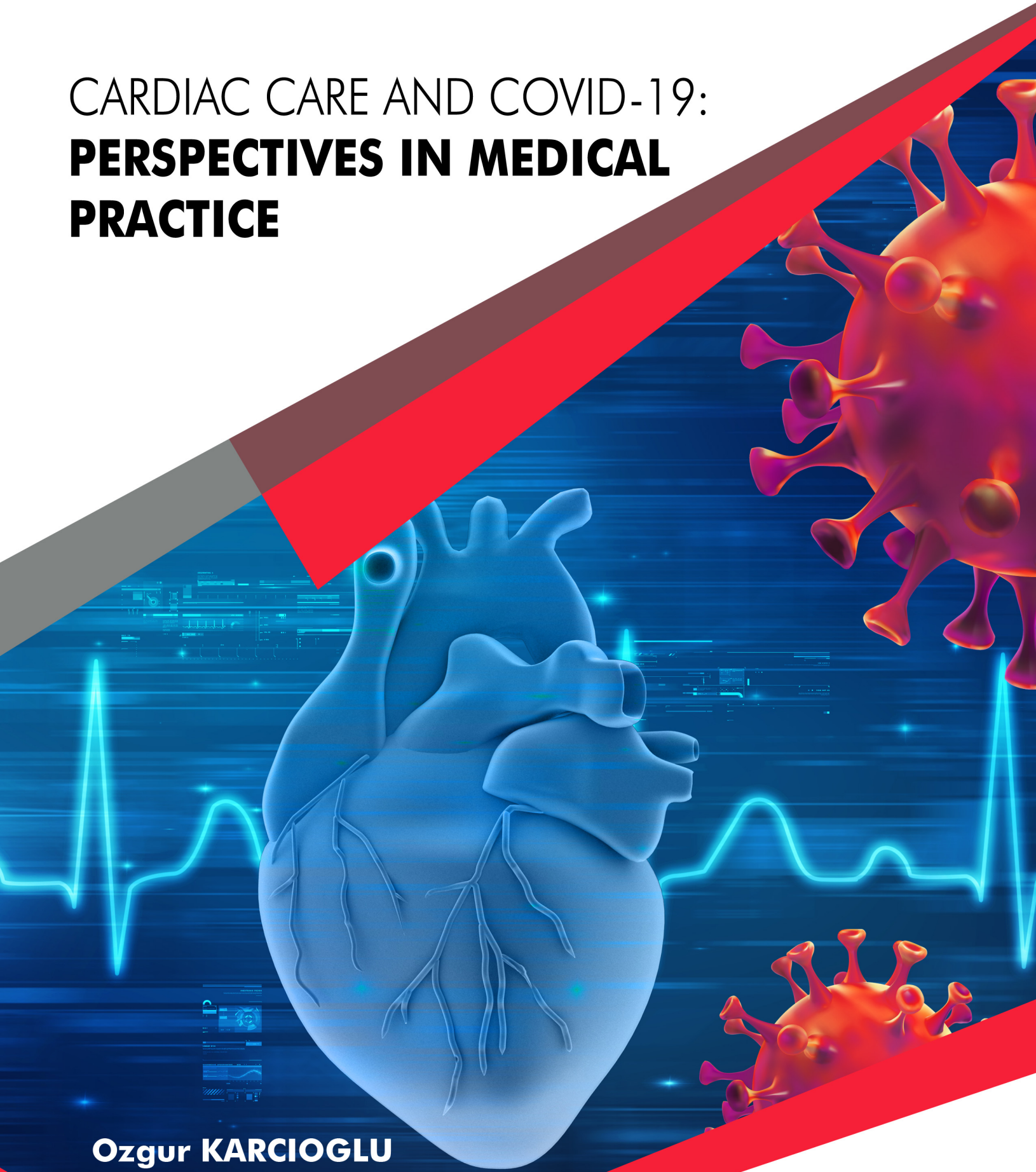
CARDIAC CARE AND COVID-19: **PERSPECTIVES IN MEDICAL PRACTICE**



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Cardiac Care and COVID-19: Perspectives in Medical Practice

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PREFACE

The optimal management of patients with cardiac disease warrants a multifaceted approach undertaken in harmony. The recent decades have witnessed major advances in methods for monitoring and interventions aiming to improve outcomes in this outstanding cause of death worldwide. Other than technological improvements, the medical community is aware that this task can only be achieved *via* a mutual collaboration of doctors in the pre-hospital phase, hospital emergency departments, intensive care units, social studies, public health professionals, and bystanders.

For nearly a year, our lives have changed like never before. The current WHO clinical guide documents cite that *'there is no current evidence to recommend any specific anti-COVID-19 treatment for patients with confirmed COVID-19'.*

In order to overcome the pandemic with minimized global losses, the scientific community, healthcare facilities, professional organizations, chambers, and state institutions should work in coordination and unison. Most importantly, only a coordinated approach with all targeted masses reached *via* awareness programmes and campaigns can create a real difference in this pandemic era.

This project, of the book **'Cardiac Care and COVID-19: Perspectives in Medical Practice'** is intended to encompass the advancements regarding diagnoses and treatment modalities for cardiac diseases in general and emergency cardiac conditions to be more specific, with respect to pandemic conditions. Apart from up-to-date descriptions of the problem and delineation of management principles, case examples were also used to highlight complex issues for a concrete understanding of the medical practitioner.

The ultimate objective is to provide a reference source with up-to-date information on the management of cardiac emergencies and resuscitation in the COVID-19 era. We aim to conduct a brief overview of epidemiological features of cardiac emergencies and their sociodemographic factors, measures to be taken for prevention, together with diagnostic and therapeutic procedures to pursue in the pandemic era.

CONSENT FOR PUBLICATION

Not applicable.

CONFLICT OF INTEREST

The author declares no conflict of interest, financial or otherwise.

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CHAPTER 1

Introduction: Cardiac Disease in the Pandemic Era: Teaching an Old Dog New Tricks?

Abstract: Nowadays, cardiac diseases, both developed *de novo* and acute exacerbations of chronic conditions, remain the most prominent death cause for the middle-aged and elderly, mostly in the developed, industrialized countries.

Since the end of 2019, COVID-19 pandemics have changed our lifestyles fundamentally, and maybe we will never find a way to return to the world of 2019. This catastrophic change had its impact on almost every aspect of our lives, including how we will manage cardiac arrest patients, how to perform perform cardiopulmonary resuscitation (CPR), ACLS, *etc*. A net effect is that protecting ourselves will take priority (more than before) in all procedures we pursue. Thus we can conclude that new generations should incorporate self-protecting behavior and techniques to benefit the patients in the most fruitful ways.

High-quality CPR cardiopulmonary resuscitation is among the most prominent issues to save humanity from the high burden of cardiac events. Relatively novel techniques such as mechanized devices for CPR, extracorporeal membrane oxygenation (ECMO), and therapeutic temperature management promise the highest possible solution to improve survival rates, in conjunction with urgent coronary angiography with revascularization.

Pandemics can be overcome not by the heroic behaviors of a few people but by the solidarity of society. The medical community should find the best solutions to help those in need with cardiac diseases even in pandemic conditions since this pandemic will not go away like magic. The aim of this book is to support patients and their next of kin, as well as health care workers, those who have dedicated themselves to healthy well-being with their relentless endeavor.

Keywords: Cardiac arrest, Cardiac arrhythmias, Cardiopulmonary resuscitation, COVID-19 pandemics, Defibrillation, Treatment.

INTRODUCTION

As an ageless phenomenon of life, medicine has been viewed as an art of recognizing and relieving human sufferings, treating diseases and wounds for ages. The last centuries have witnessed warfare, socioeconomic crises, and many

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other threats which had a great impact on the medical database used and practicing ways in cardiac resuscitation.

Cardiovascular disease (CVD) is the leading cause of death for adults. Expedient diagnosis and prompt institution of treatment can save lives, especially during the deadliest cardiac emergencies, including sudden cardiac death, acute pulmonary edema, lethal arrhythmias, and acute pericarditis.

In order to establish an easier recognition and a more holistic, systematic approach to cardiac emergencies, revolutionary steps forward and developments in cardiac markers, monitoring, defibrillation, therapeutic hypothermia or temperature management (TTM), capnographic recordings, and the like were developed after 60's and 70's. Now we can postulate that these innovations must have mitigated the hazards of cardiac diseases globally. Maybe this is why cancer and infectious diseases are championing on the morbidity and death list in most parts of the world in the last decades.

The most prominent death scenario comprises **out-of-hospital cardiac arrest (OHCA)** in the middle-aged population globally. Lethal dysrhythmias can be divided into four types: ventricular fibrillation-VF, pulseless ventricular tachycardia (PVT), asystole, and pulseless electrical activity, which are responsible for impaired cardiac functioning and even sudden cardiac death.

VF is one of the most deadly cardiac arrhythmias and certainly the most common one. It can be described as the erratic, disorganized firing of impulses from the ventricles, producing no palpable pulses in the periphery. Literature data have shown that the earlier defibrillation and bystander cardiopulmonary resuscitation (CPR) are commenced, the lower is the patient mortality. Since considerable differences can affect people's lives in this context, the role of medical command bears utmost importance to direct these patients to facilities with discrete capabilities

VF is among the most common and fatal cardiac arrhythmias. Literature findings demonstrated that patient mortality could be much lower when defibrillation is performed early and when laypeople initiate cardiopulmonary resuscitation (CPR). As important differences in this process can have an impact on our lives, the role of the EMS medical command is of great significance in directing these patients to facilities with adequate levels of resources (Stoecklein, 2018).

EMERGENT CORONARY REVASCULARIZATION

Emergent Coronary Revascularization is an outstanding life-saving intervention in patients with acute coronary syndromes, mostly AMI.

ALTERNATIVE APPROACHES TO THE MANAGEMENT OF VF

Most patients with refractory VF are resistant to conventional treatment strategies. Nonetheless, some new techniques produced promising outcomes (Bell, 2018). Of note, double sequential defibrillation can represent an option for the conventional approach for the treatment of PVT or refractory VF (Simon, 2018).

Recently, procedures like extracorporeal life support and bedside ultrasound have been launched. These may represent a logical and practical way to manage patients with refractory arrest rhythms, both in-hospital and out-of-hospital milieu. Likewise, drones have been introduced as one of the contemporary advances, to bring automated external defibrillators (AED) to the patient with OHCA. Also, digital and mobile technology have launched new apps to optimize interventions carried out by laypeople, to increase survival in this group of patients with poor expectancy for return of spontaneous circulation (ROSC) (Latimer *et al*., 2018).

