**Cardiopulmonary resuscitation-associated heart injuries are time related and can lead to misdiagnosis in sudden arrhythmic death syndrome**

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**ABSTRACT**

**Background:** Sudden arrhythmic death syndrome (SADS), defined as sudden cardiac death with a morphologically normal heart at autopsy, is an important cause of sudden death. Cardiopulmonary resuscitation (CPR) may induce injuries that mimic pathologic conditions. Detailed characterisation of CPR-related injuries in contemporary, large sudden death cohorts and whether they could confound SADS diagnosis are not described.

**Methods:** Retrospective observational study analysing all consecutive cases of sudden death prospectively referred to a United Kingdom national cardiac pathology centre between January 2017 and November 2021. Cases displaying CPR-related injuries were identified after review of clinical information and examination by expert cardiac pathologists.

**Results:** Out of 2,568 reviewed cases, 126 (4.9%) showed CPR-related injuries. Macroscopically, the commonest finding was left ventricular subendocardial haemorrhage (13.5%). Microscopically, haemorrhage and contraction band necrosis (37.7%), subendocardial acute infarction with necrotic myocytes and neutrophils (34.1%), interstitial mixed inflammatory cell infiltrates (25.4%), and healing granulation tissue (7.1%) were the most frequent histologic abnormalities and correlated to duration of survival following resuscitation. In a subcohort of 41 cases, autopsy pathologists failed to correlate these injuries to CPR in 22 (53.6%) cases and misinterpreted them as ischaemic myocardial infarction (n=7; 17%), myocarditis (n=5; 12.1%), or other pathologies (n=2; 4.8%) in 14 SADS cases.

**Conclusion:** This study provides a comprehensive characterisation of CPR-related injuries, which are time related and will help facilitate their identification by pathologists. CPR-related injuries significantly led to diagnostic confusion among pathologists. Specialist cardiac pathology assessment improved SADS diagnostic yield, prompting further genetic screening of decedents’ family members.

**Key-words:** Sudden arrhythmogenic death syndrome; autopsy; cardiopulmonary resuscitation.

**Introduction**

Sudden arrhythmic death syndrome (SADS) has emerged as an important cause of sudden cardiac death (SCD), especially among the young, with a prevalence of up to 40% in this setting (1, 2). SADS is defined as sudden cardiac death affecting an individual with a morphologically normal heart and negative toxicological results (3). Post-mortem examination, including histologic assessment of the heart, is essential to exclude non-cardiac causes of death and to ascertain the absence of pathologic features in the heart, prompting a diagnosis of SADS in the correct clinical context. Importantly, post-mortem diagnosis of SADS subsequently guides clinical decisions pertaining genetic testing of decedents’ relatives given that abnormalities predisposing to inherited cardiac disease and fatal cardiac arrythmias are found in 22% to 53% of asymptomatic family members (4, 5).

Post-mortem examination of the heart can be challenging, with autopsy pathologists frequently seeking expert opinion in cases of presumed sudden cardiac death. Notably, morphologic findings associated with cardiac arrest and cardiopulmonary resuscitation (CPR) may introduce diagnostic uncertainty in the assessment of cardiac tissue (6). For instance, irregular subendocardial haemorrhage, subendocardial infarction, and contraction band necrosis, which have previously been described in the context of CPR, can mimic histologic features seen in ischaemic myocardial infarction or other cardiac pathologies (7, 8). Therefore, failure to recognise such patterns of injury as iatrogenic consequences of CPR could undermine the diagnosis of SADS by pathologists.

Although a few CPR-related histologic features have been previously described in the heart, such observations were derived from animal studies or limited clinical cohorts (7-10). In addition, because most of these morphologic studies date back from the 1960’s to the 1980’s (7, 9, 10), it is unknown how current CPR interventions, recently introduced automated chest compression devices, and longer survival due to more effective post-CPR medical care affect macroscopic and histologic appearances of the heart (11, 12). Finally, there are no studies that have investigated whether CPR-related histologic features could confound the diagnosis of SADS in large cohorts of sudden death patients.

