BSA 0, 88 ± 0, 38 m^2	71% female	Amplatzer (St Jude Medical)
Hlebowicz et al. [18]	36 patients	Mean 52 years (22–80 years)/BSA 1, 86 ± 0, 21 m^2	81% female	Amplatzer (St Jude Medical) Figulla (Occlutech)
Saran et al. [19]	200 patients	Median 12 years (2–63 years)/BSA 1, 46 ± 1 m^2	64,5% female	Amplatzer (St Jude Medical) Heart R (Lifetech Scientific) Cocoon (Vascular Concepts Limited)

Body surface area (BSA)

The complete cohort included 391 patients undergoing pASDc, most of them female (ranging between 59% to 81%). Three of the studies included only patients older than 10 years of age and the remaining 3 manuscripts included younger patients so, at the end, patients between 1 to 80 years old were included. Only two studies including 50 patients in total, reported comorbidities such as hypertension (38%) and smoking (7%) [16,18]. In addition, in one of these studies which included 14 patients, diabetes and impaired renal function in 21.5% of patient were reported [16].

There was a high success rate during the intervention ranging from 92–100%. In all studies, the monitoring during the procedure was performed using both transesophageal echocardiography and radioscopy. In the majority of cases the patients underwent general anesthesia, although a minority received only a deep sedation. Troponin level was measured before the procedure in all studies and patients with basal elevation were not included.

In the cases that troponin I was measured (n = 155) for myocardial injury diagnosis, the cut-off value was 0.4 ng/L. Two studies measured troponin T (n = 236) with a cut-off value of 5 ng/L. Ultrasensitive test was not use in any study. There were several implanted devices, most of them Amplatzer™ occluders (Abbott, USA).

The prevalence of myocardial injury was highly variable between 16% and 100%. In the meta-analysis which included 347 patients with available data, the fixed effect model showed a prevalence of 41.8% 95% CI 36.6% to 47.2%. Tab. 2 and Fig. 2 show the results of the meta-analysis of the proportions in fixed and random meta-analysis of proportions models.

Table 2: Meta-analysis of proportion

First author, year of publication, [# ref]	Sample size	Proportion of injury (%)	95% CI
Pees et al. [14]	24	100	85, 7 to 100
Vydt et al. [16]	14	36	12, 7 to 64, 8
Chung et al. [17]	73	70	57, 9 to 80
Hlebowicz et al. [18]	36	89	73, 9 to 96, 8
Saran et al. [19]	200	16	11, 2 to 21, 8
Total (fixed effects)	347	41, 8	36, 6 to 47, 2
Total (random effects)	347	657	25, 7 to 9, 5

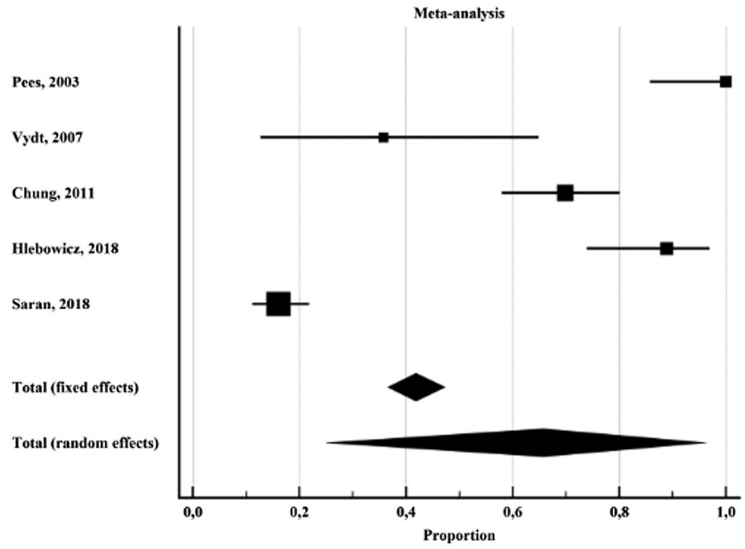

Figure 2: Forest plot of proportion of myocardial injury

Test for heterogeneity. Significance level $p < 0.0001$, I^2 (inconsistency) 97, 8%, 95% CI for I^2 96, 6% to 98, 6%.

Evaluating the characteristics of the intervention and the presence of myocardial injury, five studies found a relationship between the size of the implanted device and the presence of myocardial injury (Tab. 3 and Fig. 3). Myocardial injury also was related to the size of the ASD, the presence of multiple defects, a deficient posterior rim as well as the absence of use of a sizing balloon for ASD measurement. Additionally, the duration of the procedure and the history of dyslipidemia were also related to myocardial injury. None of the studies reported long-term follow-up and the longest follow-up period was 6 months. There were no serious short-term adverse events related to the presence of myocardial injury.