A major challenge for contemporary medicine as a whole is operating a system focused on the optimized outcomes of patients with OHCA. This challenge can only be overcome with a flexible and resourceful approach that comprises various teams, from call receivers, monitors, to emergency medical service (EMS) staff and the healthcare workers in the receiving center (McCoy *et al*., 2018). The application of these techniques for OHCA in a healthcare system will yield the most favorable outcomes for survivors without sequelae among well-known or ‘classical’ approaches.

A majority of the current management guidelines to improve survival following arrest situations are the result of efforts to improve CPR quality, increasing the chances of ROSC and the like. The emphasis on the delivery of proven techniques and the reliable implementation of these strategies through the measurement and audit of quality improvement strategies will create a foundation so that innovations in resuscitation could be designed and planned (Reed-Schrader, 2018). After all those above mentioned important developments, post-cardiac arrest interventions have aroused more curiosity. Therapeutic hypothermia or TTM, which has become the state-of-art in most hospitals, will produce the best probability of relief after the ROSC without remarkable sequelae (Walker, 2018).

THE PANDEMICS

This catastrophic change had its impact on almost every aspect of our lives, including how we should manage cardiac arrest patients, how to perform CPR, ACLS, *etc*. Bugger *et al*. investigated the net effect of the pandemic restrictions on certain major cardiovascular emergencies (myocardial infarction, pulmonary

CHAPTER 2

Cardiovascular Disease and COVID-19

Abstract: Cardiovascular disease (CVD) has long been the leading cause of global morbidity and mortality. However, with the COVID-19 pandemic, which has been the focus of attention all over the world since the end of 2019, this issue has gained different importance. The presence of CVD leads to more severe COVID-19 and an increased probability of mortality. In addition, both CVD and COVID-19 pave the way to myocardial injury, which also boosts the morbidity and death toll. Another point is the possible deprivation of usual healthcare received by cardiac patients (CVD and others) because of the shifted emphasis of the hospital and prehospital medical services on COVID-19. As the public can foresee that the pandemic will not disappear rapidly soon, healthcare organization faces a challenge to be redesigned radically. The objective of this chapter is to analyze CVD, myocardial injury, and other cardiac diseases resulting from COVID-19 itself, together with the impact of the pandemics on the usual healthcare of cardiac patients.

Keywords: Acute myocardial infarction, Cardiovascular disease, Coronavirus, COVID-19, Diagnosis, Treatment.

INTRODUCTION

"Acute coronary syndromes" (ACS) is a general term for conditions that occur as a result of a sudden blockage of the blood flow to the heart. These syndromes range from potentially reversible unstable angina (UA) phase to irreversible cell death from myocardial infarction (MI), including non-ST elevation myocardial infarction (NSTEMI) or ST-elevation myocardial infarction (STEMI).

DISTINGUISHING FEATURES OF ACUTE CORONARY SYNDROMES

UA, NSTEMI, and STEMI have a common pathophysiological origin of atherosclerotic coronary artery disease (CAD), which is characterized by plaque formation on the walls of arteries that supply blood flow to the heart. Erosion or rupture of the plaque leads to a blood clot (thrombosis) that blocks blood flow to the heart, depriving the heart of oxygen and consequently leading to myocardial necrosis (tissue death in the heart muscle).

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Cardiovascular disease (CVD) has been associated with viral infections or outbreaks for decades. It has been reported that approximately half of COVID-19 patients have CVD, and this rate increases to 70% in intensive care units (ICU) (Zhou *et al*., 2020, Wang *et al*., 2020). It is known that 50% of the cases with Middle-East Respiratory Syndrome (MERS) infection in 2012 had DM and HT, and 30% had CVD (Badawi *et al*., 2016). Meanwhile, certain CV risk factors have been thought to affect the clinical course of COVID-19 (Table **1**).

Table 1. CV risk factors that are thought to affect the clinical course of COVID-19.

• Male gender
• Advanced age
• DM
• Hypertension
• Obesity
• A history of cardiovascular or cerebrovascular disease

FINDINGS FROM DIFFERENT DATABASE STUDIES

In the study which analyzed 5700 patients in New York, frequency of hypertension (HT) was reported as 56.6%, obesity 41.7%, DM 33.8%, CAD 11.1%, and congestive heart failure (CHF) 6.9% (Richardson *et al*., 2020). In the study that included more than 72,000 patients in China, 12.8% HT, 5.3% DM, 4.2% CVD were found. The frequency of comorbidity is similar in industrialized countries, except that the HT, metabolic disorders, and obesity rates are significantly higher than in the far east, but the prevalence varies considerably.

When analyzing 22,254 patients who were screened with PCR tests between 5 March and 9 April in NYC, at least one comorbid disease was reported in about half of those with positive tests (46%) (Kalyanaraman Marcello, 2020). In the sample, 33% diabetes, 37% HT, 24% CVD, 11% chronic renal failure (CRF) were noted. Among the hospitalized patients, 28% died. Male gender, age, DM, history of heart disease, presence of CRF are risk factors for both test positivity and death.

In a case series from Detroit, Suleyman *et al*. searched for independent risk factors for admission to intensive care units: being over the age of 60, male sex, HT, DM, CRF, severe obesity (BMI>= 40), and cancer were reported to be the ones (Suleyman *et al*., 2020). Smoking was also higher in hospitalized patients. Admissions due to dyspnea, tachypnea, or hypoxia also increases the risk of hospitalization. Fever increases the likelihood of hospitalization but does not

predict poor outcomes in patients with COVID-19. Inflammatory markers were also found higher in those hospitalized in ED when compared to those discharged from ED.

Respiratory failure developed in 74% of those hospitalized in ICU, and MV was required in 81% of these. 25% of those hospitalized had to be transferred to ED. The majority of those who received MV under 40 years of age (62.5%) had severe obesity, while only 26% of those who did not need MV had the condition. Mortality in ICU was 40%, and 7% in all those hospitalized. 45.6% of those who require MV died of severe complications.

ARE ETHNIC DIFFERENCES IMPORTANT FOR COVID-19?

Marcello *et al*. conducted a study that analyzed more than 22,000 patients in NYC and reported that 26% of COVID-19s were black, and 34% were Hispanic (Marcello *et al*., 2020). They reported that comorbidities are more common in ethnic groups than others, but being black or Hispanic after adjustments is not directly related to positive PCR testing or death.

In another cohort reported from Louisiana, it is stated that blacks, which make up only 31% of the population, constitute 77% of the COVID-19 patients who are admitted into the hospital, but being black is not an independent risk factor when confounding factors are excluded (Price-Haywood *et al*., 2020). 70.6% of the dead are black.

Mechanisms that trigger myocardial damage in COVID-19:
Severe systemic inflammatory stimulus - cytokine storm
Ischemia due to increased consumption or demand
Plaque rupture
Vascular inflammation

MYOCARDIAL INJURY SEVERITY AND MORTALITY

Autopsy showed interstitial mononuclear inflammatory cell infiltration in the myocardium (Xu, 2020). Also, markers showing myocardial damage increase with COVID-19 (Xu 2020, Guo 2020, Shi, 2020). Shi *et al*. reported that myocardial damage was close to 20% in patients who died (Shi, 2020). Moreover, cardiac damage is the risk factor that affects mortality most strongly and independently (hazard ratio: 4.26). Guo *et al*. reported that high troponin levels accompanied significantly increased mortality (Guo, 2020).

In the series of Guo *et al*., which included 187 patients with COVID-19, they stated that there was a better clinical course in patients with CVD and no acute

CHAPTER 3

Myocardial Damage, Myocarditis, and COVID-19

Abstract: For centuries, complications of cardiovascular disease (CVD) have been documented as the prominent cause of mortality and morbidity worldwide. CVD and pandemic disease precipitate different kinds of damage in the myocardium, which also contribute to the death rates in the vulnerable population.

Almost all viral infections, including COVID-19 and influenza species, have the potential to inflict damage to the myocardial tissue, which also contributes to the severity of the disease itself. For instance, COVID-19 can trigger multiple organ failures with remarkable end results on cardiovascular functions. Its damage to the CVD like myocarditis, myocardial injury, *de novo* heart failure, acute coronary syndromes including STEMI, and various kinds of fatal and non-fatal dysrhythmias can be mentioned. Although most cases with myocarditis are asymptomatic or exhibit a mild course, it can precipitate acute heart failure and fatal respiratory failure.

Clinicians should be alert in patients with signs and symptoms compatible with myocarditis, pericarditis and/or endocarditis in the pandemic period and routine care because expedient diagnosis and management can prevent adverse outcomes in selected cases.

Keywords: Cardiovascular disease, COVID-19, Diagnosis, Endocarditis, Management, Myocardial damage, Myocarditis, Pericarditis.

MYOCARDIAL INJURY INFLICTED BY COVID-19

Although COVID-19 has been recognized to cause pneumonia, respiratory failure, and ARDS as the mainstays of its pathophysiology, myocarditis can also be detected in a substantial percentage of cases with COVID-19.

Acute cardiac injury (ACI) is among the most important complications of COVID-19. Zuin *et al*. published a meta-analytic study to enlighten the interactions between ACI and risks of death in these vulnerable populations (Zuin *et al*., 2020). They included 8 articles, which recruited 1686 patients (mean age 60 years) in the final analysis. Rates of ACI was recorded higher among fatalities than it was in survivors (62% *vs*. 7%, $P<0.0001$). The analysis verified an increased mortality risk in those complicated with ACI within the course of the pandemic disease ($P<0.0001$).

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There are some other studies that demonstrated that ACI is detected frequently in those with COVID-19 (Shi S, 2020). Greater proportions of patients with ACI needed noninvasive mechanical ventilation (MV) (46% *vs*. 4%; P < .001) or invasive MV (22% *vs*. 4%; P<.001) than those without ACI.

COVID-19 patients with severe myocardial damage have been shown to be exposed to a significantly higher risk of severe disease course, need for intensive care, and mortality (Li X, 2020). In this meta-analytic study, high levels of CK, CK-MB, LDH, and IL-6, and *de novo* dysrhythmias are linked with poor outcomes and the need for ICU admission, and the death rates are higher in patients with supranormal LDH and IL-6 readings. Of note, newly occurring arrhythmias have also been associated with an increased risk of grave outcome and/or need for intensive care (P<0.001).