This study aimed to (i) describe morphologic patterns of CPR-related injury in the heart; (ii) explore a potential temporal relationship between distinct histologic findings and CPR-to-death time; and (iii) assess whether the presence of CPR-related injuries could confound the diagnosis of SADS in a large, contemporary cohort of presumed sudden cardiac death cases referred to a national cardiac pathology centre.

**Methods**

**Study setting**

All cases of unexpected death in the United Kingdom are referred to the coroner, who establishes the need for a post-mortem examination. The Cardiac Risk in the Young (CRY) Centre based at St George’s University, London, provides a national expert cardiac pathology service for cases voluntarily referred by coroner’s pathologists when there is unexplained, presumed sudden cardiac death. All referred cases undergo a complete post-mortem examination at the referring centre. Cases are referred either as whole hearts or haematoxylin and eosin (H&E)-stained cardiac tissue sections. Ethical and research governance approval have been granted for this study (10/H0724/38).

**Clinical data and pathology assessment**

Clinical data and histopathology reports for all cases consecutively referred to the CRY centre between January 2017 and November 2021 were reviewed for identification of cases with CPR-related injuries. During the study time frame, data was prospectively collected from coroner’s autopsy request forms, primary care records, hospital notes, and CRY referral forms where appropriate at time of referral. Clinical data included demographics, previous medical history, prescribed medications, circumstances leading to death, duration of CPR efforts, and survival time after return of spontaneous circulation (ROSC) in cases of successful CPR.

Autopsy reports were reviewed for all cases prior to specialist cardiac pathology assessment, where available. All referred cases underwent specialist assessment by experienced cardiac pathologists (M.N.S. and J.W.). The following anatomical regions were routinely examined using H&E staining: the anterior, lateral, and posterior right ventricular walls; the right ventricular outflow tract; the interventricular septum; the anterior, lateral, and posterior left ventricular walls; the three main coronary arteries; and the ascending aorta.

**Clinicopathologic definitions**

Cases of SADS were defined as sudden death with morphologically normal hearts in the absence of positive toxicology results or an alternative non-cardiac cause of death (3). To assess a possible relationship between histologic findings and survival after CPR, time from CPR to death was defined as follows: (i) 1 day in cases of unsuccessful CPR; or (ii) 24h intervals following ROSC in cases of successful CPR.

Where more than one CPR-related histologic finding was identified in the same heart those were accounted separately for estimation of frequency and relation to CPR-to-death time.

**Statistical analysis**

Continuous data normality was assessed by data distribution visualisation as well as the Shapiro-Wilk test. Differences in distribution of CPR-related injuries among distinct anatomical locations within the heart were estimated using the Chi-square test. Differences in CPR-to-death time between different histologic patterns of injury were estimated using the Kruskal-Wallis test with Bonferroni correction for multiple comparisons. A *p*-value < 0.05 was considered statistically significant. Data was summarised as mean and standard deviation (±SD) or median and interquartile range (IQR) as appropriate. Statistical analysis was performed by SPSS Statistics software v.28.0 (IBM Corp, Armonk, New York, USA).

**Results**

A total of 2,568 consecutive cases referred to our department by more than 100 centres across the UK over the 5-year period were reviewed to identify those with macroscopic and/or microscopic features of CPR-related injuries. Such findings were described in a subset of 126 (4.9%) cases among the initial cohort. Cardiac specimens with CPR-related injuries were referred either as whole hearts (n = 84; 66.6%) or as H&E-stained heart tissue sections (n = 42; 33.4%). The mean age at death was 37.1 (±16.3) years and most cases were male (n = 71; 56.3%). The mean survival time following CPR was 2.7 (±3.4) days.

