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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;

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cardiovascular risk factors; prosthetic heart valve; thrombolytic therapy; management of coronary embolism

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]. ln 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	Karavelioğlu 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	Kitkungvan 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)

	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)

	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)

	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)

	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)

	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 (18.10
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.

Transesophageal echocardiography findings (n = 63)

	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)

	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)

	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)

	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	34 (14.7)
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)

	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)