Mechanisms of how SARS-CoV-2 Induces Inflammation and Disease

The suggested mechanisms of ACI are the viral entry of the virus to the myocardial cells and injury to the myocardium, systemic inflammation, hypoxia, cytokine storm, the immune response mediated by interferons, and plaque destabilization. The SARS-CoV-2 is an immunogenic virus that activates the innate immune system, particularly macrophages. In other disease conditions, activated macrophages produce interferons that trigger the destruction of the virus. However, in the case of SARS-CoV-2, macrophageal activation results in the release of interleukin 1 beta, which induces systemic inflammation. The pathological findings in cardiac tissue involved in COVID-19 infection exhibited only minor changes to interstitial inflammatory infiltration to the hyperactivation of inflammation, cytokine storm, myocyte necrosis, myocarditis, MI, and HF (Fig. **1**). Microthrombi and vascular inflammation have been noted in the vessels. The infection also induces systemic complications like sepsis and DIC.

COVID-19 can induce ACI other than ischemia, including broken heart syndrome or stress cardiomyopathy, acute and fulminant myocarditis (Chen C, 2020, Chen L, 2020).

Mechanisms of myocardial injury can involve discrete pathways with resultant pathophysiological effects (Table **1**).

Table 1. Mechanisms of myocardial injury.

	Hypothesized Mechanism of Injury
Myocarditis	Systemic inflammatory response; direct myocardial cell injury *via* viral entry using ACE-2 receptor; T-cell–mediated immune response

(Table 1) cont.....

	Hypothesized Mechanism of Injury
Myocardial infarction	Plaque rupture (Type I MI); myocardial oxygen supply/demand mismatch (Type II MI) from increased cardiometabolic demand
Microangiopathy/cytokine storm	Cytokine-induced activation of microvasculature predisposing to vasomotor abnormalities; augmented thrombosis and other aspects of dysfunction
Arrhythmia	Hypoxia-mediated; coronary perfusion impairment; direct tissue injury; scar-mediated injury, inflammatory response; medication-induced electrolyte abnormality

Recommended approach to COVID-19 patients with cardiogenic or mixed circulatory shock involves both the clinical status of the patient and the capabilities of the institution.

MYOCARDITIS

is the general term for the inflammatory state or disease of the myocardium. It can be precipitated by a variety of infectious and noninfectious causes (*i.e.*, toxic effects or autoimmunity). Pharmacological agents can also cause myocarditis. Time from inciting cause to the manifestation of symptoms can be as short as hours in some cases, while some are delayed to months. Young people are reported to have myocarditis more frequently than the elderly.

Cardiotropic Viruses as the Causes

Many different viruses have been accused in human myocarditis so far. The most common viral genetic material reported in myocarditis are human herpes virus 6 and parvovirus B19. Enteroviridae cause myocarditis in some regions. H1N1 influenza A infection has been recorded to precipitate fulminant myocarditis. Other injurious agents of myocardium have been reported as electric shock, hyperpyrexia, radiation (Table **2**).

Different researchers reported viruses as the etiological agents at rates between 37% and 77% in patients with myocarditis (Pankuweit 2003, Kuhl 2005). In patients with ejection fraction below 45%, viral etiology was found to be 42% (Marburg registry). Cardiac MRI revealed myocardial involvement following COVID-19 infection in 78 patients (78%) and myocardial inflammation in 60 (60%) (Puntmann, 2020).

Fulminant Myocarditis (FM)

FM is characterized by sudden and severe diffuse myocardial inflammation often

CHAPTER 4

Coagulopathies, Prothrombotic State, Thromboembolism, Bleeding, and COVID-19

Abstract: COVID-19 is known to trigger a prothrombotic state, causing thromboses and thromboembolic events (TTEE) in patients with COVID-19. Both bleeding and thrombosis can result in significant morbidity in COVID-19. The entity paves the way to arterial TTEE (*i.e.*, stroke and/or extremity ischemia) as well as small vessel thrombosis, which are commonly recorded at autopsy in the pulmonary vasculature. Elevated D-dimer is associated with a higher risk for TTEE, hemorrhage, critical illness, and mortality. Likewise, levels of fibrinogen, ferritin, procalcitonin are also higher in patients with thrombosis. There is also a propensity to develop pulmonary thromboembolism (PTE) in cases with COVID-19. Treatment with anticoagulant prophylaxis (*i.e.*, heparin and/or aspirin) is recommended in many researches, but robust evidence is still warranted to draw firm conclusions on the benefit-to-harm ratio of the agents in most patients.

Keywords: Anticoagulant prophylaxis, Cardiovascular disease, Coagulopathy, COVID-19, D-dimer, Thromboembolic events, Thrombosis.

INTRODUCTION

Bleeding diathesis, coagulopathy, and TTEE are ominous factors that predict a severe course and send the patients to intensive care units. Many studies noted that high levels of fibrin degradation products such as D-dimer are directly linked with death rates, which is suggestive of a coagulopathy associated with COVID-19 (Zhou, 2020, Samkari,2020). However, there is still scarce evidence of coagulopathy and thrombotic risk in COVID-19 patients. Paradoxically, both thromboses and bleeding diathesis are reported from samples of patients in the pandemic era. For example, intracranial hemorrhage as well as thrombotic ischemic lesions were reported in different series of COVID-19 (Helms, 2020).

Although rare, DIC, low platelet count, and hypofibrinogenemia were associated with important bleeding in those with COVID-19 (Samkari *et al*., 2020). However, Tang *et al*. pointed out that 71% of those who died of COVID-19 had the criteria of disseminated intravascular coagulation (DIC), while only 0.6% of survivors did (Tang, 2020).

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The high number of arterial and/or venous TTEE diagnosed within 24 h of admission and the high rate of positive TTEE imaging in cases with COVID-19 suggest that there is a need to improve specific algorithms to recognize TTEE and investigate the efficacy and safety of thromboprophylaxis in ambulatory patients (Lodigiani, 2020).

SARS-COV-2 involvement of the epithelial tissue of the lung precipitates the process of inflammation, which activates neutrophils and starts the construction of a neutrophil extracellular trap (NET) (Fig. **1**).

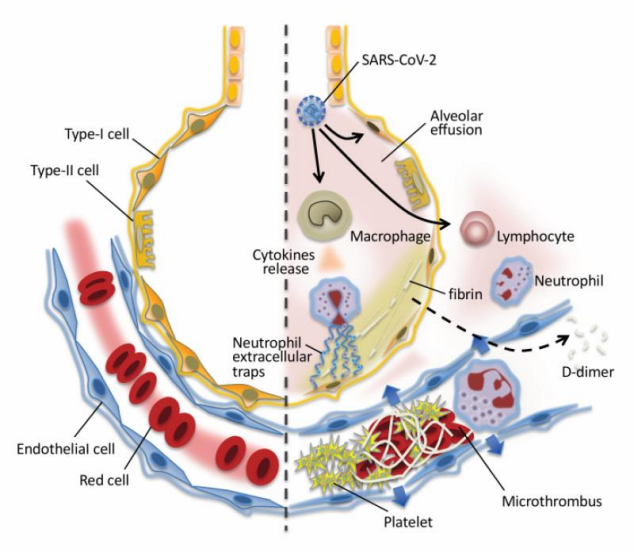

Fig. (1). Inflammatory cascade in the alveoli triggered by cytokines initiates microthrombi in the lung vessels that turn into thromboembolism in some cases.

An algorithm is developed as a workflow in patients suspected to have VTE, in the setting of COVID-19 (Aryal *et al*. 2020).

Microvascular thromboses can also ensue during COVID-19. Some patients who died of COVID-19 underwent autopsies which revealed pulmonary microvascular thrombosis (Menter, 2020, Ackermann, 2020).

COVID-19 infection precipitates a marked increase in fibrinogen and D-dimer. This phenomenon is postulated to represent systemic hypercoagulability which gives rise to venous TTEE. This is the rationale behind the fact that the extent of D-dimer increases correlates with death rates in patients with confirmed COVID-19.

LABORATORY VARIABLES

Thrombocytopenia and high d-dimer are the two parameters which suggest high risk of need for intensive care and mortality (Lippi, 2020).

In-hospital mortality has been linked with advanced age, high D-dimer (> 1000 μg/L), and high SOFA score on admission (Zhou, 2020).

High levels of D-dimer obtained on admission is suggestive for hemorrhage, VTE/TTEE, and grave outcome.

Mouhat *et al*. reported that D-dimer levels above 2590 ng/mL can predict PTE in COVID-19 patients with clinical deterioration (Mouhat, 2020).

COVID-19 cases have mostly been shown to exhibit low platelet count probably resulting from high turnover of platelets (Wool, 2020) (Table **1**). Nonetheless, disseminated intravascular coagulopathy (DIC) and hemorrhage are infrequent in cases with COVID-19.

ESR, CRP, fibrinogen, ferritin, and procalcitonin were higher in patients with thrombotic complications than in those without.

Elevated NLR, and PLR have been postulated as prognostic and risk stratifying factor of severe form of disease (Chan 2020, Rokni, 2020).

Laboratory data showing increased risk for thrombosis were summarized in Table **2**.

Table 1. Platelet variables in cases with COVID-19 (Adapted from Wool, 2020).

-	**COVID-19 patients**		**Non-COVID-19 ICU patients**		**Normal range**
	Mean (SD)		**Mean (SD)**		
Platelet count, $x10^9$/L	234.1	151.1	250.3	152.7	150–450
MPV, fL	11.58*	1.04	10.49	1	9.0–12.4
-	median	range	median	range	
IPF $x10^9$/L	42.57	12.2–99.5	4.25	3.3–5.2	1.25–7.02
IPF %	14.63	4.2–25.0	7.05	6.4–7.7	3.3–8.6

N = 20, *i.e*., 10 pairs of patients matched for platelet count.
* *p* = 0.013, paired Student's *t* test (compared to non-COVID-19 patients). MPV, mean platelet volume; IPF, immature platelet fraction.

CHAPTER 5

Chest Pain and Acute Coronary Syndromes (ACS)

Abstract: Acute coronary syndromes (ACS), especially acute myocardial infarction (AMI), is the leading cause of death in the world. These represent damage to the cardiac myocytes in the setting of acute cessation of blood supply. Chest pain is a common presentation in patients with AMI; however, there are multiple non-cardiac causes of chest pain. The diagnosis cannot always be made based on the initial presentation. The emergent evaluation of a patient with probable ACS includes a careful assessment of history, risk factors and presenting signs and symptoms, *de novo* ECG abnormalities, and workup of cardiac troponins. Validated risk scores, such as HEART, TIMI, and GRACE, can be helpful in predicting outcomes and the likelihood of ACS in a patient with chest pain. ECG should be performed within 10 minutes of presentation. ST elevation MI (STEMI) is diagnosed with elevated ST segments in two consecutive leads on ECG. Likewise, elevated levels of cardiac troponins in the first hours of presentation are mostly a prerequisite for diagnosis.