Various macroscopic injury patterns following CPR were observed. On external examination of whole hearts, 9 (10.7%) cases showed evidence of epicardial haemorrhage ranging from small petechiae in the epicardial fat surrounding blood vessels to extensive haemorrhage in the epicardial fat at the atrioventricular groove extending into the atrial subendocardial tissue (**Figures 1A** – **1C**). Within the heart itself, there was subendocardial haemorrhage (SEH) in 17 (13.5%) cases, predominantly circumferentially within the left ventricular walls but also within the interventricular septum (**Figure 1D**) or focally confined to the trabeculae and papillary muscles (**Figure 1E**). Most severe changes with circumferential haemorrhage in mid ventricular wall and subendocardium were noted in cases in which a mechanical Lucas machine was used (**Figure 1E**).

A wide range of histologic features was identified, with distinct injury patterns often occurring simultaneously in a single case and exhibiting a clear temporal relationship with CPR-to-death time (**Figure 2** and **Table 1**). The most frequent finding was widespread interstitial haemorrhage with leakage from capillaries and myocyte contraction bands (n=50; 39.7%)(**Figures 2A** – **2D**), which was noted more frequently within the left ventricle (46%; p = 0.042) in cases which did not survive initial CPR or who died within 24h of ROSC (**Table 1**). Haemorrhage into the conduction system was seen in 4 (3%) cases (**Figure 2C**). Widespread interstitial and/or perivascular inflammatory cell infiltrates with neutrophils, lymphocytes, and macrophages associated with oedema, separation of myocytes, and individual cell myocytolysis were equally observed within both ventricles (p = 0.051) and atria in the initial 2 days post-successful CPR in 31 (24.6%) cases (**Table 1**; **Figure 2E**). This was followed by established subendocardial acute infarction with necrotic hypereosinophilic myocytes surrounded by infiltrating neutrophils in individuals that survived 1 to 5 days after initial resuscitation (n=44; 34.9%), predominantly within the left ventricle (68.2%; p = 0.035) (**Table 1**; **Figure 2F**). Histologic hallmarks of healing granulation tissue with myofibroblasts, newly formed capillaries, macrophages, and lymphocytes were noted within the subendocardium in 9 (7.1%) cases significantly later at 7 to 11 days after CPR (p<0.001 compared to mixed inflammatory cell infiltrates, haemorrhage, and contraction band necrosis; p=0.010 compared to subendocardial acute infarction) (**Figure 2G**). One case of SADS had evidence of focal subendocardial fibrosis 8 years after initial cardiac arrest with survival (**Figure 2H**).

CPR-related injuries co-existed with a spectrum of different cardiac pathologies but were most frequently seen in otherwise morphologically normal hearts (**Table 2**). In 75 (59.5%) cases, the cause of death following specialist cardiac pathology examination was sudden death with a morphologically normal heart, either due to SADS (n = 55; 43.6%) or to non-cardiac causes (n = 20; 15.9%) (**Table 2**). The remaining cases were almost equally distributed among other aetiologies of sudden cardiac death, (**Table 2**). Since most CPR-related injuries were seen in cases of SADS, it was hypothesized that they could confound the assessment of morphologically normal hearts by general pathologists and consequently the diagnosis of SADS.

