Coronary Embolism and Myocardial Infarction: A Scoping Study

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Abstract

Coronary embolism is a cause of acute myocardial infarction (AMI) in which obstructive foci enter the coronary circulation, block normal blood flow and precipitate ischemia. Precise studies focusing on patient population affected, pathophysiological mechanisms, and treatment strategies are scanty, in spite of a reported prevalence estimated at 2.9%. As the understanding of myocardial infarction without evidence of coronary artery disease continues to grow, an in-depth review of this previously seldomly reported subtype of coronary ischemia was in order. Patients suffering coronary embolism are 15 to 20 years younger than traditional AMI patients with a slight predominance towards male sex, which resembles the gender data of the populations affected by non-traditional myocardial infarction in published reports. While the expected prevalence rate of cardiovascular disease risk factors such as hypertension and hyperlipidemia are present, this population also has a relatively high prevalence of atrial fibrillation and valve pathology, especially endocarditis. Initial presentation is indistinguishable from other causes of myocardial infarction however fever is commonly present, when endocarditis with valvular involvement is the primary cause of the coronary embolism. Mechanical thrombectomy is the mainstay of treatment, followed by percutaneous coronary intervention. Mortality is the highest in patients who do not receive targeted treatment for the coronary embolism, particularly if only antimicrobial agents or anticoagulation without thrombolytic agents are employed. The unique features of coronary embolism highlighted in this historical study justify further examination in contemporary patient populations.

Keywords

coronary embolism; acute myocardial infarction; non-traditional myocardial infarction; paradoxical coronary embolism; infectious endocarditis; valvular vegetations; atrial fibrillation;
1. **Introduction**

Heart disease is the leading cause of morbidity and mortality worldwide [1]. In the United States, heart attack or acute myocardial infarction (AMI) has an annual incidence of about 805,000 cases with a death rate of 96.8 per 100,000 [2]. Though coronary artery disease (CAD) is the most common cause of AMI [3], infarction can happen without significant coronary artery stenosis. Myocardial infarction with nonobstructive coronary arteries (MINOCA) includes syndromes of the epicardium, microvasculature, and emerging concepts like myocardial necrosis due to oxygen supply-demand mismatch [4]. A systematic review of 27 clinical trials/AMI registries reports MINOCA prevalence of 6% of AMI cases [5].

Coronary embolism (CE) is a heterogenous cause of MINOCA that can be divided into direct, paradoxical, and iatrogenic [6]. Direct CE is when an embolism enters the coronary circulation, originating from systemic circulation or an intracardiac focus on the left side of the heart. Paradoxical CE enters coronary circulation after passing from venous circulation via a septal defect or patent foramen oval. Iatrogenic encompasses any emboli associated with a procedure, usually cardiothoracic surgery or percutaneous coronary intervention (PCI). There is overlap between the different categories and other organization schema have also been proposed.

In spite of early autopsy series identifying CE [7] and advancements in cardiovascular imaging, prevalence of CE has not yet been evaluated across multiple trials/registries. A single center, retrospective study of 1776 de novo AMI patients reports CE prevalence of 2.9% of AMI cases [8]. As it could represent almost half of MINOCA cases, CE should not be considered a rare entity. We here undertake the first scoping study of CE to understand risk factors, diagnosis, sources of emboli, management, and mortality.

2. **Methods**

On July 23rd, 2019, a systematic search was conducted using Pubmed, Google Scholar, CINAHL, Cochrane CENTRAL and Web of Science databases. Studies listing the keywords “embolism, myocardial infarction” were used to identify cases of myocardial arrhythmias associated with marijuana use. The reference list of each report was reviewed for potential additional cases. All cases were reviewed in detail. Data reviewed included demographic data, cardiovascular (CV) risk factors, electrocardiography (EKG) findings, troponin levels, transthoracic echocardiography, electrophysiology study, urine drug screen findings, coronary angiogram and management when available.
3. Results

3.1. Demographics

A total of 232 unique cases were identified in 190 publications (Table 1). Males comprised 55.2% of the cases reported and females comprised 44.8%. Overall age range was 4 months to 87 years with mean 50.2 ± 17.9 years and median 51 years; 57.3% of cases were younger than 55 years and 42.7% of cases were 55 years or older. Male age range was 4 months to 87 years with mean 49.6 ± 18.1 years and median 51 years. Female age was 12 to 87 years with mean 51.0 ± 17.9 years and median 51 years (Table 2).

3.2. Cardiovascular Risk Factors

The most prevalent cardiovascular risk factors and comorbidities in the population were as follows: atrial fibrillation 17.2%, hypertension 16.8%, hyperlipidemia 15.9%, prosthetic heart valves 15.9%, chronic valvular disease not treated with valve replacement 15.9%, rheumatic heart disease 12.5%, diabetes 9.91%, obesity 9.05%, history of CAD 9.05%, cerebrovascular disease 9.05%, and smoking 8.62%. Comprehensive risk factors and comorbidities are listed in Table 3.