Although cardiac catheterization is viewed as the standard diagnostic modality for coronary artery disease, exercise testing, stress studies, echocardiography, and coronary computed tomography angiography (CCTA) may be important adjuncts. Cardiac catheterization laboratory (CCL), coronary care units, EDs, EMS, and primary care institutions need to cooperate in unison to produce the best results for public health.

This chapter gives a brief outline of the diagnosis and management of ACS in the pandemic period.

Keywords: Acute coronary syndrome, Acute myocardial infarction, Cardiac catheterization, COVID-19, ST elevation myocardial infarction.

INTRODUCTION

Cardiovascular disease is the most common cause of death among adults in most parts of the world. These may be deaths in a short time following Acute Myocardial Infarction (AMI) or may develop as a result of other acute coronary syndromes (ACS). Approximately half of the patients with out-of-hospital cardiac arrest with the first rhythm identified as VF and who survive hospital admission have evidence of acute MI. Of all out-of-hospital cardiac arrests, .50% will have significant coronary artery lesions on acute coronary angiography (Al-Khatib SM, 2018). Sudden cardiac death (SCD) constitutes major public health problems,

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accounting for approximately 50% of all cardiovascular deaths. For this reason, a great economic resource is allocated for the prevention of cardiovascular diseases (CVD) in the world, especially in developed countries. In developing countries, on the other hand, larger bills are faced because therapeutic approaches are prominent rather than preventive medicine.

ETIOLOGY

The inability to meet the oxygen requirement of the heart with the supplied blood for a certain period of time and the accumulation of substances such as lactic acid and free radicals in the myocardial tissue precipitates chest pain (CP). In other words, it is acute ischemia of myocardial cells that directly triggers the pain.

The amount of blood passing through a vein is proportional to the diameter of the vein. When atherosclerosis reduces the vessel diameter by half, there is a serious decrease in the blood carried by vessel. As a rule, reduced blood flow to the coronary arteries is caused by atherosclerosis. However, sometimes abnormal spasm of the arteries can also cause insufficient blood flow, which is called vasospastic angina or “Prinzmetal's angina”.

CP is the most common complaint of AMI. However, CP has many causes other than AMI or CVD. History is an important aid in distinguishing them. Pain or discomfort radiating to the shoulder, arm, neck, or jaw may indicate heart disease. Since ischemia afflicts dermatomes between C8 and T4, this kind of spreading pain occurs. In many cases, pain can be defined in the areas listed in addition to CP, or in some cases, only these pains can be noted. For example, an AMI case may present with neck or arm pain without CP.

PATHOGENESIS

CP is divided into visceral or somatic, in accord with the mechanism. Visceral pain is pain caused by internal organs such as the heart, blood vessels, esophagus, and visceral pleura. Somatic pain is easily identified, its location is well-defined and indicated (*e.g.*, by the finger), and it is a sharp pain, while visceral pain is not well localized due to pain fibers entering the spinal cord at different levels, difficult to describe, vaguely defined, unclear and blunt. There is also a psychological component in vital diseases such as ACS, DAA, and PE. This consists of fear of death (*angor animi*, severe anguish, nonspecific fear, and anxiety).

Noncardiac Chest Pain

NCCP is also a common presentation encountered in routine practice. Most of

them are classified in the 'pleuritic' CP and are of the nature of “somatic pain” (Table **1**). Well-defined, sharply circumscribed area of pain is generally in this category, but it should be noted that there may be exceptions. In other words, a pain that appears to be precisely somatic may, in fact, be a harbinger of severe visceral pain, for example, ACS or aortic dissection.

Table 1. Causes of pleuritic or somatic CP include.

• Pulmonary embolism (PE).
• Pneumothorax.
• Pneumonia.
• Pericarditis.
• Serositis/connective tissue disease.
• Malignancies involving the pleura.
• Pathologies below the diaphragm.
• Musculoskeletal disorders

Angina pectoris is examined under two headings: **Stable angina and unstable angina pectoris (USAP).** Stable angina pectoris (SAP) is the feeling of pain with ischemia as the oxygen requirement of the heart increases during effort without coronary thrombus. SAP attacks always begin with physical or emotional stress, and often the patient recognizes and predicts this pain. SAP usually resolves when the patient is at rest or with the use of agents such as isosorbide dinitrate or with oxygen. USAP is not so easily relieved. USAP can start at rest, even while asleep. It is also called preinfarction angina because it often represents underlying severe atherosclerosis (Table **2**). Fig. (**1**) illustrates the advancement of coronary arterial atherosclerotic process and its reflections on ECG.

Table 2. The criteria sought for the definition of USAP.

- anginal pain that started for the first time in the last 1.5 months
- the change in the duration and characteristics of the pain (*e.g.*, it used to last 3 minutes but now it is 15 minutes, or it used to be start while running but now walking)
- Pain within the first 2 weeks after AMI
- Pain precipitated in the early period after PTCA
- Pain concurrent with changes in ECG findings.

CHAPTER 6

Heart Failure and Acute Pulmonary Edema (APEd)

Abstract: Heart failure (HF) is a complex syndrome in which the cardiac output cannot meet the demand, *i.e.*, metabolic needs of the tissues and reflect the impairment of the heart's pump function. This condition is also referred to as congestive heart failure (CHF) as it is mostly associated with fluid retention.

The four main factors that determine the pump function of the left ventricle, which are contractility (contractility), preload, afterload and heart rate.

Accepted guidelines divided patients with HF into three groups according to their left ventricular ejection fraction (EF). The group with a EF below 40% continues to be known as a "low/reduced EF" (HF-REF), and a group of 50% and above remains "preserved EF" (HF-PEF), while a group of 40–49% is at the border (mid-range), thus it was named mildly reduced EF" (HF-MREF). The incidence of HF-PEF increases with age. The majority of cases in the elderly is due to HF-PEF. Acute decompensated HF is a deadly cause of cardiac dysfunction that can present with acute respiratory distress. There are many different causes of APEd, though cardiogenic pulmonary edema is usually a result of acutely elevated cardiac filling pressures. Clinical findings develop as a result of impaired perfusion and/or venous distension, with resultant surge in pressure. The patient mostly present with progressive symptoms of HF or acutely appeared signs of left-sided decompensation.

Patients who are diagnosed with HF for the first time and who is admitted with APEd should be hospitalized and treated accordingly. HF develops in 10 to 20% of AMI cases. Since this group has a high mortality, it must be identified and treated.

The main objective of the treatment in the Acute Left HF is to provide the respiratory and cardiovascular stability as soon as possible. The main goal is to "dry" the lungs, not just throwing off water.

COVID-19 pneumonia and respiratory distress can masquerade APEd in the pandemic period. Most "typical" radiological findings including ground-glass opacities are common in both entities. It is very frequent that a clinician mixes up the two entities, especially misinterpret APEd as COVID-19, because the outbreak affects so many people that every physician is conditioned to see the viral pneumonia. Therefore, educational resources should stress on how to implement correct differential diagnosis of cardiopulmonary entities including AHF/APEd in the pandemics in both hospital

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and outpatient conditions. This chapter provides a general overview of the diagnosis and management of HF and APEd with a special emphasis on the acute presentation in the pandemic era.

Keywords: Acute pulmonary edema, Congestive heart failure, COVID-19, Dyspnea, Heart failure, Left ventricular dysfunction.

INTRODUCTION

Heart failure (HF) is a complex syndrome in which the cardiac output cannot meet the demand, *i.e.*, metabolic needs of the tissues and reflect the impairment of the heart's pump function. This condition is also referred to as congestive heart failure (CHF) as it is mostly associated with fluid retention. Between 90% and 95% of patients with HF are considered congestive patients.

ETIOLOGY FRANK-STARLING LAW

The mechanisms underlying the "Frank-Starling Law of the heart" states that the stroke volume of the left ventricle will increase as the left ventricular volume increases due to the myocyte stretch causing a more forceful systolic contraction. This assumes that other factors remain constant. The functional importance of the Frank-Starling mechanism lies mainly in adapting left to right ventricular output. In a clinical situation, when increased volumes of blood flow into the heart (increasing preload), the walls of the heart stretch (Seres, 2011). The myocytes contract with increased force and, within limits, empties the expanded chambers with increasing stroke volume. Lengths of myocytes and sarcomere eventually become supranormal and therefore contraction forces are reduced below normal.

Types and Clinical Presentation

Patients with HF mostly present to the ED or call an ambulance with left ventricular dysfunction, briefly "left HF" and resultant respiratory signs and complaints (such as dyspnea/APEd, shortness of breath, paroxysmal nocturnal dyspnea, *etc.*).

The basic mechanism of "right HF" is that excess water is sequestered in the body (venous system) due to pump failure. Complaints occur with pretibial edema, fluid accumulation in third cavities, hepatomegaly, hepatojugular reflux, and jugular venous distention. However, life-threatening or critical condition due to right HF is very rare.

European and American Cardiology associations have published new consensus reports on the diagnosis and treatment of HF. For example, in the 2016 ESC HF

guidelines, patients with HF were divided into three groups according to their left ventricular ejection fraction (EF). The group with a EF below 40% continues to be known as a "low/reduced EF" (HF-REF), and a group of 50% and above remains "preserved EF" (HF-PEF), while a group of 40% to 49% is at the border (mid-range), thus it was named mildly reduced EF" (HF-MREF) (Ponikowski *et al*.; Yancy *et al*., Nadar S).

Characteristics of patients with HF

The EAHFE registry has prospectively collected 13,971 consecutive AHF patients diagnosed in 41 Spanish EDs (Llorens, ICA-SEMES Research Group, 2018). Compared to other large registries, patients in the EAHFE registry were older (80 years), more frequently women (55.5%), and had a higher prevalence of hypertension (83.5%) and a lower prevalence of ischaemic cardiomyopathy (29.4%). *De novo* AHF was observed in 39.6%. More than half of the sample (56.1%) had HF-PEF. 56.8% of the patients arrived at the ED by ambulance, 4.5% arrived hypotensive, and 21.3% hypertensive. Direct discharge from the ED was noted in one-fourth of the patients. The length of hospitalisation was 9.3 (8.6) days, and in-hospital, 30-day, and 1-year all-cause mortality were 7.8, 10.2 and 30.3%, respectively; and 30-day re-hospitalisation and ED revisit rates due to AHF were 16.9 and 24.8%, respectively.

