14 Cases of Covid-19 mRNA Vaccine-Associated Myocarditis, Autopsied

Tables 1-7 detail published autopsy findings (references 1-9) from 14 fatal cases of post-covid mRNA vaccine-associated myocarditis. Ultimately, after extensive investigation, 12 of the 14 were deemed likely covid mRNA vaccine caused fatal myocarditis cases, while 2 were considered possibly caused by mRNA vaccination.

Table 1. Cases 1 and 2

Ref. (Country)	Case # Age/Sex	Clinical Presentation	Pathology
1a. (U.S.)	Case 1. Teenage Boy	BMI=21. History of attention deficit hyperactivity disorder but on no current meds. No complaints of fever, chest pain, palpitations, or dyspnea. Found dead in bed, without attempted resuscitation, 4-days after 2nd dose of Pfizer-BNT covid-19 vaccine. "Complained of a headache and gastric upset but felt better by postvaccine day 3. There was no history of prior medical problems (he took prescribed amphetamine/dextroamphetamine during the school year for attention deficit hyperactivity disorder but was not currently receiving it) or prior SARS-CoV-2 infection."	280 gram, normal heart. "There was global myocardial injury with areas of coagulative myocytolysis and contraction bands, with a perivascular pattern of inflammation consisting of predominantly neutrophils with histiocytes, scant lymphocytes, and occasional eosinophils. In some sections, the myocardial injury was predominantly subepicardial, and in other sections it was patchy and transmural. In the posterior wall, there was subepicardial/transmural fibrous scar, without fatty replacement. There were no acute or organizing thrombi. The overall pattern of injury was consistent with stress cardiomyopathy with contraction bands and a neutrophilic/ histiocytic infiltrate PCR tissue testing performed by the CDC on heart and lung found no molecular evidence of SARS-CoV-2 infection."
1b. (U.S.)	Case 2. Teenage Boy	Obese (BMI 30). No complaints of fever, chest pain, palpitations, or dyspnea, and no complaints, prior health issues, or prior SARS-CoV-2 infection. Found dead in bed, without attempted resuscitation, 3-days after 2nd dose of Pfizer-BNT covid-19 vaccine.	520 g heart with biventricular dilatation and marked pulmonary edema (combined lung weight= 1481 g). "There was global myocardial injury similar to that seen above (in case A), but with more widespread transmural ischemic changes and more interstitial inflammation, again with a predominant neutrophil component with histiocytes and scant lymphocytes. Several sections had transmural, confluent areas of hypereosinophilic myocytes; confluent areas of contraction bands apart from any inflammation; and florid neutrophilic inflammation with some histiocytes. In this case, a subepicardial distribution of injury was not seen. There were no acute or organizing thrombi. PCR tissue testing performed by the CDC on heart and lung found no molecular evidence of SARS-CoV-2 infection."