3.3. Presentation

Patients presented with chest pain 66.8%, fever 19.0%, sudden death 6.03%, hypotension 3.35%, shortness of breath 3.35%, stroke 0.862%, and without symptoms 0.431% (Table 4).

3.4. ECG Findings

In the 219 cases reporting ECG findings (Table 5), ST elevation was found in 43.8%, ST depression in 11.4%, unspecified “infarct changes” in 26.5%, atrial fibrillation in 14.2%, T wave changes in 10.5%, and Q waves 3.65%.

3.5. Angiographic Findings

A total of 173 cases included angiographic findings (Table 6). Occluded arteries were found in 95.4% of cases. Lesions were found in the left anterior descending artery 52.0%, left circumflex artery 17.3%, right main coronary artery 17.3%, left main coronary artery 9.83%, obtuse marginal artery 2.89%, posterolateral artery 2.31%, diagonal artery 1.73%, and intermediate artery 0.578%.

3.6. Trans-thoracic Echocardiography

Of the 94 cases reporting TTE, the most common findings were wall motion abnormalities in 47.9% of cases, valvular dysfunction in 29.8%, systolic dysfunction in 25.5%, valve masses or vegetations in 22.3%, intracardiac masses in 18.1%, and chamber enlargement in 11.7%. Normal studies were reported in 9.57% of cases; additional findings are listed in Table 7.
3.7. Trans-esophageal Echocardiography

Transesophageal echocardiograms were reported in 63 cases. The most common findings were valves masses or vegetations in 39.7% of cases, intracardiac masses in 33.3%, and valvular dysfunction in 25.4%. Other findings are listed in Table 8.

3.8. Causes of Coronary Embolism

The reason for CE, as per the case authors, were endocarditis 22.8%, prosthetic heart valve complications 15.1%, thromboembolism 13.4%, atrial fibrillation 12.5%, iatrogenic 12.5%, non-thrombotic embolic sources (including solid tumors, bone marrow, and septic emboli) 9.91%, chronic valvular disease 8.62%, and rheumatic heart disease 6.47%. A comprehensive list of CE causes is listed in Table 9.

3.9. Management

The most common interventions performed were thrombectomy 31.0%, PCI 22.0% (stent placed in 31.4% of these cases), anticoagulation 14.7%, thrombolytic therapy 9.48%, and antibiotics/antifungals 8.19%. A comprehensive list of interventions is listed in Table 10.

3.10. Mortality

Death was reported in 35.7% of cases (Table 11), of which 34.9% occurred after no intervention was performed, 15.7% after anticoagulation, 12.0% after antibiotic/antifungal therapy alone were given, 6.02% after surgical intervention, 4.82% after aspiration thrombectomy, 3.61% after valve repair/replacement, and 2.41% after thrombolytic therapy. Recurrence of coronary embolism was reported in 7 cases, 3 of which resulted in death.

4. Discussion

The average age in the study population was approximately 15 years younger for males and 20 years younger for females than expected for all causes of MI, with females also representing a smaller percentage in all age groups relative to men than expected for all causes of MI [9]. While younger age and skew towards male sex may be due to small sample size and known underdiagnosis of MI in females [10], these demographic data correlate with findings in larger reviews of MINOCA [5]. Whether there is a unique patient population for CE should be confirmed in future studies.

Risk factors for CE overlapped with those for MI due to CAD (MI-CAD), specifically hypertension, hyperlipidemia, diabetes, CAD, CVD, and smoking. History of atrial fibrillation (AF), which is more commonly associated with cerebrovascular disease (CVD), was present in 17.2% of patients. Given the risk of thrombus formation in the left atrium in AF and its proximity to the coronary aortic cusps, thromboembolic events can occur in a similar fashion in the coronary circulation as they do in CVD. CE may occur less frequently than CVD in this setting due to the fast rate of flow of blood across the coronary ostia, high resistance to flow of the smaller caliber coronary vessels as dictated by Poiseuille’s Law, and the acute angle of the origin of the coronary arteries [11]. Interestingly, demand ischemia from AF with rapid ventricular response has also been proposed as a mechanism for MINOCA due to AF [12].
Additional risk factors in CE patients are chronic valvular disease with or without a history of rheumatic heart disease and prosthetic heart valves. Complications of chronic valvular disease and malfunctioning prosthetic valves usually include heart failure and/or pulmonary hypertension, rather than MI [13,14,15]. Abnormal blood flow and changes in left atrial volume caused by mitral valve disease, especially in AF, may cause thrombus formation [16,17] thereby contributing to thromboembolic CE. Thrombosed prosthetic valves have the potential to cause thromboembolic events [18] and may cause CE this way.