The incidence of HFpEF increases with age. The majority of cases in the elderly is due to HFpEF. Acute decompensated HF is a deadly cause of cardiac dysfunction that can present with acute respiratory distress. In decompensated cases, pulmonary edema and the rapid accumulation of fluid within the interstitial and alveolar spaces leads to significant dyspnea and respiratory distress. There are many different causes of APEd, though cardiogenic pulmonary edema is usually a result of acutely elevated cardiac filling pressures (King, 2020) (Table **1**).

Table 1. HF types (from severe to mild) and associated clinical and laboratory variables.

HF-LEF (low EF)	HF-MEF (medium EF)	HF-PEF (protected EF)
Symptoms + signs		
LVEF<40%	LVEF=40%-49%	LVEF>50%
BNP can rise (not a rule)	BNP rises	
Systolic dysfunction	Structural heart disease and/or diastolic dysfunction	

Pathogenesis

Four main factors determine the pump function of the left ventricle: Contractility, preload, afterload and heart rate. CHF occurs because of a marked decrease in

CHAPTER 7

Acute Pulmonary Embolism (APE)

Abstract: Acute pulmonary embolism (APE) is one of the diseases posing immense death rates and a great burden to public health. APE defines a blood clot or other substance in the deep leg/calf vein that traverses through the right heart and blocks the pulmonary arterial bloodflow (venous thromboembolism, VTE). The severity of the signs and symptoms of APE depends on the size of the thrombus and location of the occlusion, together with the previous reserves of the individual. A presentation template that will fit all cases cannot be put forward. "Massive" or hemodynamically unstable PE has a high death rate despite contemporary management. Healthcare personnel should be alerted to recognize untreated patient with high probability for APE in the ED and primary care institutions. Treatment should be expedient and aggressive in accord with the patient's instability. Systemic or catheter-mediated thrombolysis, anticoagulation and other approaches should be contemplated immediately after general supportive measures.

This chapter delineates diagnostic dilemmas, distinctive properties and management principles of APE in the emergency setting. Also, challenges brought into scene with COVID-19 pandemics is discussed.

Keywords: Acute pulmonary embolism, Thromboembolism.

APE causes 200,000 deaths annually in the USA and is the 3rd cause of death in hospitalized patients. Most cases are recognized at autopsy, which means they die without being recognized. Less than one-tenth of fatal cases can receive specific treatment.

The reason it is difficult to recognize is that its symptoms and signs are usually among those that can be found in many other entities.

APE is a pathologically formed blood clot or other substance in the deep leg/calf or –rarely- upper extremity venous system that passes through the right heart and blocks the pulmonary arterial bloodflow. When the clot migrates (embolism), it is called venous thromboembolism (VTE) (Table **1**).

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Table 1. Some materials reported to be embolized into the pulmonary circulation are as follows.

Material	Mechanism/Cause
air	neurosurgical interventions, central venous catheter,
amniotic fluid	labor
fat	long bone fracture
foreign bodies	injection in IV drug addicts,
parasite eggs	schistosomiasis,
septic embolism	acute bacterial endocarditis,
tumor cells	renal cell carcinoma.

Risk factors for thrombosis in the venous system are prolonged immobility (such as prolonged bed rest, post-operative period), thrombophlebitis, use of certain drugs (such as oral contraceptives), heart failure, advanced age, DVT, malignancy and some rare blood diseases (Table **2**).

Table 2. Predisposing factors for APE.

Strong (OR > 10)	Moderate (OR 2-9)	Weak (OR<2)
Lower extremity fractures	Arthroscopic knee surgery Use of agents	Bed rest >3 days
Hospitalization with heart failure or atrial	stimulating erythropoiesis.	Diabetes mellitus
fibrillation/flutter in the last 3 months	Congestive heart failure/respiratory	Hypertension
Hip or knee replacement surgery	failure	Prolonged journeys
Major trauma	Malignancy and/or metastases	Advanced age
ACS/AMI in the last 3 months	Infections (esp. pneumonia, UTI/HIV)	Laparoscopic surgery
Past APE/VTE	IVF procedures	Obesity
Spinal cord injury	CVA, stroke	Pregnancy
	Inflammatory bowel diseases	Varices

Thromboembolism is defined as 'provoked' if it occurs with temporary or reversible reasons within 6 to 12 weeks from diagnosis (such as surgery, trauma, immobilization, pregnancy, use of oral contraceptives).

Since pulmonary arterial bloodflow is blocked due to APE, the right heart works against a substantially increased resistance. This causes an increase in pulmonary capillary pressure (wedge pressure, PAWP). As a result of the sequestration of the area fed by the occluded vessel, the mechanics of A-a gradient, in other words gas exchange, are impaired.

The severity of the signs and symptoms of VTE and APE depends on the size of the thrombus and occlusion, which vessels are occluded, and the person's previous reserves. A presentation template that will fit all cases should not be sought.

The patient usually complains of acute onset, dyspnea unexplained by an obvious cause. This may be accompanied by chest pain and tachypnea. Immobilization history is often detected but is not the rule. Dyspnea occurs in 75% to 85% of the cases, and pain on inspiration occurs in 65% to 75%. Tachypnea is the only reliable finding found in more than half of the patients.

Pearl: If none of The Trio *i.e.*, Dyspnoea, Tachypnea, Chest Pain, Tachycardia is Present in a Given Patient, APE Can Be Safely Ruled Out.

In the PIOPED I (Prospective Investigation of Pulmonary Embolism Diagnosis) study involving patients with angiographically proven PE, at least one of the following three findings was found in 97% of the patients; dyspnea, chest pain triggered with inspiration and tachypnea (PIOPED Investigators, 1990). On physical examination, tachypnea, dyspnea and tachycardia are usually found together. In a patient with massive PE, signs of right HF/ventricular strain such as neck vein distension and sometimes hypotension are observed.

Massive or nonmassive? Massive PE was defined as featured by hypotension (systolic BP below 90 mmHg or a drop of >= 40 mmHg persisted for at least 15 min which is not caused by *de novo* arrhythmias) or apparent findings consistent with shock (Sekhri, 2012). Massive PE has a high death rate despite contemporary management. Of note, some patients with hemodynamically stable, nonmassive PE but documented to have right ventricular dysfunction *via* echocardiography is classified as “submassive PE”.

CASE

In Italy, a 50-year-old man presented with acute chest pain and exertion dyspnea eventually had respiratory failure worsening with PTE, during the COVID-19 pandemics (Lorenzo, 2020). There is no thrombosis related medical history. There is a history of fever, weakness and cough two weeks before admission. The home PCR test had been found positive and the patient was isolated at home. He recovered within 10 days without any medications. On the 13th day, she presented to the ED with sudden-onset dyspnea and chest pain. Hypoxic and hypocapnic respiratory failure were noted. Hypercoagulability has been demonstrated by thrombelastometry. Hemodynamics are stable. USG revealed DVT in the left tibial vein, and CTPA demonstrated left lobar, segmental and subsegmental PTE, and bilateral interstitial pneumonia (Fig. **1**). IgM and IgG tests are also positive for mycoplasma pneumonia.

CHAPTER 8A

Hypertension and Aortic Diseases in The Pandemic Era

Abstract: The term hypertension (HT) is a chronic condition that leads to damage to target organs if untreated expediently. On the other hand, a hypertensive emergency refers to an acute elevation in blood pressure (BP) with evidence of end-organ injury, while hypertensive urgency defines acute BP elevation without progressive target organ dysfunction. Hypertensive emergencies comprise pulmonary edema/left ventricular failure, coronary syndromes, neurological deficits/intracranial hemorrhage, acute kidney injury, retinal hemorrhages, dissecting aortic aneurysm, and eclampsia.

The BP needs to be decreased expediently in the management of hypertensive emergencies. In the rest of the cases, the BP should be reduced in a gradual manner to preclude dangerously reduced cerebral perfusion pressure.

HT is also linked to COVID-19 as a comorbidity linked to a severe clinical course of the infection. On the other hand, dissecting aortic aneurysm (DAA) is most commonly seen after the age of 50 in hypertensive men who smoke. Most emergent aortic diseases appear to be a complication of HT and represents a major threat to public health. Clinicians should be alerted to recognize untreated patient with HT and aortic catastrophes in the emergency setting and primary care institutions.

Keywords: Aortic aneurysm, Aortic diseases, Aortic dissection, Hypertensive emergency, Hypertensive urgency.

HYPERTENSION: BOTH ACUTE AND CHRONIC DISEASE OF THE EMERGENCY DEPARTMENT

Although hypertension (HT) is mostly recognized as a disease that damages specific tissues in the chronic process, these patients can be admitted to the EDs with acute elevations in blood pressure (BP), hypertensive urgencies, and rarely, hypertensive emergencies. When it is presented with end organ damage, it is called hypertensive emergency and requires emergency treatment. HT as a chronic disease, whose prevalence is as high as 8% to 10% common in the society, is frequently encountered in the ED and primary care units.

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CLINICAL OUTCOMES OF HT

Chronic persistence of HT from the fourth decade (thirties) causes damage to many organs with advancing age. Renal failure, retinal hemorrhages, intracranial hemorrhage and other forms of stroke, acute coronary syndromes, peripheral artery disease, pulmonary edema are among them.

In the NHANES (National Health and Nutrition Examination Survey) study, which enrolled more than 23.000 patients in the USA, it was reported that more than 50% of mortality due to coronary syndromes and stroke were recorded in hypertensive cases.

BP is an important vital sign which directs clinical decision making processes. Accurate and consistent measurement of BP is a main part of patient assessment in most clinical settings. In general, two values are recorded during the measurement of blood pressure. The first, systolic pressure, represents the peak arterial pressure during systole. The second, diastolic pressure, represents the minimum arterial pressure during diastole (Rehman, 2021). A third value, mean arterial pressure, can be calculated from the systolic and diastolic pressures (Table **1**, Fig. **1**).