To explore this hypothesis, a subset of 41 (32.5%) cases containing preliminary histopathologic reports by autopsy pathologists at time of referral were compared to specialist cardiac pathologist reports. Overall, there was disagreement in 22 (53.6%) cases (**Table 3**). The nature of the disagreement consisted in general pathologists either (i) failing to correlate histologic features to CPR and subsequently referring such cases for expert opinion on their clinical significance; or (ii) entirely misinterpreting CPR-related injuries as pathologic and referring the case for expert confirmation of a preliminary misdiagnosis. Noteworthy, three cases had been referred by forensic or cardiology teams following assessment by an autopsy pathologist to seek an expert opinion due to incongruent clinicopathologic correlations. In 14 (34.1%) cases of SADS, CPR-related histologic features had been initially interpreted by autopsy pathologists as ischaemic myocardial infarction (n = 7; 17%), myocarditis (n = 5; 12.1%), haemorrhage into the conduction system due to viral lower respiratory tract infection (n = 1; 2.4%) or hydropic myocyte change of uncertain significance (n = 1; 2.4%) (**Table 3**). Similarly, in 5 (12.1%) cases of sudden death from non-cardiac causes with morphologically normal hearts, CPR-related injuries were also initially interpreted as pathologic, introducing uncertainty in the formulation of cause of death (**Table 3**). In addition, injury patterns associated with CPR obscured diagnostic morphologic features of arrhythmogenic cardiomyopathy (ACM; n =1) and obesity cardiomyopathy (n = 1) (**Table 3**). Finally, accurate diagnosis of SADS, ACM, and obesity cardiomyopathy cases (n = 16; 39%) facilitated by the identification of iatrogenic CPR-related histologic findings prompted further action in terms of cardiology assessment and genetics testing of family members.

**Discussion**

In this study including a large contemporary cohort of sudden death cases referred to a national cardiac pathology service, we describe various macroscopic and microscopic patterns of CPR-related injury at post-mortem examination of the heart and their relationship to survival time following resuscitation. In addition, we show that CPR-related injuries, more often observed in morphologically normal hearts, misled referring pathologists to suspect of common cardiac pathologies such as ischaemic heart disease and myocarditis in most referred cases. More importantly, accurate identification of CPR-related morphologic features by expert cardiac pathology examination was translated in improved diagnosis of SADS and prompted referral for cardiological assessment of family members.

Cardiopulmonary resuscitation is a key element in a chain of interventions that aims to increase the chance of survival with preservation of neurological function following cardiac arrest (13). Despite being potentially lifesaving, the current CPR technique of external cardiac massage either by manual or automated chest compressions can lead to a myriad of skeletal and non-skeletal injuries due to repetitive blunt force trauma (11, 14-17). Traumatic injuries to the heart, such as epicardial contusions, intramural haematomas, and myocardial lacerations, have been reported in 2.8% to 12.8% of patients subjected to CPR (15, 16). In this study, no life-threatening macroscopic injuries to the heart were seen, which reinforces that bystander or first responder CPR should not be withdrawn due to fears of potential damage to the heart. The most frequent macroscopic CPR-related finding was subendocardial haemorrhage, which has been previously associated with higher level of adrenaline use and prolonged resuscitation efforts (18). In addition, extensive circumferential SEH was seen more frequently with use of the automated LUCAS® chest compression device. While such devices may lead to more severe visceral injuries as a result of higher mechanical forces compared to manual CPR (11, 17), it is also possible that they may be allowing the transit of individuals who would have previously died due to prolonged downtime to reach intensive care, which would result in more extensive SEH due to reperfusion injury. Because SEH may occur in the context of ischaemic myocardial infarction, our findings reiterate that autopsy pathologists should be aware of CPR-related SEH as a diagnostic pitfall in the absence of significant coronary artery disease, lack of correlation with coronary artery supply territory distribution, or haemorrhage focally confined to the papillary muscles and trabeculae (6). We recommend that such cases be referred to cardiac pathologists for expert examination.