Table 2. Cases 3 and 4

Ref. (Country)	Case # Age/Sex	Clinical Presentation	Pathology
2. (U.S.)	Case 3. 42yo M	"presented with dyspnea and chest pain 2 weeks after mRNA- 1273 vaccination (second dose). He did not report a viral prodrome, and a PCR test was negative for SARS-CoV-2. He had tachycardia and a fever, and his electrocardiogram showed diffuse ST-segment elevation. A transthoracic echocardiogram showed global biventricular dysfunction (ejection fraction, 15%), normal ventricular dimensions, and left ventricular hypertrophy. Coronary angiography revealed no coronary artery disease. Cardiogenic shock developed in the patient, and he died 3 days after presentation"	"An autopsy revealed biventricular myocarditis. An inflammatory infiltrate admixed with macrophages, T-cells, eosinophils, and B cells was observed," consistent with "histologically confirmed, fulminant myocarditis."
3. (S. Korea)	Case 4. 22yo M	History of mild hypertension, but otherwise healthy. "5 days after the first dose of Pfizer-BNT162b2 mRNA vaccination, he complained to a colleague of chest pain at 1:00 AM, during a smoke break, and went to bed. At 8:00 AM, he was found unconscious hunched beside the bed. He was taken to an emergency department and was found to have ventricular fibrillation on electrocardiography. Cardiopulmonary resuscitation was performed for two hours, but he could not be resuscitated"	"The heart weighed 470 g and had multiple petechiae on its surface. The pericardium was smooth with no fibrin deposition or exudate. The coronary arteries were patent, and the heart valves were unremarkable. The myocardium was of normal thickness and there was no dilation of the atria or ventricles. The myocardium was homogeneously brown with no obvious necrosis or fibrosis. On microscopic examination, diffuse inflammatory infiltration, with neutrophil and histiocyte predominance, was observed within the myocardium. Notably, the inflammatory infiltrates were dominant in the atria, and around the sinoatrial (SA) and atrioventricular (AV) nodes whereas ventricular area displayed minimal or no inflammatory cells. Occasional myocyte necrosis or degeneration was found adjacent to the inflammatory infiltrates, without abscess formation or bacterial colonization. There was also scattered single-cell necrosis of myocytes without accompanying inflammation. Multiple scattered foci of contraction band necrosis (CBN) were identified throughout the myocardium, predominantly in the left ventricle. No other specific pathological changes were found in the lung, liver, kidney, spleen, pancreas, or brain on macroscopic or microscopic examination. Masson's trichrome staining highlighted dense eosinophilic intracellular strips of myocytes, consistent with CBN. CD68 and CD3 immunostaining showed a moderate number of histiocytes and sparse lymphocytes in the inflammatory infiltrates (**Fig. 2A and B**). Degenerated or ischemic myocytes exhibited positive C4d immunoreactivity. The cause of death was determined to be myocarditis, [g]iven that the myocarditis showed a temporal relationship to vaccine administration and there was no other explanation for the sudden cardiac death."