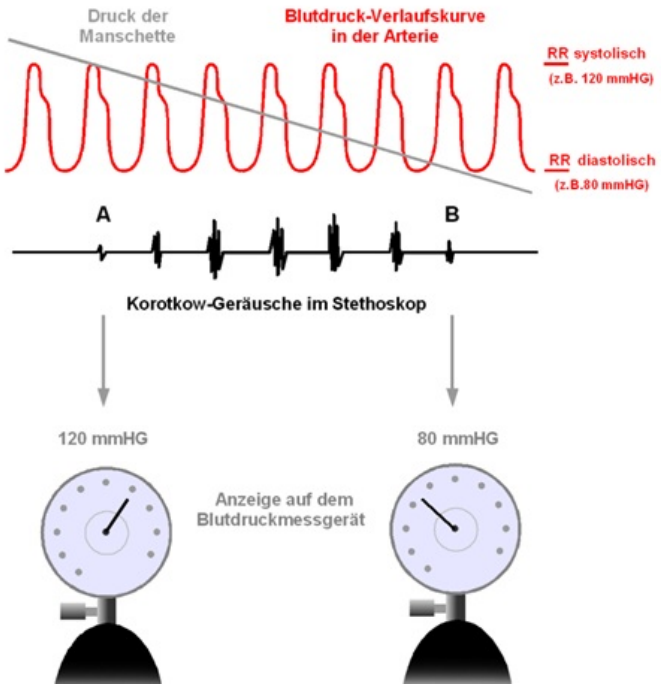

Fig. (1). Schematization of blood pressure evaluation.

Table 1. Definitions in blood pressure measurement.

SBP	Korotkoff Sounds: The First Phase
DBP	Korotkoff sounds: The fifth phase
Pulse pressure	SBP-DBP
Mean arterial pressure (MAP)	DBP + 1/3 Pulse pressure
Mid-BP	Average of DBP and SBP

Hypertensive Emergency

acute, progressive target organ dysfunction with severe BP elevation (> 180/120 mmHg).

Hypertensive Urgency

Acute, severe BP elevation without progressive target organ dysfunction.

How can One Discern HT Emergency and Urgency?

There is no threshold value of BP to distinguish between hypertensive emergency and urgency. The distinctive feature is detection of target organ damage. The diagnosis is made with the patient's clinical status, not figures indicative of the BP.

Findings Compatible with Hypertensive Emergencies

Hypertensive encephalopathy, Ischemic stroke, Intracranial hemorrhage/SAH, pulmonary edema, *de novo* heart failure, Acute MI and other coronary syndromes, Aortic dissection, renal failure, Eclampsia, Sympathetic crisis.

Hypertensive Urgent Situations

BP value higher than Stage II HT and accompanying severe headache, dyspnea, nosebleeds or agitation.

PHYSICAL EXAM AND EVALUATION OF THE HYPERTENSIVE PATIENT

The evaluation for hypertensive conditions varies with the severity of the presentation, *i.e.*, symptoms and signs on admission. The patient should be questioned about if the manifestations suggest an emergency condition, adjunctive studies such as cranial computed tomography (CT), ECG, laboratory markers of damage to kidney and heart, such as urea and creatinine, electrolytes, B type

CHAPTER 8B

Aortic Diseases: Abdominal Aortic Aneurysm (AAA) and Dissecting Aortic Aneurysm (DAA)

Abstract: Aneurysmal dilation is most common in the aorta, distal to the kidney vessels and proximal the iliac artery bifurcation. It is much more frequent in males than in females. It most commonly develops in middle aged and geriatric patients, patients with chronic HT, atherosclerosis, smoking history, and those with a genetic propensity for AAA, although none of this is an absolute rule.

The width of the aorta varies depending on the race, body area, gender and age, and the average aortic diameter is between 2.5 and 3.7 cm in general. Aortic diameter measuring 50% more (1.5 times) than expected is considered an aneurysm. If the diameter of the aorta is > 5 cm, the possibility of rupture increases and requires surgical intervention. In the abdominal aorta, which is generally located infrarenal,> 30 mm for both sexes is described as AAA.

In recent years, the term "Acute Aortic Syndrome" has also been used for all aortic emergencies. Signs and symptoms of AAA varies with the patient's physiologic reserves, age and the extent of the disease with resultant organ damage (Table **1**).

Keywords: Abdominal aortic aneurysm, Aortic diseases, Dissecting aortic aneurysm, Hypertension, Management.

If any of the above are found, you should suspect AAA. The patient's abdomen can be gently palpated. Excessive or forceful palpation can lead to rupture.

Crawford classification is used to describe the extent of AAA (Table **2** and Fig. **1**).

A diagnosis of AAA is considered if the ascending aorta has reached 5.5 cm in diameter, or:

• If the diameter of the ascending aorta has increased by 5 mm in the last 6 months.

• If the ascending aorta is 5 cm and the patient has labile hypertension.

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• If the descending aorta is 6 cm in diameter or a 5 mm increase in diameter has been noted in the last 6 months.

• If there is compression to any other organ.

• If the abdominal aorta is 5.5 cm or there has been an increase in diameter of 5 mm in the last 6 months.

• In all aneurysms, if there is a suspicion of bleeding, aortic aneurysms should be treated either surgically or by endovascular (interventional) method (EVAR, TEVAR).

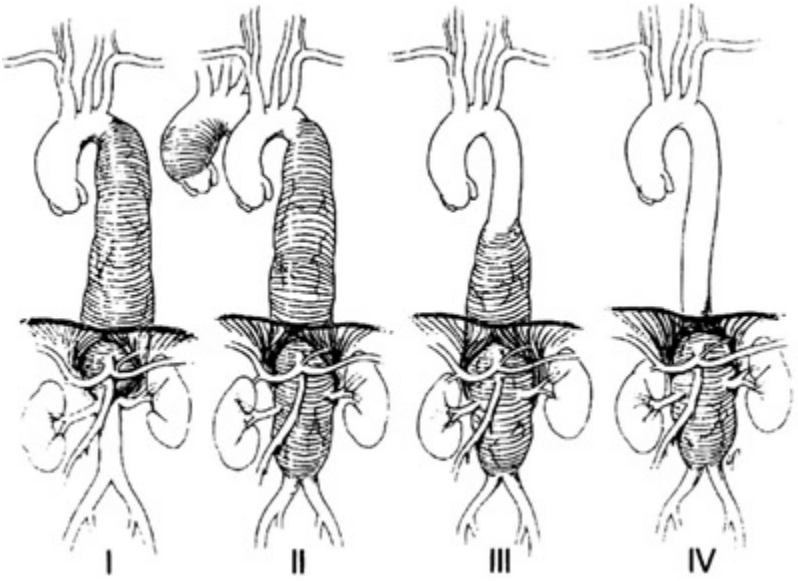

Fig. (1). Crawford classification.

Table 1. AAA signs and symptoms.

• abdominal pain
• back and flank pain
• hypotension
• feeling of defecation due to retroperitoneal bleeding
• pulsatile mass (usually palpable if the diameter of the mass is above 4-5 cm in thin or normal body builds.)
• weak femoral pulse (unilateral or bilateral)
• signs of gastrointestinal bleeding if the aneurysm forms a fistula into the intestines

Table 2. Crawford classification is used to describe the extent of AAA. According to this classification (Fig. 1).

Type I: Aneurysm starts in the proximal descending aorta and ends at the proximal renal artery bifurcation.
Type II: The abdominal aorta is aneurysmatic, from the proximal descending aorta to the distal of the renal arteries.
Type III: The abdominal aorta is aneurysmatic to the distal descending aorta and to the distal of the renal arteries.
Type IV: The abdominal aorta is aneurysmatic starting below the diaphragm, involving the visceral arterial origins.

In most patients with AAA, the clinical manifestation is very mild or even asymptomatic unless the aneurysm is ruptured. Symptomatic patients complain of chest or back pain. The type of pain can be in different ways. Symptoms related to compression to adjacent structures may be remarkable: superior vena cava syndrome, hoarseness, stridor, dysphagia, Horner syndrome, rupture and tamponade-related embolism, CHF, shock. In addition, 16% of the cases have CAD, 10% have peripheral artery disease, and 10% have CVA.

RUPTURED AAA

Anterior intraperitoneal (20%), posterior retroperitoneal (75-80%) rupture can be seen. More rarely, rupture may occur in the form of fistulization to adjacent structures, stomach or intestine. In the case of more frequent retroperitoneal rupture, cardiovascular collapse is delayed due to the buffering effect of surrounding structures, often only temporary syncope/presyncope attack occurs. In anterior intraperitoneal rupture, sudden cardiovascular collapse occurs more rapidly.

A small group of patients may also have chronic rupture. These patients can present with chronic low back or groin pain.

DIAGNOSIS

The sensitivity of palpation in the diagnosis of AAA is around 68%, it can be up to 82% in some large AAA. Therefore, when AAA is suspected, especially in hypertensive men and smokers over 60-65 years of age, it is not satisfactory to complete the physical examination. Bedside USG should be performed and time should not be wasted for more advanced techniques including CT. It should be kept in mind that USG is not sensitive enough for retroperitoneal rupture.

Although contrast-enhanced tomography/angiography is the gold standard in diagnosis, patients with renal insufficiency can be recognized with non-contrast

CHAPTER 9

Supraventricular Arrhythmias and Their Management in the Emergency Setting: PSVT and AF

Abstract: Supraventricular tachycardia (SVT) is a type of tachyarrhythmia with a narrow QRS complex and regular rhythm (heart rate >100 bpm). These patients are often symptomatic and present to the emergency department (ED) in acute attacks called paroxysmal SVT (PSVT). Most SVTs are regular rhythms. It starts suddenly with the reentry mechanism in the majority of patients. 60% of the patients have reentry with Atrioventricular (AV) node, and 20% have reentry *via* bypass pathways. Coronary artery disease, anginal chest pain and dyspnea occur in patients due to tachycardia. Heart failure and pulmonary edema may occur with left ventricular dysfunction. Vagal maneuvers and adenosine appear to be the treatments of choice for termination of stable SVT.

Keywords: Adenosine, Paroxysmal supraventricular tachycardia, Supraventricular tachycardia, Tachyarrhythmia, Vagal maneuvers.

IN MULTIFOCAL ATRIAL TACHYCARDIA (MAT)

On the other hand at least 3 different P wave morphologies originating from the atrium are observed in the ECG tracks. There are also variable PP, PR and RR ranges. Treatment is to correct the underlying disease. Specific antiarrhythmic therapy is rarely required.

PATIENTS WITH ATRIAL FLUTTER (AFL)

Also clearly sense that the attack has started, and they tend to come to the ED with more unstable findings. It may also accompany acute coronary syndromes.