To the best of our knowledge, this is the first study to demonstrate a clear relationship between different microscopic features of CPR-related injury and survival time post-resuscitation. Contraction band necrosis (CBN) associated with haemorrhage were the most frequent microscopic CPR-related injuries, predominantly observed in patients who did not survive CPR or who died within 24h of resuscitation. CBN, classically associated with acute myocardial infarction, can be induced by cardiac massage itself and has increased frequency in individuals receiving catecholamines and defibrillation as part of resuscitation efforts (8, 9, 19, 20). Of note, haemorrhage into the sinoatrial node and atrioventricular conduction system observed in our study has also been previously reported in patients subjected to CPR (21). It is important to recognise that haemorrhage into the conduction system can be an iatrogenic consequence of CPR as its occurrence in a different clinical context could be fatal and lead to misdiagnosis regarding the cause of death, as exemplified by one of our cases. The second most prevalent injury pattern observed in this study was subendocardial myocyte necrosis associated with neutrophils, which occurred between 1 to 5 days post-resuscitation. Accordingly, a small series of 3 paediatric autopsy cases reported the presence of myocyte necrosis and dystrophic calcification in the heart of infants who survived from 9 to 60 days post-resuscitation (22). In addition, our study also showed occurrence of healing granulation tissue between 7 to 11 days post-CPR and subendocardial fibrosis in one case with history of successful reperfusion 8 years prior to death. These are not though to be due to CPR itself but rather manifestations of tissue repair in response to hypoperfusion injury associated with cardiac arrest in those individuals who survived long enough after successful CPR for such reparative mechanisms to take place. Taken together, these findings demonstrate that CPR-related morphologic features in the heart may encompass different stages of tissue injury and repair according to the length of time elapsed between ROSC and death. Therefore, pathologists should be aware that not only history of CPR but also survival time post-CPR are important factors in the assessment of the heart at autopsy.

This study also showed that CPR-related injuries were a significant confounder for accurate assessment of hearts at autopsy as demonstrated by the substantial level of diagnostic disagreement (53.6%) between referring and cardiac pathologists. This is higher than previously reported by a study that found a 41% discrepancy rate pertaining the diagnosis of cardiac pathologies in a large cohort of sudden death cases (23), suggesting that CPR-related changes might add an extra layer of complexity to the already challenging histopathological evaluation of such cases. In our study, the disagreement largely centred around the diagnostic suspicion of ischaemic myocardial infarction and myocarditis by referring pathologists. This might be explained by the fact that most CPR-related changes consisted of myocyte contraction bands and coagulative necrosis as well as interstitial inflammatory cell infiltrates, which are histologic features observed in these two conditions, respectively. Noteworthy, inflammatory cell infiltrates in CPR cases were widespread in the heart and associated with only individual cell myocytolysis as opposed to more focal distribution and significant myocyte damage typically seen in myocarditis. Inability to associate such findings to CPR and misinterpretation as pathologic features could have hindered the diagnosis of SADS. Our study demonstrated that specialist cardiac pathology assessment was able to improve the diagnosis of SADS by 34% in this cohort, which resulted in appropriate referral of such cases to cardiologists with an interest in inherited cardiac diseases for consideration of genetic screening of family members. Hence, clinical teams should consider referral for second opinion by cardiac pathologists if there are incongruent clinicopathologic correlations following examination by autopsy pathologists and a suspicion of SADS, as indeed happened in three of our cases.

This study has some limitations. First, the higher frequency of CPR-related morphologic changes in SADS compared to other causes of death might reflect a selection bias as our service has a special interest in SADS. Second, given that this is a single centre study, potential interpretation bias cannot be entirely excluded. However, most of our cases were reviewed by two experienced cardiac pathologists who reached diagnostic agreement prior to issuing a histopathological report in order to mitigate systematic diagnostic errors. Moreover, there was missing data for CPR-to-death time in 14 (11%) cases as this was reliant on documentation provided by referring centres. Accurate information regarding the use of vasopressors and direct current defibrillation during resuscitation attempts was unavailable for most cases, hence the distribution of CPR-related injuries between these treatment modalities was not ascertained. Finally, we did not perform immunohistochemical profiling of the inflammatory cell infiltrates in our cases, which should be further explored in future studies.