Chest pain was the presenting complaint in 66.8% of CE cases, but it is not specific for ischemia and does not distinguish MI due to CE from MI-CAD [19]. Shortness of breath, found in 3.35% of patients, is also a classic symptom for MI-CAD. Fever, found in 19.0% of cases, is an unusual symptom of an underlying cardiac issue except in infective endocarditis (IE) and pericarditis [20]. In cases where CE was later determined to have been caused by IE, fever was the initial complaint 69.8% of the time. Stroke was the initial reason for hospitalization in 2 cases, reinforcing that CE can be caused by a similar mechanism as embolic CVD.

Diagnostic investigations included ECG in 94.4% of cases, angiography in 74.6%, TTE in 40.5%, and TEE in 27.2%. The most common ECG finding was ST-elevation, followed by “infarct changes” and AF. These reports did not provide enough data to draw conclusions about how CE presents on ECG versus MI-CAD. Vessel distribution, determined angiographically, followed a similar pattern to MI-CAD [21]. However, micro-emboli and advancement of CE into small caliber vessels may lead to infarction in territories of angiographically normal vessels, contributing to underdiagnosis [22,23]. TTE identified the probable embolic source in 40.4% of cases when performed and TEE identified the probable source in 76.2%. TEE is 92% sensitive and 98% for detecting thrombi in the left atrial appendage, which is the most common area for thrombus formation [24,25,26]. TEE is 90% sensitive for native valve endocarditis and 85% sensitive for prosthetic and device-related endocarditis [27].

Causes of CE were varied, but primarily involved pathology of the aortic and mitral valves. Prevalence of endocarditis (22.8%) was high in this population despite less than 4% of patients having a known history of endocarditis, valve vegetations, and intravenous drug use. 44.4% of patients had conditions predisposing to IE, including chronic valvular disease, prosthetic valves, and rheumatic heart disease [28]. This association of CE and IE may be a useful step towards creating a predictive tool for CE, especially because the Duke criteria for predicting IE is well-validated [29]. Additionally, since the incidence of IE has increased over a 10-year period in the United States [30], it may become easier to assess prevalence of CE and the need for specific management strategies in IE patients.

Iatrogenic CE occurred most commonly in valve repair, valve replacement, and PCI at an overall rate equal to AF. Coronary catheterization is the best studied in this context, having been reported to cause CE with subtherapeutic heparinization or insufficient flushing of coagulated blood in the catheter, as well as incomplete aspiration of air [31,32]. These procedures likely have unique risks for CE, warranting further investigation.
Nearly every case reviewed initially treated the patient for presumptive MI-CAD, with antiplatelet therapy, symptomatic treatment, and angiography playing a central role. Thrombectomy, particularly aspiration thrombectomy was the most frequently chosen intervention, followed by PCI with or without stent placement. Reason for using aspiration thrombectomy versus PCI were not explicitly states in these cases, though previous international guidelines recommend routine use of aspiration thrombectomy with primary PCI and in cases of increased risk factors or high thrombus burden [17]. There was no difference in mortality at 180-days and a slightly higher incidence of stroke for patients who underwent aspiration [33]. In the present study, all patients who underwent PCI survived, while 4 deaths occurred after aspiration thrombectomy. The ability to pathological examine relatively intact aspiration specimens has enhanced current understanding of the causes of CE, in spite of its questionable survival benefits.

Anticoagulation was the most common medical therapy given, with 14.7% of patients receiving it in the hospital and 43.1% receiving it upon discharge. Thrombolytic therapy was also given, most often as the primary therapy [34], though its use is not well-studied and may even result in distalization of the thromboembolism to a smaller branch [35]. In the absence of clear guidelines, therapeutic approach to CE in patients of clinical presentation varies greatly. Reasons for not including other therapies including risk of stent infection in endocarditis [36], lack of evidence for thrombolytic therapy [37], and “free floating” emboli determined unlikely to respond to angioplasty [38].

Death occurred at a higher rate in cases where CE was not explicitly treated. Mortality was 93.5% in patients receiving no intervention or symptomatic treatment, 58.8% in patients treated with antibiotics or antifungals alone, and 48.0% in patients treated with anticoagulation alone. One study [8] showed CE MI patients had a significantly higher mortality rate (hazard ratio 3.82) and cardiac death rate (HR, 5.39; 95% CI, 2.38–10.6) than MI-CAD patients, which underscores the need for better understanding of CE.

5. Conclusion

CE is an understudied cause of MI. The at-risk populations appear to be younger and more male than the general MI-CAD population and have unique risk factors in addition to those typically associated MI-CAD, including AF and valvular heart disease. While clinical presentation and ECG was indistinct from MI-CAD, TTE and TEE often demonstrated an embolic focus. When the clinical picture is considered alongside imaging, the most likely causes of CE were determined to be endocarditis with valvular involvement, malfunctioning or thrombosed prosthetic heart valves, intracardiac thrombi, and atrial fibrillation. There are no validated diagnostic algorithms for CE and as such it should be considered a possibility alongside the more traditional causes of MI, especially when the patient has known valve pathology or hemodynamically compromised valves on echocardiography. Definitive management strategies are challenging, in part due to the heterogeneity of causes of CE. The study shows that, in cases where the embolism itself was not directly addressed by thrombectomy, PCI, or thrombolysis, CE mortality was high.
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### Table 1.