Table 3: Procedural results

First author, year of publication, [# ref]	ASD dimension; device dimension	ASD sizing method	Troponin, (reference limit)	Prevalence of myocardial injury	Association of myocardial injury and risk factors
Pees et al. [14]	ASD dimension not reported; Devices between 15–35 mm.	Sizing balloon (stretched balloon)	Troponin I (AXSYM Abbott); (0, 4 ug/L)	100% (between 4 y 15 hours post procedure)	Not reported
Tarnok et al. [15]	ASD dimension not reported; devices between 12–34 mm.	Not reported	Troponin I (AXSYM Abbott); (0, 1 ug/L)	Not reported	Correlation between amount of myocardial injury and size of the implanted device (r= 0,35; p=0,01)
Vydt et al. [16]	ASD mean dimension 17, 5 mm; mean device dimension 27 mm.	Sizing balloon (stretched balloon)	Troponin I (Ortho-Clinical Diagnostics); (0, 4 ug/L)	35,7% (6 hours post procedure)	Multivariate analysis: dyslipidemia (p = 0.004); device size (p = 0.008)
Chung et al. [17]	ASD mean dimension 17 mm; devices dimension between 7–38 mm.	Sizing balloon (stretched balloon)	Troponin I (Manufacturer not reported); (0, 4 ug/L)	70% (12 hours post procedure)	Correlation between myocardial injury amount and device size/body surface ratio (r = 0.276; p = 0.02)
Hlebowicz et al. [18]	ASD mean dimension 22, 8 mm; device mean dimension 22, 8 mm.	Sizing balloon (stretched balloon)	Troponin T (Manufacturer not reported); (5 ng/L)	89% (24 hours post procedure)	Correlation between myocardial injury amount and ASD diameter (r = 0.424; p = 0.010), device size (r = 0.542; p = 0.001) and duration of the intervention (r = 0.348; p = 0.035)
Saran et al. [19]	ASD mean dimension 17, 5 mm; device mean dimension 21, 3 mm.	Not reported	Troponin T (Radiometer AQT90 FLEX; (5 ng/L)	16% (4–6 hours post procedure)	Multiple defects (OR 13.2) and posterior rim deficit (OR 13.2). Correlation between device size and myocardial injury extension (r2 = 0.125; p = 0.047)

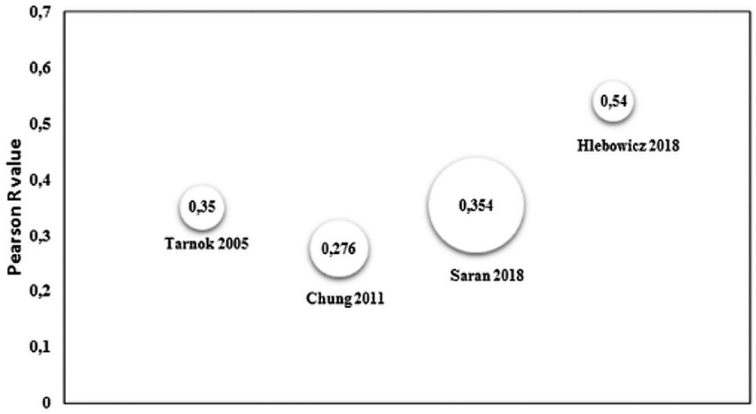

Figure 3: Four studies reported the correlation between the ASD dimension and the presence of myocardial injury. One remaining study (Vydt et al.) [16] did not reported this association as correlation and was not included in this graph. Number of patients in each study and Pearson *R*-value

4 Discussion

The main finding of the present review is the confirmation of the existence of myocardial injury defined by elevation of cardiac troponins after pASDc. The reported prevalence was variable among the different studies (ranging from 16 to 100%) depending on the type of biomarker reported, the time frame of measurement and the cut-off value utilized. The follow-up time of these patients has been short and the presence of myocardial injury has had no relevant short-term clinical consequences.

Cardiac troponins are widely used as biomarkers of myocardial injury and may be associated with non-ischaemic cardiac conditions as well as non-cardiac conditions [10]. Several studies have shown that myocardial injury, defined by an elevated cardiac troponin value, is frequently encountered and is associated with an adverse prognosis [20]. However, the prognosis is often related to comorbidities. Elevation of cardiac troponin levels are able to predict subsequent subclinical and clinical morbimortality in children after cardiac surgery included patients with ASD [21]. Troponin elevation has never been shown to worsen the prognosis after ASD percutaneous occlusion.