Table 3. Cases 5 and 6

Ref. (Country)	Case # Age/Sex	Clinical Presentation	Pathology
4. (New Zealand)	Case 5. 57yo F	"A previously well 57-year-old woman received the first Pfizer-BioNTech vaccine in July 2021. The following day she experienced increasing lethargy and had to leave work early because of worsening fatigue. She had one episode of breathlessness and complained of a stiff neck as well as upper limb pain. She had a sore throat but pointed to her sternum. During the remainder of the day, she became increasingly unwell. The following day she consulted her primary care physician with a sore throat, back pain, fatigue and an episode of hematuria, which had occurred the previous night. She had difficulty getting out of the car and experienced a fall at the family physician's surgery. She did not complain of palpitations. On day 2, a complete blood count (CBC) was normal but she was noted to have an increased C-reactive protein (CRP). She had a raised ferritin and alanine transaminase (ALT) but the aspartate aminotransferase (AST) was not undertaken, although the other liver enzymes were normal. There was no eosinophilia. On the third day, she was diagnosed with an *Escherichia coli* urinary tract infection, which was treated with trimethoprim. During that night, she was found deceased in bed. Apart from long-term omeprazole and the recently commenced trimethoprim, she was on no other treatment. There was no history of autoimmunity or allergic disease."	Gross autopsy findings other than a large thymoma, and mild splenomegaly, were unremarkable. "There was no pericardial effusion and there was no intracardiac thrombosis. The remainder of the autopsy examination was unremarkable. There were no abnormalities of other organs." "**Histological examination of the heart sections showed fulminant necrotizing eosinophilic myocarditis**. There were multifocal aggregates of lymphoid cells, histiocytes and abundant eosinophils with focal myocyte necrosis in the free walls of both ventricles, interventricular septum and around the conduction system (sinoatrial and atrio-ventricular nodes). No parasitic organisms or giant cells were identified. The eosinophilic infiltrate would make autoimmune myocarditis less likely. There was no evidence of eosinophils in other organs or eosinophilic vasculitis. Histological examination of the left pleural space mass showed a thymoma, WHO subtype AB. Antibodies to SARS-CoV-2 were negative on histological paraffin blocks... viral PCR tests including enterovirus, metapneumovirus, parainfluenza, rhinovirus, adenovirus, respiratory syncytial virus, SARS-CoV-2, parechovirus and influenza A and B were negative. A toxicology screen was negative."
5. (Japan)	Case 6. 27yo M	"A 27-year-old man was transferred to the emergency room in cardiopulmonary arrest. The patient had just received the first dose of the mRNA-1273 SARS-CoV-2 vaccine (Moderna) 8 days earlier and had no symptoms such as chest pain suspected of myocarditis or general fatigue suspected of low cardiac output after the vaccination until the emergency room visit. He was a professional athlete; his teammates called for an ambulance when he was found sitting unconscious during practice. Upon arrival at the emergency room, he presented asystole. Despite cardiopulmonary resuscitation, fatal ventricular arrhythmias repeated, and he eventually received venoarterial extracorporeal membrane oxygenation (VA-ECMO) and Impella CP, after 2 h of his visit. A chest radiograph obtained in an emergency room showed an enlarged heart and pulmonary congestion. The patient had undergone right elbow medial collateral ligament reconstruction 3 months before the recent hospitalization. An ECG abnormality and mild cardiac shadow enlargement in chest radiograph had been noted preoperatively; however, since he was asymptomatic, no further examination was performed. Additionally, he had no family history of cardiovascular disease or sudden death...After initiation of ventricular assist devices with VA-ECMO and Impella CP, steroid pulse therapy, and IVIG therapy, cardiac enzyme level decreased. He was able to wean off VA-ECMO on day 4; however, anisocoria appeared on the same day and head computed tomography revealed diffuse severe brain edema. On day 7, his LVEF improved to 45 %, and Impella CP was weaned. On day 9, the electroencephalogram became flat. The patient died on day 28 due to progressive multiorgan failure. The blood eosinophil count showed no significant increase until his death."	"An autopsy revealed asymmetric left ventricular hypertrophy, thickening of the right ventricular wall (550 g; LV wall, 11–16 mm; RV wall, 5–7 mm), myxomatous degeneration of the posterior leaflet of the mitral valve, and hypertrophy of the posteromedial papillary muscle. Microscopic findings revealed that cardiac myocytolysis and widespread fibrosis were observed and significant mixed inflammatory infiltration (T cells, macrophages, and eosinophils) was observed in the left ventricular free wall and the anterior potion of the ventricular septum, which led to the diagnosis of myocarditis. There was no evidence of CD138+ CD79a+ CD20− plasmocytes. Although asymmetric left ventricular hypertrophy was observed, cardiac muscle cell disorganization that is characteristic of hypertrophic cardiomyopathy was not observed. In contrast to the left ventricular free wall, the posterior potion of the ventricular septum and right ventricular free wall had almost no inflammatory cell infiltration or fibrosis, and almost normal myocardium was maintained. Additionally, the posterior papillary muscles showed a similar inflammatory cell infiltrate at the left ventricular myocardium, with extensive and severe fibrosis.... **the autopsy showed obvious severe myocardial inflammation findings, leading to the diagnosis of myocarditis**... no findings characteristic of hypertrophic cardiomyopathy were seen on autopsy. Therefore, we considered that his left ventricular hypertrophy was a consequence of [the] athlete's heart and mitral regurgitation. **Microscopic findings showed infiltration of T cells, macrophages, and eosinophils. Such myocarditis with lymphocytic and eosinophilic infiltration has been reported after mRNA COVID-19 vaccination**."