Cases of CE included in the study

| Serial Number | Year | Author              | Reference |
|---------------|------|---------------------|-----------|
| 1             | 1950 | Moragues et al.     | [39]      |
| 2             | 1952 | Walker et al.       | [40]      |
| 3             | 1952 | Glushien et al.     | [41]      |
| 4             | 1954 | Segall et al.       | [42]      |
| 5             | 1958 | Gill et al.         | [43]      |
| 6             | 1958 | Hoffman et al.      | [44]      |
| 7             | 1958 | Lillington et al.   | [45]      |
| 8             | 1958 | Kavanaugh et al.    | [46]      |
| 9             | 1959 | Boas et al.         | [47]      |
| 10            | 1959 | Gelpi et al.        | [48]      |
| 11            | 1958 | Wegner et al.       | [49]      |
| 12            | 1960 | Marietta et al.     | [50]      |
| 13            | 1960 | Winters et al.      | [51]      |
| 14            | 1961 | Menzies et al.      | [52]      |
| 15            | 1961 | Oakley et al.       | [53]      |
| 16            | 1961 | Miyahara et al.     | [54]      |
| 17            | 1962 | Jerie et al.        | [55]      |
| 18            | 1962 | Shanoff et al.      | [56]      |
| 19            | 1964 | Rivera et al.       | [57]      |
| 20            | 1965 | Liban et al.        | [58]      |
| 21            | 1965 | Harris et al.       | [59]      |
| 22            | 1966 | Watt et al.         | [60]      |
| 23            | 1967 | Ritch et al.        | [61]      |
| 24            | 1968 | Woo-Ming et al.     | [62]      |
| 25            | 1969 | Tsuchiya et al.     | [63]      |
| 26            | 1969 | Parameswaran et al. | [64]      |
| 27            | 1969 | Reddy et al.        | [65]      |
| 28            | 1969 | Hall et al.         | [66]      |
| 29            | 1971 | Benchimol et al.    | [67]      |
| 30            | 1971 | Richardson et al.   | [68]      |
| 31            | 1974 | Hartveit et al.     | [69]      |
| 32            | 1974 | Schatz et al.       | [70]      |
| 33            | 1974 | Attai et al.        | [71]      |
| 34            | 1976 | Pfeifer et al.      | [72]      |
| 35            | 1977 | Fayemi et al.       | [73]      |
| 36            | 1978 | Bedetti et al.      | [74]      |
| 37            | 1978 | Saenz et al.        | [75]      |
| Serial Number | Year | Author                  | Reference |
|---------------|------|-------------------------|-----------|
| 38            | 1979 | Sridhar et al.          | [76]      |
| 39            | 1979 | McHenry et al.          | [77]      |
| 40            | 1982 | Lin et al.              | [78]      |
| 41            | 1984 | Przybojewski et al.     | [79]      |
| 42            | 1984 | Przybojewski et al.     | [80]      |
| 43            | 1985 | Marx et al.             | [81]      |
| 44            | 1986 | Ueda et al.             | [82]      |
| 45            | 1986 | Presant et al.          | [83]      |
| 46            | 1987 | Culver et al.           | [84]      |
| 47            | 1987 | Maddoux et al.          | [85]      |
| 48            | 1987 | Ackermann et al.        | [86]      |
| 49            | 1988 | Mercereau et al.        | [87]      |
| 50            | 1988 | Backer et al.           | [88]      |
| 51            | 1988 | Jungbluth et al.        | [89]      |
| 52            | 1990 | Vasiljevic et al.       | [90]      |
| 53            | 1991 | Herzog et al.           | [91]      |
| 54            | 1992 | Valente et al.          | [92]      |
| 55            | 1993 | Bell et al.             | [93]      |
| 56            | 1995 | Eckstein et al.         | [94]      |
| 57            | 1996 | Haynes et al.           | [95]      |
| 58            | 1996 | Abascal et al.          | [96]      |
| 59            | 1997 | Iwama et al.            | [97]      |
| 60            | 1998 | Quinn et al.            | [98]      |
| 61            | 1998 | Matsumoto et al.        | [99]      |
| 62            | 1999 | Lanza et al.            | [100]     |
| 63            | 2000 | Perera et al.           | [101]     |
| 64            | 2000 | Takada et al.           | [102]     |
| 65            | 2000 | Tun et al.              | [103]     |
| 66            | 2000 | Chan et al.             | [104]     |
| 67            | 2001 | Mentzelopoulos et al.   | [105]     |
| 68            | 2002 | Aslam et al.            | [106]     |
| 69            | 2002 | Beldner et al.          | [107]     |
| 70            | 2002 | Hernández et al.        | [108]     |
| 71            | 2002 | Tobar et al.            | [109]     |
| 72            | 2003 | Hung et al.             | [110]     |
| 73            | 2003 | Ramos et al.            | [111]     |
| 74            | 2003 | Meier-Ewert et al.      | [112]     |
| 75            | 2004 | Kotooka et al.          | [113]     |
| 76            | 2004 | Mahmood et al.          | [114]     |
| Serial Number | Year | Author | Reference |
|---------------|------|--------|-----------|
| 77            | 2004 | Haghi et al. | [115] |
| 78            | 2004 | Eguchi et al. | [116] |
| 79            | 2004 | Orban et al. | [117] |
| 80            | 2004 | Petinaux et al. | [118] |
| 81            | 2005 | Ozaydin et al. | [119] |
| 82            | 2005 | Adachi et al. | [120] |
| 83            | 2014 | Deepali et al. | [121] |
| 84            | 2005 | Braun et al. | [122] |
| 85            | 2005 | Taniike et al. | [123] |
| 86            | 2005 | Pindado et al. | [124] |
| 87            | 2005 | Kirkpatrick et al. | [125] |
| 88            | 2006 | Vanoverbeke et al. | [126] |
| 89            | 2006 | Kiernan et al. | [127] |
| 90            | 2006 | Mejia et al. | [128] |
| 91            | 2006 | Cay et al. | [129] |
| 92            | 2006 | Bodor et al. | [130] |
| 93            | 2006 | Bracco et al. | [131] |
| 94            | 2006 | Breithardt et al. | [132] |
| 95            | 2006 | Wilson et al. | [133] |
| 96            | 2007 | Yazici et al. | [134] |
| 97            | 2007 | Sakai et al. | [135] |
| 98            | 2007 | Van de Walle et al. | [136] |