The etiology of myocardial injury is unclear. It has been postulated that it could be due to transient instability of the myocardial cell membrane due to the pressure exerted by the device on adjacent structures. Parietal stress or rubbing of the device on these structures is also proposed as potential mechanism. If these postulates were certain, children would be more susceptible to develop injury, however, studies comparing the prevalence of myocardial injury between children and adults have not found this association [15].

Hlebowicz et al. [18] reported 15% of myocardial injury after PFO closure. The PFO device has a very narrow waist, suggesting that the discs of the device may contribute to the release of troponins. Moreover, Pees et al reported also elevation of troponins after PFO closure without differences regarding closure of an interatrial septum communication [14].

Another possible cause is air embolization to the coronary circulation. In general, the studies have electrocardiographic and echocardiographic controls, both during and after the procedure, and no electrocardiographic disorders compatible with ischemia, arrhythmias, or regional motility disorders have been reported to support this mechanism as the main etiology.

The association between the presence and level of myocardial injury and the size of the implanted device is remarkable and it has been mentioned in several of the studies. Oversizing of the device compared to the size of the defect has been recognized as one of the main erosion risk factors [22] due to friction or rubbing of the device on the aortic root or into the roof of the atrium. These are the most frequent sites of laceration as demonstrated in pathology specimens and after direct exploration during emergency surgery due to erosion occurrence [22,23]. The reported time to erosion is variable and it can occur even years after the implantation, although most of them occur within the first days after the intervention. In this scenario, the presence and level of myocardial injury could be useful to stratify patients requiring a closer follow-up and surveillance.

There are few reports specifying anatomical variables of the ASD that might be considered as the cause of myocardial injury. Saran et al. [19] reported an association between myocardial injury and the presence of multiple defects or posterior rim deficiency. In addition, Hlebowicz et al. indicated that longer duration of the intervention, which could be explained for challenges implanting the device with multiple attempts or changes in the device size before release, could be also associated with myocardial injury [18].

4.1 Insufficient ASD Anatomical Details

Patients with deficient aortic rim may not have been included. Currently, the aortic rim deficiency is not a contraindication for pASDc and it is a very frequent finding in clinical practice [22,24–25] being recognized in approximately 90% of erosion cases. Also, superior rim deficiency and the implantation of an oversized device were postulated as risk factors for erosion [7].

Recently, a Japanese group [23] reported their experience with cardiac erosion after pASDc and stated that the aortic Valsalva wall deformation secondary to compression by both disks of the device and the

maximum depth of the disks relative to the standard curve of the Valsalva sinus wall (named Dent), would be another erosion risk factor to be considered.

Interatrial septum malalignment relative to the aortic root has not been mentioned as a potential cause of injury and have been probably not explored [26]. In the same way, there is little or no information about the implication of the number of attempts made at the time of implantation as well as different maneuvers during the implantation that might cause local trauma [27].

Moreover, there is very little experience regarding the incidence of myocardial injury associated to balloon sizing of the defect. Pees et al. failed to identify a case among seven patients undergoing to balloon measurement of the defect and Hlebowicz et al. found the association in 33% of their patients [14,18].

The method of selecting the size of the device was not uniform in all studies. The most frequently reported method includes the advancement of a sizing balloon within the interatrial septum and performing the "stretching technique" visualized by transesophageal echocardiography. Current recommendations endorse the "stop-flow technique" (up to the point to observe elimination of the interatrial shunting) to reduce the risk of erosion [17].

After extensive literature search, the possible relationship of risk factors of erosion and myocardial injury is only a speculation. The correlation between myocardial injury and the device size could also be related to greater technical difficulties at the time of implantation being the cause of the injury.

The present review and meta-analysis have several limitations. The prevalence of myocardial injury is very heterogeneous among the studies, probably due to the different types of troponin used, different cut points to define myocardial injury, different measurement times after device implantation as well as potential influence by different types of devices and percutaneous techniques during the implantation.

We believe that the usefulness of troponin dosage after pASDc has been insufficiently studied and there are still areas of uncertainty. The cause of the presence of myocardial injury or its long-term relevance is unknown. Further prospective studies are necessary to elucidate this issue. The routinely use in a standardized protocol would be mandatory to determine which patients need a closer follow-up.

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