Atrial fibrillation (AF) is the term used to define the inactive 'worm bag-like' oscillations of the atria, which means there is no true atrium contraction. Ruling out atrial or ventricular thrombi with echocardiography/POCUS is important to avoid embolization. Priority should be given to hemodynamic stability and deter-

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mination of factors that trigger the underlying disease. IV beta-blocker and diltiazem or verapamil are the drugs of choice for acute rate control in AF with rapid ventricular response.

SUPRAVENTRICULAR TACHYCARDIAS

Supraventricular tachycardia (SVT) is a type of arrhythmia with a narrow QRS complex (<120 msec) and regular rhythm. Heart rate is over 100 per minute. In its pathophysiology, it has been shown that the factor causing arrhythmia is impulse formation and abnormalities in the conduction pathways. The most common mechanism is the reentry mechanism. Patients may present with asymptomatic, minor palpitations or severe symptoms. They are often symptomatic and present to the emergency department (ED) in acute attacks called paroxysmal SVT (PSVT).

Epidemiology

The incidence of SVT is approximately 1 to 3/1000. Its prevalence has been reported as 2.25/1000 and the number of new cases seen each year as 35/100000. Its prevalence increases with age. Atrioventricular (AV) Nodal Reentrant Tachycardia (AVNRT) is most common in middle age and older, while SVT with accessory conduction is more common in young adults. PSVT can also be seen in patients who present with previous myocardial infarction (MI), mitral valve regurgitation (MVR), rheumatic heart disease, pericarditis, pneumonia, chronic obstructive pulmonary disease (COPD) and intoxications *via* alcohol/caffeine/energy drink, apart from healthy individuals.

Ectopic SVT usually originates in the atrium and the atrial velocity is 100-250 bpm (most often 140 to 200 bpm). Regular P waves may be misdiagnosed as atrial flutter or 2: 1 AV block, sinus rhythm. Reentrant SVT is seen in most patients with SVT. A total of 60% of the patients have reentry with AV node, and 20% have reentry *via* bypass pathways. The normal heart tolerates the typical SVT rate of 160-200 bpm in days or hours. However, cardiac output is generally decreased and may lead to signs of heart failure (HF) in the intact myocardium in those with high heart rates.

Pearl: The rhythm is considered supraventricular if the QRS complex is narrow (<0.12 seconds), or if the QRS is wide in the setting of a previously known branch/fascicular block or rate-dependent aberrant conduction.

Reentrant SVT usually starts when the AV node encounters an ectopic atrial

impulse while the AV node is in the partial refractory period (Fig. **1**). From here, the impulse proceeds with two different functional parallel arms. The node is below the ventricular ending and above the atrium. In the case of AV nodal reentry, QRS complexes usually hide the P waves and are not visible. These have a 1: 1 message and QRS complexes are normal.

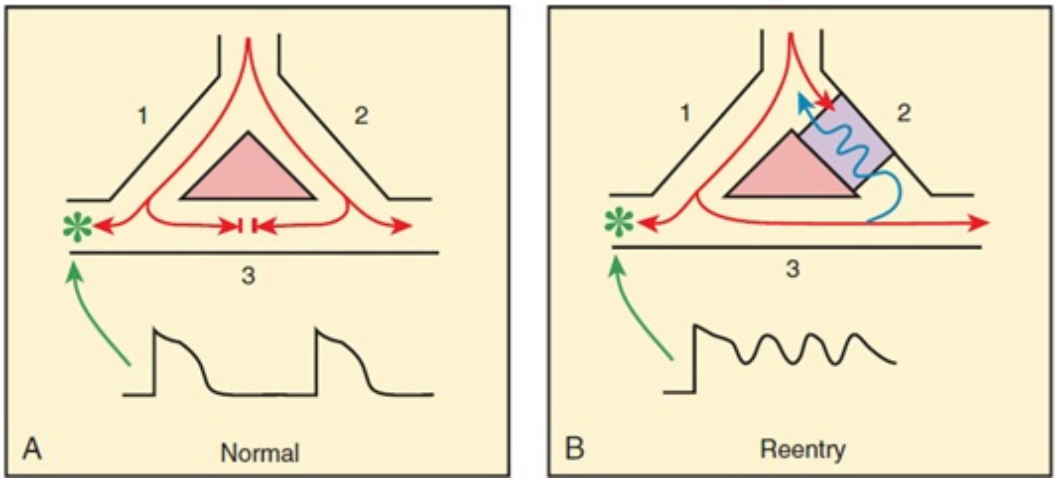

Fig. (1). Cardiac conduction in SVT. A goes down from normal depolarization pathways 1 and 2 and fades away at 3 (repolarization). B, Reentrant pathway. 1: Normal conduction; 2: there is delay/slowing down due to unilateral block; 3: normal conduction pathway.

Symptomatology

Most patients with SVT are symptomatic (Table **1**). The most common symptom is palpitations. Complaints can last from a few minutes to hours. Patients can sometimes present with shortness of breath, air hunger or chest pain. There may be dizziness, lightheadedness and rarely fainting. Dizziness and syncope are more common in those with a heart rate above 170. Syncope in SVT is mostly related to vasovagal factors. Although symptoms such as loss of consciousness and syncope increase the risk, SVT is generally not a serious or life-threatening condition. However, if any of these symptoms develop, they should be investigated with close monitoring.

Table 1. Clinical symptoms and percentages of patients presenting with PSVT.

Symptom	**%**
Palpitation	96
Dizziness/lightheadedness	75
Dyspnea	47
Chest pain	35

CHAPTER 10

Agents Used in the Treatment of Arrhythmias and Advanced Cardiovascular Life Support

Abstract: Advanced Cardiovascular Life Support (ACLS) guidelines recommend certain drugs for hemodynamic stabilization, prevention of collapse, stabilization of a perfusing rhythm, improving peripheral resistance and cardiac output, and restoration of organ perfusion. It is known that no antiarrhythmic agent increases the percentage of patients discharged with good neurological status. For this reason, the commencement of medications and establishing vascular access should not delay high-quality CPR.

ACLS guidelines recommend drug adrenaline in the asystole algorithm and in those with cardiac arrest due to ventricular fibrillation (VF). For pulseless electrical activity (PEA)-related cardiac arrest, adrenaline and, in some cases, sodium bicarbonate is recommended. The drugs used in VF and pulseless VT (PVT) apart from adrenaline are vasopressin, amiodarone, lidocaine, esmolol, magnesium, and procainamide in selected situations. This chapter provides a brief outline of arrhythmias commonly encountered in routine clinical practice, together with principles of ACLS and indications and usage of resuscitative agents employed in these situations.

Keywords: Adrenaline, Advanced Cardiovascular Life Support, Amiodarone, Antiarrhythmics, CPR, Lidocaine, Sodium bicarbonate, Ventricular fibrillation.

INTRODUCTION

According to the ACLS guideline, the recommended drug in the asystole algorithm is adrenaline. For pulseless electrical activity (PEA)-related cardiac arrest (CA), adrenaline and, in some cases, sodium bicarbonate are recommended. The drugs used in ventricular fibrillation (VF) and pulseless VT (PVT) are adrenaline and/or vasopressin, amiodarone, lidocaine, esmolol, magnesium, and procainamide in certain situations.

DRUGS IN ACLS CAN BE EXAMINED UNDER TWO SUBHEADINGS

1. Those used for ACLS at the moment of CA manifested with VF/PVT, Adrenaline, lidocaine, and amiodarone are in this group. ACLS providers should bear in mind that it is unlikely that a perfused rhythm can be instituted merely by drug administration without a high-quality CPR.

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2. Those used in post-arrest management (that is after ROSC is achieved): Beta-blockers (esmolol) and lidocaine infusion can be used in this context. Evaluation should be made case by case basis. These agents are not recommended routinely for every post-arrest patient. *(Class IIb, LOE C-LD)*.

Amiodarone OR Lidocaine may be considered in the treatment of persistent VF and PVT where defibrillation fails to convert to a perfusing rhythm. These drugs are particularly effective when the duration of administration is short, for example, in witnessed arrests due to VF/PVT (Class IIb, Level B-R).

In adult CA, routine use of magnesium is not recommended. (Class III: No Benefit, LOE C-LD). Magnesium is recommended for Torsades de pointes (polymorphic VT accompanying the long QT interval) (Class IIb, LOE C-LD).

Inotropic Support: 70% of CA cases show myocardial dysfunction after ROSC is achieved. Therefore, positive inotropic support should be given in patients with hypotensive patients despite adequate fluid therapy (Marcolini, 2017).

EXTRACORPOREAL CPR (ECPR)

ECPR is bypassing the patient's physiological cardiopulmonary system and providing extracorporeal circulation and oxygenation. For this, cannulation of a large artery and vein and venoarterial extracorporeal circulation and oxygenation are required. The purpose of ECPR is to provide vital organ perfusion if the underlying conditions are deemed reversible.

ECPR is a complex intervention that requires a well-trained and organized team, specialized equipment, and multidisciplinary support within the healthcare system. So far, there are no randomized controlled trials (RCT) that investigate the effects of the use of ECPR for in-hospital or OHCA. Some observational studies have shown that ECPR when used for selected patient groups, increases survival with good neurological outcomes.

Should ECPR be performed? According to the 2019 updates:

1. There is currently insufficient evidence to recommend ECPR in the routine management of CA.

2. ECPR may be a rescue therapy for selected patients when the traditional CPR fails if it can be performed immediately by the specially trained healthcare provider (Class 2B, Level of Evidence C-LD).

END-TIDAL CO_2 ($ETCO_2$) MONITORING

$ETCO_2$ value is the partial pressure of the CO_2 exhaled at the end of expiration. CO_2 production is directly affected by alveolar ventilation and pulmonary blood flow.

In meta-analyses and other studies which recent guidelines are based on, it has been reported that $ETCO_2$ monitoring can predict the clinical course after CA. It is best viewed by waveform capnography. The capnograph curve shows the partial $ETCO_2$ pressure ($PETCO_2$) exhaled after intubation in the vertical axis in mmHg. After the patient is intubated, a significant amount of CO_2 exhaled through the tube is detected, confirming the correct placement of the tracheal tube. The value of the $PETCO_2$ varies through the respiration, the highest point being at the end of expiration.

In the study we conducted in 1997 in patients with nontraumatic CA, we demonstrated that the $ETCO_2$ value at the time of arrest can be an important tool in predicting the survival of CA cases (Karcioglu, 2000).