In conclusion, we provided a comprehensive characterisation of CPR-related changes in the heart whilst describing their temporal association with survival time post-CPR, which will help facilitate their identification by autopsy pathologists. We also demonstrated that CPR-related histomorphologic features led to diagnostic confusion among autopsy pathologists. The establishment of regional or national cardiac pathology networks may be an important step towards widening access to specialist examination of cases with equivocal features, especially as we showed that expert assessment improved SADS diagnostic yield and prompted further genetic screening of decedents’ family members. Finally, our findings provide evidence for a role of genetic testing in all cases of SCD in young individuals to allow identification of genetic causes behind the cardiac arrest and help avoid confusion among non-expert pathologists.

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**Figures**

**Figure 1.**

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**Figure 2.**

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**Figure 1. Macroscopic appearances of CPR-related injuries. A)** Posterior view of heart showing small epicardial petechiae along the trajectory of blood vessels. **B)** Lateralview of the right atrioventricular groove showing prominent haemorrhage into the epicardial fat. **C)** Photograph of the endocardial surface of the right atrium depicting foci of haemorrhage between the fossa ovalis and the ostium of the coronary sinus. **D)** View of left ventricle opened longitudinally along its lateral wall showing extensive haemorrhage in the interventricular septum. **E)** Cranial view of transverse sections of different hearts at the mid-papillary muscle level showing the spectrum of haemorrhagic changes associated with CPR within the left ventricle, with discrete haemorrhage confined to the papillary muscles and trabeculae (upper panel), more prominent haemorrhage extending into the subendocardium (middle panel), and extensive, circumferential haemorrhage extending to the mid ventricular wall (lower panel).

**Figure 2. Spectrum of microscopic injury patterns associated with cardiopulmonary resuscitation. A)** Photomicrograph of the right atrium showing extensive intramyocardial haemorrhage (H&E staining, 40x magnification); **B)** Photomicrograph showing extensive haemorrhage into the conduction system (H&E staining, 40x magnification); **C)** Histologic section featuring haemorrhage confined within a left ventricular papillary muscle (H&E staining, 40x magnification); **D)** Histologic section of left ventricular myocardium featuring contraction band necrosis and haemorrhage with leakage from capillaries (H&E staining, 200x magnification); **E)** Photomicrograph of left ventricular myocardium showing interstitial oedema and widespread mixed acute and chronic inflammatory cells associated with individual myocytolysis (H&E staining, 200x magnification); **F)** Photomicrograph showing acute myocardial infarction associated with an interstitial neutrophilic inflammatory cell infiltrate (H&E staining, 100x magnification); **G)** Histologic section of papillary muscle displaying a focus of granulation tissue comprising new capillaries, myofibroblasts, lymphocytes, and macrophages (H&E staining, 100x magnification); **H)** Photomicrograph showing foci of replacement-type fibrosis within a papillary muscle (H&E staining, 100x magnification).

**Tables**

**Table 1. Frequency and anatomical location of microscopic findings of reperfusion injury associated with cardiopulmonary resuscitation and their relation to survival time following CPR.**

|  |  |  |  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- | --- | --- | --- |
| Microscopic findings | n (%) | Anatomical location within the heart | | | | | | | CPR-to-death time, days  [median (IQR)] |
| LV | RV | LV + RV | LV + IVS | PM | CS | p-value§ |
| Subendocardial haemorrhage and CBN | 50 (39.7) | 23 (46%) | 2 (4%) | 14 (28%) | 2 (4%) | 5 (10%) | 4 (8%) | **0.042** | 1 (1 – 2) |
| Subendocardial acute infarction with necrotic myocytes and neutrophils | 44 (34.9) | 30 (68.2%) | 1 (2.3%) | 6 (13.6%) | 3 (6.8%) | 4 (9.1%) | 0 (0%) | **0.035** | 2 (1 – 5)\* |
| Interstitial mixed inflammatory cell infiltrate | 31 (24.6) | 14 (45.2%) | 5 (16.1%) | 10 (32.3%) | 2 (6.5%) | 0 (0%) | 0 (0%) | 0.051 | 1 (1 – 1.25) |
| Subendocardial granulation tissue | 9 (7.1) | - | - | - | - | - | - | - | 9.5 (7 – 11.5)\*# |
| Subendocardial fibrosis | 1 (0.7) | - | - | - | - | - | - | - | 8 years |