Table 4. Case 7

Ref. (Country)	Case # Age/Sex	Clinical Presentation	Pathology
6. (Japan)	Case 7. 90s M	A Japanese male in his 90 s consulted a doctor because he experienced several days of general fatigue and dyspnea. His legs were edematous, and chest X-ray showed right pleural effusion. Elevated *N*-terminal pro-brain natriuretic peptide (NT-pro BNP; 3,706 pg/mL) and C-reactive protein (47.9 mg/L) were detected. The electrocardiogram results showed no abnormal change. He was diagnosed with heart failure but refused hospital admission. The patient was prescribed a 3-day course of diuretic medication, which relieved his symptoms and decreased the NT-pro BNP level. However, he was found lifeless in his kitchen on the morning of the fourth day after consulting the doctor. He had received a third dose of BNT162b2 approximately 2 weeks before death. No previous illness was reported. He did not have a history of smoking or habitual alcohol consumption. A police investigation at the man's home revealed no suspicious activity.	A medical examiner found no external abnormalities, including in the left deltoid injection site; therefore, an autopsy was performed 35 h postmortem. The deceased was 156 cm in height and weighed 52 kg. The pericardial sac was filled with dark red clots. The ascending aorta had a 2.5 cm intimal tear at 4 cm above the aortic annulus . The aortic media was dissected, and the adventitia was perforated within the pericardial cavity. The heart weighed 458 g and had a white villous surface. Coronary arteries showed mild atherosclerosis. Disrupted coronary artery plaques, coronary aneurysms, and pulmonary emboli were not detected...Microscopic examination revealed fibrously thick epicardium with inflammatory cell infiltration predominantly composed of macrophages and lymphocytes... The pericardial membrane was thick with fibrin deposition and hypertrophic fibroblasts. Macrophages and lymphocytes were also detected in the membrane... Laboratory examinations of the femoral blood were negative for antibodies to parvovirus-B19, cytomegalovirus, coxsackie virus-A4, ECHO virus-11 and □ 14, adenovirus, and influenza A (H1N1 and H3N2) and B (B-1 and B-2) viruses. A neutralization test for ECHO virus- 9 was positive at a titer of 32. The serum was positive for anti-SARS-CoV- 2 spike protein IgG antibody (583 AU/mL). Headspace gas chromatography revealed no ethanol in the venous blood, urine, or cerebrospinal fluid... The present patient developed pericarditis after a third dose of COVID-19 vaccine, and mildly elevated IgG antibodies were detected in the postmortem autopsy sample... We presumed that death in the present case was caused by pericarditis-induced fragility of the aortic wall followed by cardiac tamponade.

Table 5. Case 8

Ref. (Country)	Case # Age/Sex	Clinical Presentation	Pathology
7. (Germany)	Case 8. 76yo M	This report presents the case of a 76-year-old male with a history of Parkinson's disease (PD) who passed away three weeks after his third COVID-19 vaccination. On the day of his first vaccination in May 2021 (ChAdOx1 nCov-19 vector vaccine), he experienced pronounced cardiovascular side effects, for which he repeatedly had to consult his doctor. After the second vaccination in July 2021 (BNT162b2 mRNA vaccine/Comirnaty), the family noted obvious behavioral and psychological changes (e.g., he did not want to be touched anymore and experienced increased anxiety, lethargy, and social withdrawal even from close family members). Furthermore, there was a striking worsening of his PD symptoms, which led to severe motor impairment and a recurrent need for wheelchair support. He never fully recovered from these side effects after the first two vaccinations but still got another vaccination in December 2021. Two weeks after the third vaccination (second vaccination with BNT162b2), he suddenly collapsed while taking his dinner. Remarkably, he did not show coughing or any signs of food aspiration but just fell down silently. He recovered from this more or less, but one week later, he again suddenly collapsed silently while taking his meal. The emergency unit was called, and after successful, but prolonged resuscitation attempts (over one hour), he was transferred to the hospital and directly put into an artificial coma but died shortly thereafter. The clinical diagnosis was death due to aspiration pneumonia. According to his family, there was no history of a clinical or laboratory diagnosis of COVID-19 in the past.	The autopsy was requested and consented to by the family of the patient because of the ambiguity of symptoms before his death. The autopsy was performed according to standard procedures including macroscopic and microscopic investigation. Gross brain tissue was prepared for histological examination including the brain (frontal cortex, Substantia nigra, and Nucleus ruber) as well as the heart (left and right ventricular cardiac tissue)...The 76-year-old deceased male patient had PD, which corresponded to typical post-mortem findings. The main cause of death was recurrent aspiration pneumonia. In addition, necrotizing encephalitis and vasculitis were considered to be major contributors to death. Furthermore, there was mild lympho-histiocytic myocarditis with fine- spotted myocardial fibrosis as well as systemic arteriosclerosis, which will have also contributed to the deterioration of the physical condition of the senior. The final diagnosis was abscedating bilateral bronchopneumonia (J18.9), Parkinson's disease (G20.9), necrotic encephalitis (G04.9), and myocarditis (I40.9). Immunohistochemistry for SARS-CoV-2 antigens (spike protein and nucleocapsid) revealed that the lesions with necrotizing encephalitis as well as the acute inflammatory changes in the small blood vessels (brain and heart) were associated with abundant deposits of the spike protein SARS-CoV-2 subunit 1. Since the nucleocapsid protein of SARS-CoV-2 was consistently absent, it must be assumed that the presence of spike protein in affected tissues was not due to an infection with SARS-CoV-2 but rather to the transfection of the tissues by the gene-based COVID-19-vaccines.