| 99            | 2007 | Ural et al. | [137] |
| 100           | 2007 | Greig et al. | [138] |
| 101           | 2008 | Yavari et al. | [139] |
| 102           | 2008 | Caciolli et al. | [140] |
| 103           | 2008 | Baek et al. | [141] |
| 104           | 2009 | Kessavane et al. | [142] |
| 105           | 2009 | Nanjappa et al. | [143] |
| 106           | 2009 | Murthy et al. | [144] |
| 107           | 2009 | Martín et al. | [145] |
| 108           | 2009 | Camaro et al. | [146] |
| 109           | 2009 | Lacunza-Ruiz et al. | [147] |
| 110           | 2009 | Sial et al. | [148] |
| 111           | 2009 | Budavari et al. | [149] |
| 112           | 2009 | Teixera et al. | [150] |
| 113           | 2009 | Lin et al. | [151] |
| 114           | 2009 | Dogan et al. | [152] |
| 115           | 2009 | Steinwender et al. | [153] |
| Serial Number | Year | Author                  | Reference |
|---------------|------|-------------------------|-----------|
| 116           | 2009 | Shim et al.             | [154]     |
| 117           | 2010 | Ferlan et al.           | [155]     |
| 118           | 2010 | Nakazone et al.         | [156]     |
| 119           | 2010 | Pawlaczyk et al.        | [157]     |
| 120           | 2010 | Motreff et al.          | [158]     |
| 121           | 2010 | Bae et al.              | [159]     |
| 122           | 2011 | Yuce et al.             | [160]     |
| 123           | 2011 | Levis et al.            | [161]     |
| 124           | 2011 | Saraiva et al.          | [162]     |
| 125           | 2011 | Gavrielatos et al.      | [163]     |
| 126           | 2011 | George et al.           | [164]     |
| 127           | 2011 | Najib et al.            | [165]     |
| 128           | 2011 | Acikel et al.           | [166]     |
| 129           | 2011 | Roxas et al.            | [167]     |
| 130           | 2011 | Rifai et al.            | [168]     |
| 131           | 2011 | Vasconcellos et al.     | [169]     |
| 132           | 2012 | Aykan et al.            | [170]     |
| 133           | 2012 | Martin et al.           | [171]     |
| 134           | 2012 | Marella et al.          | [172]     |
| 135           | 2012 | Bennett et al.          | [173]     |
| 136           | 2012 | Staico et al.           | [174]     |
| 137           | 2012 | Brito et al.            | [175]     |
| 138           | 2012 | Kaya et al.             | [176]     |
| 139           | 2013 | Kim et al.              | [177]     |
| 140           | 2013 | Karavelioglu et al.     | [178]     |
| 141           | 2013 | Smith et al.            | [179]     |
| 142           | 2013 | Angulo-Llanos et al.    | [180]     |
| 143           | 2013 | Zasada et al.           | [181]     |
| 144           | 2013 | Kirubakaran et al.      | [182]     |
| 145           | 2013 | Tiong et al.            | [183]     |
| 146           | 2014 | Lacunza-Ruiz et al.     | [184]     |
| 147           | 2014 | Tsang et al.            | [185]     |
| 148           | 2014 | Karaoyun et al.         | [186]     |
| 149           | 2014 | Abecasis et al.         | [187]     |
| 150           | 2014 | Giri et al.             | [188]     |
| 151           | 2014 | Gagliardi et al.        | [189]     |
| 152           | 2014 | Seo et al.              | [190]     |
| 153           | 2014 | Krikungvan et al.       | [191]     |
| 154           | 2014 | Dauvergne et al.        | [192]     |
| Serial Number | Year | Author                        | Reference |
|---------------|------|-------------------------------|-----------|
| 155           | 2014 | Steiner et al.                | [193]     |
| 156           | 2014 | Heseltine et al.              | [194]     |
| 157           | 2015 | Wee et al.                    | [195]     |
| 158           | 2015 | Senguttuvan et al.            | [196]     |
| 159           | 2015 | Plymen et al.                 | [197]     |
| 160           | 2015 | Iannaccone et al.             | [198]     |
| 161           | 2015 | Medda et al.                  | [199]     |
| 162           | 2015 | Wongrakpanich et al.          | [200]     |
| 163           | 2015 | Mallouppas et al.             | [201]     |
| 164           | 2015 | Nakamura et al.               | [202]     |
| 165           | 2015 | Mallouppas et al.             | [203]     |
| 166           | 2015 | Hartung et al.                | [204]     |
| 167           | 2015 | Sultan et al.                 | [205]     |
| 168           | 2016 | Koutsampasopoulos et al       | [206]     |
| 169           | 2016 | Castelli et al.               | [207]     |
| 170           | 2016 | Winkler et al.                | [208]     |
| 171           | 2016 | Zachura et al.                | [209]     |
| 172           | 2016 | Ito et al.                    | [210]     |
| 173           | 2014 | Sousa et al.                  | [211]     |
| 174           | 2016 | Nogales-Romo et al.           | [212]     |
| 175           | 2016 | Chikkabasavaiah et al.        | [213]     |
| 176           | 2016 | Rozado et al.                 | [214]     |
| 177           | 2017 | Rotta Detto Loria et al       | [215]     |
| 178           | 2017 | Liu et al.                    | [216]     |
| 179           | 2017 | Rivera-Juárez et al.          | [217]     |
| 180           | 2017 | Sinha et al.                  | [218]     |
| 181           | 2017 | Manchurov et al.              | [219]     |
| 182           | 2017 | Pavani et al.                 | [220]     |
| 183           | 2017 | Dallaglio et al.              | [221]     |
| 184           | 2017 | Martinez et al.               | [222]     |
| 185           | 2018 | Cvetković et al.              | [223]     |
| 186           | 2018 | Sakagami et al.               | [224]     |
| 187           | 2017 | Jiao et al.                   | [225]     |
| 188           | 2016 | Ahmad et al.                  | [226]     |
| 189           | 2017 | Cay et al.                    | [227]     |
| 190           | 2016 | Virk et al.                   | [228]     |
### Table 2.