Apart from ineffective CPR, low $ETCO_2$ values may also result from bronchospasm, trachea, bronchial or tubular obstruction, air leakage in the ventilation circuit, pulmonary edema, and hyperventilation. $ETCO_2$ measurement gives incorrect values in patients in whom supraglottic airway device is inserted, or BVM is attempted. There are not enough studies on this situation, and $ETCO_2$ should not be solely used as a guide (Class III: Harm, LOE C-EO).

$ETCO_2$ levels below 10 mmHg after intubation means there is insufficient CPR or ventilation. If the patient still has a low value after 20 minutes of CPR, the patient's poor clinical course (death) is almost certain (Class IIb, LOE C-LD). On the other hand, initial values above 20 mmHg predict a good clinical course (Ahrens 2001).

$ETCO_2$ may not provide reliable information if the factors above are not excluded. Therefore, caution should be exercised and not interpreted alone (Class IIb, LOE C-LD).

How are the Drugs Given?

The essential route of drug administration within the context of ACLS is IV administration. IO route is the first option when IV administration is not secured in 3 consecutive trials or 90 seconds. Different approaches are also recommended in the literature. If IV or IO administration is not possible, some drugs can be administered *via* the endotracheal tube (ETT) (Table **1**). In this application, the

CHAPTER 11

Electrotherapies: Emergency Defibrillation, Cardioversion, and Transcutaneous Pacing

Abstract: Emergency cardioversion and defibrillation are life-saving procedures that exert direct electric current to the heart through the chest wall in order to terminate lethal tachyarrhythmias. Early defibrillation is life-saving in the survival of adult patients who develop sudden cardiac arrest. In the defibrillation process, myocardial cells are depolarized, and VF is terminated by delivering a certain amount of direct current to the heart, passing through the chest wall. Proper timing and accurate performance of these procedures have a vital role in both survival and recovery post-resuscitation neurological functions without sequelae. Return of spontaneous circulation (ROSC) rates in defibrillation performed without losing time (within 20-30 seconds) can be up to 100% following the occurrence of these lethal rhythms. While cardioversion is performed in pulsating contraction rhythms, defibrillation is an electrical stimulation procedure applied in rhythms that do not generate pulses. In the cardioversion, synchronous energy is exerted onto the QRS complex to convert the rhythm into a sinus rhythm.

When there are signs of instability in rhythms with a pulse, emergency cardioversion (ECV) can be preferred over all other treatments if it is known to have acute onset (less than 48 hours) in atrial rhythm disorders, Transcutaneous pacing (TCP) is a recommended practice for temporary stabilization and invasive techniques such as transvenous pacing (TVP) should be attempted for longer pacing requirements. This chapter gives a brief outline on the outstanding features of electrotherapies (*i.e.*, ECV; defibrillation; TCP, TVP) both in case of life-threatening dysrhythmias and also in urgent non-lethal situations.

Keywords: Cardioversion, Defibrillation, Electrotherapies, Transcutaneous pacing, Transvenous pacing.

INTRODUCTION AND DEFINITIONS

Cardioversion and defibrillation can be summarized as passing a direct electric current through the chest wall to terminate tachyarrhythmias. The purpose of the defibrillation process is to provide high electrical current to the thoracic cage from the outside in a very short time to end lethal arrhythmias, namely, ventricular fibrillation (VF) and pulseless ventricular tachycardia (PVT). A certain amount of non-synchronized direct electrical current is exerted in a random moment of the

cardiac cycle. With the given current, all the excitable tissue of the heart is depolarized, and in this way, reentry foci are stopped. In brief, the heart is "silenced" totally all of a sudden. The myocardial cells all come in line with the same phase, and the dominant pacemaker (*i.e.*, the sinus node, as a rule) is activated. In this way, we target to line up the heart cells all in the same phase and to precipitate coordinated, organized conduction and contraction. The duration of the direct current applied to the thorax for these procedures lasts for less than one second.

The longer the time between the time the PVT or VF occurs and the defibrillation procedure, the smaller the chance of success in defibrillation. ROSC rates in defibrillation performed without losing time (within 20-30 seconds) can be up to 100% following the occurrence of these lethal rhythms. In real life, healthy discharge rates from the hospital do not exceed 50% in the most advanced systems.

Proper timing and accurate performance of BLS have an important role in both survival and recovery post-resuscitation neurological functions without sequelae.

Early defibrillation is life-saving in the survival of adult patients who develop sudden cardiac arrest (SCA) for the following reasons:

1. The most common initial rhythm is VF in patients with witnessed SCA.

2. The treatment of VF is defibrillation.

3. The probability of success with defibrillation decreases significantly over time.

4. When the VF arrests are not defibrillated, they will degenerate in a few minutes to turn into asystole, which is the arrest rhythm with the worst prognosis.

Each minute increases the mortality rate by 7-10% when BLS cannot be started, and defibrillation is not performed in SCA cases due to the lack of a rescuer at the scene at the moment. Defibrillation performed in the first 4 to 5 minutes by the layperson, bystanders who witnessed the event has the potential to save the lives of many adults without neurological sequelae due to hypoxia in the brain. In cases where the defibrillator cannot be reached immediately, it is very important to continue cardiac compressions until the defibrillator arrives. In this way, with the blood flow provided, the duration of VF can be extended and the impairment of brain functions can be delayed (Fig. **1**).

Fig. (1). Delay in defibrillation results in a significant decrease of success rates in time.

The amount of energy to be delivered is adjusted to be the lowest but the most effective energy to terminate VF. The meaning of the success of the defibrillation process is the conversion of VF within 5 seconds following the shock. Although VF may recur after successful defibrillation, these recurrences should not mean that defibrillation has failed. There are many factors that affect the successful outcome of resuscitation, and defibrillation is one of the critical ones. Success in defibrillation procedure and successful resuscitation are separate phenomena. The success of resuscitation includes ROSC, survival and healthy discharge of the patient from the hospital.

Electrical cardioversion (ECV) and defibrillation are different practices. While cardioversion is performed in pulsating contraction rhythms, defibrillation is an electrical stimulation procedure applied in rhythms that do not generate pulses. In the ECV process, synchronous energy is exerted onto the QRS complex to convert the rhythm into a sinus rhythm.

There are only four types of rhythms that are intervened with ECV:

- wide complex tachycardias with a pulse (VT),

- atrial fibrillation,

- atrial flutter and

- paroxysmal supraventricular tachycardias.

CHAPTER 12

Conclusion

"NOTHING WILL BE THE SAME ANYMORE"

The COVID-19 infection, which has been at the forefront of the agenda with the pandemic it has created, harms humanity both by causing death and diseases and by socioeconomic impairments since the end of the year 2019. In this context, public health measures are of vital importance in minimizing losses. The rational use of diagnostic kits, isolation of diagnosed patients, and supportive treatment are indispensable conditions in reducing deaths. Emergency health services, primary care institutions, emergency medicine, internal medicine, infection, and intensive care clinics have to work in cooperation and unison. Algorithms for case management in hospitals should be updated according to contemporary requirements. More importantly, the public should act in cooperation with healthcare professionals and institutions. Both individual and social measures for the prevention and mitigation of the disease can be effective when implemented together with the legislations and regulations of the state. Immediate isolation of the cases with high suspicion or diagnosis and even applying quarantine is key in reducing the famous 'Rt' values below 1.

Combining physical/individual isolation with social solidarity and personal hygiene is vital in order to prevent the production of new cases.

Although the COVID-19 PCR test and antibody tests are important in diagnosis, they are not *sine qua non*, and hospitalization and discharge decisions should be made by keeping the clinical conditions, signs, and symptoms of the patients ahead of the tests. Radiological findings are the most important adjuncts, and sometimes they can be a more important indicator than all other tests.

Our problem with COVID-19 is not limited to diagnostic challenges. New WHO guidelines cite that, there are no specific agents with proven benefit in the treatment of COVID-19. The world, which is far away from creating a truly standardized approach to treatment, has left mankind facing the experience and common sense of local physicians from previous epidemics, and with the knowledge gained from the others to follow on the internet, and the difficulties of the situation itself. Fortunately, some developing countries, including our country,

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have a long tradition of community medicine, public health, preventive medicine, as well as clinical experience, so this process is being overcome with as little loss as possible.

COVID-19 is not the only item to solve in the agenda of emergency medicine. We are the champion of multi-tasking, especially when faced with problems such as reducing cardiovascular deaths, standardization of education, combating the risks of overcrowding in EDs, and preparing for disasters. However, we have lost many of our colleagues, friends in the pandemic era.

And what about our 'sweet hearts'? Cardiac diseases are still one of the greatest threats to one's health on the planet, despite all technological advances achieved in the last decades. For example, life-threatening cardiac arrhythmias including ventricular fibrillation leads to a loss of cardiac function and sudden cardiac death. Guidelines for contemporary therapeutic interventions and for management of patients with in- or out-of-hospital cardiac arrest and refractory arrhythmias in prehospital and emergency settings are key approaches to increase survival and save lives. Advanced modalities for invasive management, including urgent coronary angiography, extracorporeal membrane oxygenation, and other innovated strategies for managing cardiac emergencies are still in development in most parts of the world. Beyond any doubt, pandemic circumstances represent a real challenge to provide usual care to those in need, both in prevention and management of the deadly cardiac diseases.

In brief, it is clear that we will have long years and new generations to live with masks, sanitizers and hand disinfectants. Therefore, long-term evolution of sociocultural codes should be considered to maintain an everlasting struggle with the disease, while preserving welfare of ourselves as human-being. For this, the education of pre-school and school children and women plays a key role.

In summary, cardiac diseases and pandemic threats will not be defeated only by the heroism of physicians, nurses and healthcare professionals. When social and economic measures adapt to healthcare interventions globally, a sought-after 'peak' of the pandemics will be found in a short time, then the disease will be eradicated safely. Only after this, we will be able to continue our search for a mutual and happily shared future as the peoples of the world and society that had learned lessons and experienced from all of these. Above all, respecting natural habitats will help prevent future pandemics, while healthy foods and exercise opportunities for everyone will alleviate risks of cardiac emergencies.

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He has more than 150 articles to his credit published in scientific journals. Besides, he has edited a book which has been published in collaboration of InTechOpen. Another book "Demystifying COVID-19: Understanding the disease, its Diagnosis and Treatment" has been published by Bentham Science Publishers. All these publications have been cited 1544 times so far (ResearchGate).

He's also an instructor of the Advanced Cardiac Life Support Course. His research interests are resuscitation, advanced life support, pain management, airway emergencies, geriatric emergency medicine, disaster medicine, trauma and toxicology, and lastly, COVID-19.