Table 6. Cases 9,

Ref. (Country)	Case # Age/Sex	Clinical Presentation	Pathology
8. (Germany)	Case 9. 55yo M	A 55-year-old male patient was vaccinated with the ChAdOx1 nCov-19 vector vaccine (AstraZeneca) in May 2021, and with BNT162b2 mRNA vaccine (Pfizer-BioNTech COMIRNATY) in July 2021, according to a copy of the vaccination book. The patient died in November 2021. An autopsy diagnosis was general atherosclerosis. The cause of death was acute myocardial infarction and lymphocytic myocarditis. The lumen was occluded with a platelet thrombus on an atherosclerotic bed in the right coronary artery. This condition was associated with a significant lymphocytic vasculitis of the vasa vasorum. The family of the patient sought and approved the autopsy since the patient's symptoms before passing away were unclear. Standard procedures were followed throughout the autopsy, including a macroscopic and microscopic examination. All parenchymatous tissues were examined histopathologically	The final diagnosis was acute myocardial infarction (I21.9), arteritis of the right coronary artery and acute myocarditis (I40.9), and generalized atherosclerosis (I70.9). The identification of SARS-CoV-2 spike protein on the endothelial surface correlates the findings with the host immune response to the received vaccine. A recent infection with SARSCoV- 2 could be excluded as the source of the spike protein based on the absence of concomitantly expressed nucleocapsid protein... The autopsy study of a 55-year-old deceased person revealed evidence of thrombus formation in the large RCA, small arterial branches, and arterial capillaries that could not be attributed to atherosclerosis plaque rupture. Moreover, there was inflammatory cell infiltration including CD4 and CD8 T cells and histiocytes which is typical of an autoimmune reaction. The histopathological examinations revealed the extensive presence of scattered fibrotic areas, indicating that cardiac cell necrosis had occurred at these sites during a time period well preceding death. The abundance of small fibrotic scars, in combination with fresh inflammatory lesions,,as revealed in this study, has never been described in any cardiac affliction hithertofore. A crucial finding, in this study, was the detection of spike protein by immunohistochemistry in the vascular and cardiac tissue in the absence of the nucleocapsid protein.

Table 7. Cases 10,11,12,13,14

Ref. (Country)	Case # Age/Sex	Clinical Presentation	Pathology
9. (Germany)	Cases 10-14. 46yo M 50yo F 62yo F 55yo M 75yo F	See table attached, below, and the following: "Based on the autopsy findings and all available data, no other cause of death except (epi-) myocarditis was identified in any of the cases presented here. Hence, myocarditis has to be considered the likely cause of death... Myocarditis-related acute cardiac arrest due to either asystole or ventricular fibrillation is a well-established pathomechanism in other causes of acute myocarditis as well."	

Gender	Age	BMI	Vaccine type	Dose	Time from vaccination to death (days)	Time from death to autopsy (days)	Comorbidity	Grading – myocarditis (0 – 3)	Grading – epicarditis (0 – 3)	PCR analysis	Assessment of causal relationship
male	46	31.8	Cormirnaty (BioNTech)	First	0	7	AH	2	0	Negative	Likely
female	50	20	Spikevax (Moderna)	First	1	3	-	1	2	Negativ	Likely
female	62	22.5	Cormirnaty (BioNTech)	First	7	3	COPD	1	1	Negative	Possible
male	55	30.1	Cormirnaty (BioNTech)	Second	4	3	-	2	3	Negative	Likely
female	75	27,9	Cormirnaty (BioNTech)	First	1	9	AH, DM, Hashimoto´s thyroiditis	2	2	HHV6	Possible

AH=arterial hypertension; COPD= chronic obstructive pulmonary disease; DM=diabetes mellitus

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