Study population demographics (n = 232)

|                       | Total  | Males     | Females   |
|-----------------------|--------|-----------|-----------|
| Age (years)           |        |           |           |
| Mean (SD)             | 50.2 (17.9) | 49.6 (18.1) | 51 (17.7) |
| Range                 | 0.3 to 87 | 0.3 to 87 | 12 to 87  |
| Gender                |        |           |           |
| Male n (%)            | 128 (55.2) |           |           |
| Female n (%)          | 104 (44.8) |           |           |
**Table 3.**

Historical cardiovascular risk factors (n = 232)

| Risk Factor                                      | N (%)  |
|--------------------------------------------------|--------|
| Atrial fibrillation                              | 40 (17.2) |
| Hypertension                                     | 39 (16.8) |
| Hyperlipidemia                                   | 37 (15.9) |
| Chronic Valvular Disease                         | 37 (15.9) |
| Mitral valve                                     | 21 (9.05) |
| Aortic valve                                     | 16 (6.90) |
| Prosthetic heart valve                           | 37 (15.9) |
| Mitral valve                                     | 20 (8.62) |
| Aortic valve                                     | 11 (4.74) |
| Aortic and mitral valves                         | 6 (2.59) |
| Rheumatic heart disease                          | 29 (12.5) |
| Valve unspecified                                | 18 (7.76) |
| Mitral                                           | 9 (3.88) |
| Aortic                                           | 2 (0.862) |
| Diabetes                                         | 23 (9.91) |
| Coronary artery disease                          | 21 (9.05) |
| Cerebrovascular disease                          | 21 (9.05) |
| Obesity                                          | 21 (9.05) |
| Smoking                                          | 20 (8.62) |
| End stage renal disease                          | 9 (3.88) |
| Congestive heart failure                         | 7 (3.02) |
| Deep vein thrombosis/pulmonary embolism          | 7 (3.02) |
| Valve vegetations                                | 7 (3.02) |
| Cardiomyopathy                                   | 5 (2.16) |
| Atrial septal defect                              | 3 (1.29) |
| Alcohol abuse                                    | 2 (0.862) |
| Drug abuse                                       | 2 (0.862) |
| Other arrhythmia                                  | 2 (0.862) |
| Aortic dissection                                 | 2 (0.862) |
| Obstructive sleep apnea                           | 2 (0.862) |
| Anemia                                           | 2 (0.862) |
| Patent foramen ovale                              | 2 (0.862) |
| Pregnancy                                        | 2 (0.862) |
| Systemic lupus erythematosus                     | 2 (0.862) |
| Intracardiac thrombus                             | 2 (0.862) |
| Arteritis                                        | 1 (0.431) |
| Aortic coarctation                               | 1 (0.431) |
| Hypertrophic obstructive cardiomyopathy          | 1 (0.431) |
| Hyperthyroidism                                  | 1 (0.431) |
| Condition                  | N (%)       |
|---------------------------|-------------|
| Liver disease             | 1 (0.431)   |
| Ventricular septal defect | 1 (0.431)   |
| Endocarditis              | 1 (0.431)   |
| Heart transplant          | 1 (0.431)   |
| Tuberculosis              | 1 (0.431)   |
Table 4.