An individual case may be represented in more than one category of microscopic findings. CBN, contraction band necrosis; CPR, cardiopulmonary resuscitation; CS, conduction system; IQR, interquartile range; IVS, interventricular septum; LV, left ventricle; PM, papillary muscles; RV, right ventricle. \* p< 0.001 compared to interstitial inflammatory cell infiltrate and haemorrhage and contraction band necrosis; # p = 0.010 compared to subendocardial acute infarction with necrotic myocytes and neutrophils (Kruskall-Wallis test with Bonferroni correction for multiple comparisons). § p-value estimated by Chi-square test. n = 126; missing data regarding timing from CPR to death in 14 (11%) cases.

**Table 2. Causes of death following specialist cardiac pathologist assessment in cases with CPR-related injury.**

|  |  |
| --- | --- |
| Morphologic diagnosis | n (%) |
| Sudden death with morphologically normal heart | 75 (59.5) |
| Sudden arrhythmic death syndrome (SADS) | 55 (43.6) |
| Definite non-cardiac aetiology\* | 20 (15.9) |
| Idiopathic left ventricular hypertrophy | 10 (7.8) |
| Inherited cardiomyopathies | 9 (7) |
| Arrhythmogenic cardiomyopathy | 6 (4.7) |
| Dilated cardiomyopathy | 2 (1.5) |
| Hypertrophic cardiomyopathy | 1 (0.8) |
| Ischaemic heart disease | 6 (4.7) |
| Sudden death in congenital heart disease | 5 (3.9) |
| Hypertensive heart disease | 4 (3.14) |
| Obesity cardiomyopathy | 4 (3.14) |
| Mucoid degeneration of mitral valve | 4 (3.14) |
| Myocarditis NOS | 2 (1.5) |
| Other diagnoses# | 7 (5.5) |

CPR, cardiopulmonary resuscitation; NOS, not otherwise specified; \* non-cardiac aetiologies included drug overdose, sepsis, alcohol abuse, pulmonary embolism, and sudden unexpected death in epilepsy (SUDEP); **#** other diagnoses included post-operative deaths (n=3), cardiac sarcoidosis (n=1), fibroelastosis (n=1), commotio cordis (n=1), and transplant vasculopathy (n=1).

**Table 3. Diagnostic discrepancies between referring pathologists and specialist cardiac pathologists concerning cases with CPR-related injuries.**

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
| General pathologist  diagnostic opinion  prior to referral | n (%) | Specialist cardiac pathologist diagnosis | n (%) | Prompted further clinical action(\*) |
| Ischaemic myocardial infarction | 10 (24.3) | SADS | 7 (17.1) | Yes |
|  |  | ACM | 1 (2.4) | Yes |
|  |  | Morphologically normal heart | 1 (2.4) | No |
|  |  | Mitral valve degeneration | 1 (2.4) | No |
| Myocarditis | 9 (21.9) | SADS | 5 (12.2) | Yes |
|  |  | Morphologically normal heart | 3 (7.3) | No |
|  |  | Obesity cardiomyopathy | 1 (2.4) | Yes |
| Inflammation associated with heroin use | 1 (2.4) | Morphologically normal heart | 1 (2.4) | No |
| Haemorrhage into conduction tissue due to viral LRTI | 1 (2.4) | SADS | 1 (2.4) | Yes |
| Hydropic myocyte change of uncertain significance | 1 (2.4) | SADS | 1 (2.4) | Yes |

(\*) Referral of family members to cardiologist with a special interest in inherited cardiac diseases for consideration of genetic screening. ACM, arrhythmogenic cardiomyopathy; CPR, cardiopulmonary resuscitation; LRTI, lower respiratory tract infection; SADS, sudden arrhythmic death syndrome; n = 41.

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