Presenting complaints (n = 232)

| Complaint          | n   | (%)  |
|--------------------|-----|------|
| Chest pain         | 157 | 66.8 |
| Fever              | 44  | 19.0 |
| Sudden death       | 14  | 6.03 |
| Hypotension        | 8   | 3.35 |
| Shortness of breath| 8   | 3.35 |
| Stroke             | 2   | 0.862|
| Asymptomatic       | 1   | 0.431|
### Table 5.

**Infarct ECG findings (n = 219)**

|                      | n (%)  |
|----------------------|--------|
| ST changes           | 114 (52.1) |
| ST elevation         | 96 (43.8)  |
| ST depression        | 25 (11.4)  |
| ST change, unspecified| 3 (1.37)  |
| Infarct changes, unspecified | 58 (26.5) |
| Atrial fibrillation  | 31 (14.2)  |
| T wave changes       | 23 (10.5)  |
| T wave inversion     | 18 (8.22)  |
| T wave depression    | 2 (0.913)  |
| T wave change, unspecified | 2 (0.913) |
| Peaked T wave        | 1 (0.457)  |
| Q waves              | 8 (3.65)   |
### Table 6.
Location of lesion on cardiac catheterization (n = 173)

| Location                       | n   | (%)  |
|--------------------------------|-----|------|
| Left anterior descending artery| 90  | 52.0 |
| Left circumflex artery         | 30  | 17.3 |
| Right main coronary artery     | 30  | 17.3 |
| Left main coronary artery      | 17  | 9.83 |
| Posterior descending artery    | 8   | 4.62 |
| No findings                    | 8   | 4.62 |
| Obtuse marginal artery         | 5   | 2.89 |
| Posterolateral artery          | 4   | 2.31 |
| Diagonal artery                | 3   | 1.73 |
| Intermediate artery            | 1   | 0.578|
Table 7.

Transthoracic echocardiography findings (n = 94)

| Condition                      | N (%)  |
|-------------------------------|--------|
| Wall motion abnormalities     | 45 (47.9) |
| Valvular dysfunction          | 28 (29.8) |
| Mitral regurgitation          | 13 (13.8) |
| Aortic regurgitation          | 9 (9.57) |
| Tricuspid regurgitation       | 3 (3.19) |
| Prosthetic valve              | 2 (2.13) |
| Aortic stenosis               | 1 (1.06) |
| Mitral valve prolapse         | 1 (1.06) |
| Systolic dysfunction          | 24 (25.5) |
| Valve mass or vegetation      | 21 (22.3) |
| Intracardiac mass             | 17 (18.1) |
| LV                            | 8 (8.51) |
| LA                            | 7 (7.45) |
| RA                            | 2 (2.13) |
| Chamber enlargement           | 11 (11.7) |
| RV                            | 5 (5.32) |
| LV                            | 3 (3.19) |
| RA                            | 2 (2.13) |
| LA                            | 1 (1.06) |
| Normal                        | 9 (9.57) |
| Pulmonary hypertension        | 4 (4.26) |
| PFO                           | 3 (3.19) |
| Cardiomyopathy                | 2 (2.13) |
| Aortic dissection             | 1 (1.06) |
Table 8.

Traneseophageal echocardiography findings (n = 63)

| Condition                        | N   | (%)  |
|----------------------------------|-----|------|
| Valve mass or vegetation         | 25  | (39.7) |
| Intracardiac mass                | 21  | (33.3) |
| LA                               | 17  | (27.0) |
| LV                               | 2   | (3.17) |
| RA                               | 2   | (3.17) |
| Valvular dysfunction             | 16  | (25.4) |
| Mitral regurgitation             | 9   | (14.3) |
| Prosthetic valve                 | 3   | (4.76) |
| Aortic regurgitation             | 2   | (3.17) |
| Aortic stenosis                  | 1   | (1.59) |
| Mitral valve prolapse            | 1   | (1.59) |
| Wall motion abnormalities        | 8   | (12.7) |
| Normal                           | 8   | (12.7) |
| PFO                              | 6   | (9.52) |
| Systolic dysfunction             | 6   | (9.52) |
| Aortic mass                      | 3   | (4.76) |
| Pulmonary embolism               | 1   | (1.59) |
| Gas bubbles                      | 1   | (1.59) |
Table 9.

Reason for thromboembolism (n = 232)

| Reason                        | n (%)    |
|-------------------------------|----------|
| Endocarditis                  | 53 (22.8)|
| Valve unspecified             | 23 (9.91)|
| Aortic valve                  | 11 (4.74)|
| Mitral valve                  | 10 (4.31)|
| Aortic and mitral valves      | 5 (2.16) |
| Mitral and tricuspid valves   | 1 (0.431)|
| Prosthetic heart valves       | 35 (15.1)|
| Mitral valve only             | 19 (8.20)|
| Aortic valve only             | 10 (4.31)|
| Aortic and mitral valve       | 6 (2.59) |
| Thrombus                      | 31 (13.4)|
| Left ventricular thrombus     | 9 (3.88) |
| DVT                           | 9 (3.88) |
| Arterial thrombus             | 5 (2.16) |
| Left atrial thrombus          | 3 (1.29) |
| Right atrial thrombus         | 2 (0.862)|
| Pulmonary artery thrombus     | 2 (0.862)|
| Aortic cusp thrombus          | 1 (0.431)|
| Atrial fibrillation           | 29 (12.5)|
| Procedure/iatrogenic          | 29 (12.5)|
| Valve repair/replacement      | 11 (4.74)|
| PCI                           | 6 (2.59) |
| Ablation                      | 3 (1.29) |
| Coronary angiography          | 2 (0.862)|
| ASD/VSD repair                | 2 (0.862)|
| Ascending aortic dissection repair | 1 (0.431)|
| AV cusp decalcification       | 1 (0.431)|
| Hip replacement               | 1 (0.431)|
| Central venous nutrition injection | 1 (0.431)|
| Radial endarterectomy         | 1 (0.431)|
| Non-thrombotic embolic source | 23 (9.91)|
| Solid tumor                   | 20 (8.62)|
| Septic                        | 2 (0.862)|
| Bone marrow                   | 1 (0.431)|
| Chronic valvular disease      | 20 (8.62)|
| Mitral valve                  | 14 (6.03)|
| Aortic valve                  | 6 (2.59) |
| Rheumatic heart disease       | 15 (6.47)|
| Mitral valve                  | 10 (4.31)|
| Condition                      | n (%)  |
|-------------------------------|--------|
| Valve unspecified             | 4 (1.72) |
| Aortic valve                  | 1 (0.431) |
| PFO                           | 11 (4.74) |
| Hypercoagulable state         | 8 (3.35) |
| Cardiomyopathy                | 8 (3.35) |
| ASD                           | 5 (2.16) |
| Unknown                       | 5 (2.16) |
| MVP                           | 2 (0.862) |
| Other arrhythmia              | 1 (0.431) |
| VSD                           | 1 (0.431) |
| Double chamber LV             | 1 (0.431) |
Table 10.

Interventions (n = 232)

| Intervention                        | n (%) |
|-------------------------------------|-------|
| Thrombectomy                        | 72 (31.0) |
| Aspiration thrombectomy             | 53 (22.8) |
| Mechanical thrombectomy             | 11 (4.74) |
| Surgical thrombectomy               | 8 (3.45) |
| PCI                                 | 51 (22.0) |
| Without stent placement             | 31 (13.4) |
| With stent placement                | 16 (6.90) |
| Anticoagulation alone               | 25 (10.8) |
| No intervention                     | 31 (13.4) |
| Thrombolytic therapy                | 22 (9.48) |
| Antibiotics/antifungal              | 19 (8.19) |
| Antibiotics/antifungal alone        | 17 (7.33) |
| Excision of embolic source          | 18 (7.76) |
| Valve surgery                       | 18 (7.76) |
| Intervention not reported           | 18 (7.76) |
| CABG                                | 8 (3.45) |
| Antiplatelet therapy alone          | 6 (2.58) |
| PFO closure                         | 2 (0.862) |
| Unspecified surgical intervention    | 2 (0.862) |
| Heart transplant                    | 1 (0.431) |
| RVAD                                | 1 (0.431) |
| Femoral vein clamp                  | 1 (0.431) |
Table 11.
Mortality and intervention attempted (n = 83)

| Intervention                      | n   | (%)  |
|-----------------------------------|-----|------|
| No intervention                   | 29  | 34.9 |
| Anticoagulation                   | 13  | 15.7 |
| Anticoagulation alone             | 12  | 14.5 |
| Antibiotics/antifungals alone     | 10  | 12.0 |
| Surgical intervention             | 5   | 6.02 |
| Aspiration thrombectomy           | 4   | 4.82 |
| Valve surgery                     | 3   | 3.61 |
| Thrombolytic therapy              | 2   | 2.41 |