Myocardial Infarction (MI): The Role of Critical Thinking in Intervention Effectiveness

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Abstract

In the case of H.G. presented, classic signs and symptoms of chest pain (that could be associated with MI) are evident. For instance, H.G. experiences chest pain radiating to the left arm and his jaw. In addition, the patient experiences diaphoresis and shortness of breath. With the perceived lack of oxygen supply to the myocardial cells, this outcome could be attributable for the chest pain. Through angioplasty or angiogram tests, H.G. could be exposed to a cardiac catheterization with the aim of visualizing both the recanalization of the artery and the occlusion of his coronary arteries. An additional test entails a chest X-ray to evaluate the potentiality of aortic dissection or heart failure. Similarly, myocardial perfusion scanning could be done to determine the pattern of the flow of blood to the walls of the heart. Lastly, a CT-scan could be administered to determine the extent to which the myocardium may have been destroyed.

Introduction

In the case of H.G. presented, classic signs and symptoms of chest pain (that could be associated with MI) are evident. For instance, H.G. experiences chest pain radiating to the left arm and his jaw. In addition, the patient experiences diaphoresis and shortness of breath. With the perceived lack of oxygen supply to the myocardial cells, this outcome could be attributable for the chest pain. When the intima of the artery is exposed to an accumulation of the lipids, the latter evolves to yield fibrous-fatty lesions. According to Goff, Lloyd-Jones and Bennett et al. (2014), these lesions may contain fibrous caps. When ruptured, the caps are likely to allow the lipid center into the patient's bloodstream. The eventuality is that the formation of clots is stimulated, translating into the process of thrombogenesis. When a patient undergoes the latter, vessels are blocked. In turn, oxygenated blood is blocked from passing and myocardial cells are deprived of nutrients (O'Gara, Kushner & Ascheim et al., 2013).

It is also worth noting that oxygen deprivation triggers electrolyte disturbances due to outcomes such as the loss of magnesium, calcium and potassium from the cells. Upon nutrient deprivation, myocardial cells lose their capacity of contractility. The patient ends up feeling pain that radiates to the arms, back, neck, and the jaw. Similarly, the patient may experience shortness of breath due to the heart's decreased capacity of contractility (Steg, James & Atar et al., 2012). Following the experience of declined muscle contractility, blood from the heart may back up into the patient's lungs. The eventuality is a decrease in the rate of respiration that, in turn, causes shortness of breath. Similarly, shortness of breath is attributed to anxiety among patients. Regarding the presence of diaphoresis in the patient, Mosca, Benjamin and Berra et al. (2011) affirmed that the condition results from sympathetic nervous system stimulations – upon the release of catecholamines. Combined with peripheral vasoconstriction, diaphoresis causes clammy and cool skins.

Methods

The ECG change indicative of the presence of myocardial injury is that which involves a lengthened PRI and the ST-segment elevation. Often associated with the inferior wall myocardial infarctions, the common experience of conduction disturbances arises from an RCA that perfuses the sinoartrial node, leading to an increase in PRI (Peberdy, Callaway & Neumar et al., 2010). Given that the RCA constitutes the coronary arteries exposed to occlusion, the outcome suggests that the heart's inferior wall is the affected area.

Results and Discussion

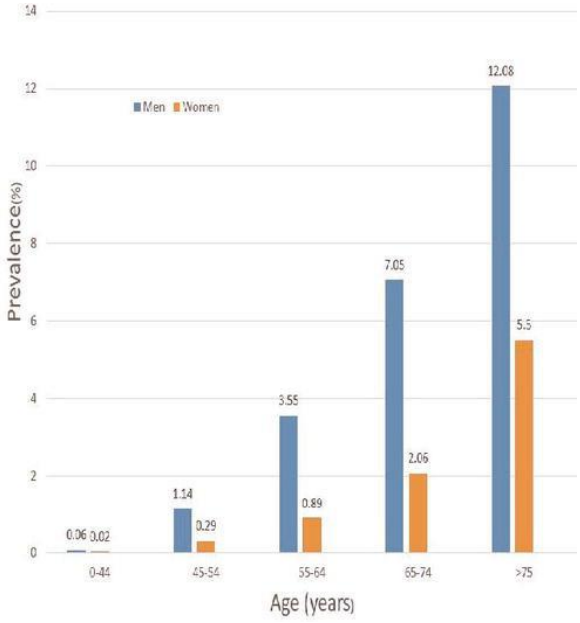

Figure 1: Myocardial Infarction Statistics

Upon damage to myocardial cells, the cardiac biomarkers form proteins that are released into the patient's bloodstream. Notably, each cardiac biomarker poses unique specificity. For example, Troponin is documented to be 100 percent specific to myocardial damage and the onset is reported in three hours, peaking in 24 hours. The condition lasts between 5-10 days (Goff, Lloyd-Jones and Bennett et al., 2014). Another cardiac biomarker that results from the damage of myocardial cells is the Creatine kinase musle/brain (CK-MB). This biomarker is affirmed to be 94 percent specific to myocardial damage and its onset ranges from four to eight hours, peaking in 24 hours. It is further notable that CK-MB lasts between 48-72 hours. The third biomarker used to determine MI is Myoglobin. Its onset is reported to begin in a period between three and six hours, and that it peaks in six to seven hours while the estimated duration within which it lasts is 24 hours. Apart from these cardiac enzymes, LDH forms an additional maker that is used less often and its specificity is rated between 75 and 90 percent. It is further noting that LDH's onset is between 24-48 hours and peaks in a period ranging from 48 to 72 hours, lasting between seven and ten days (O'Gara, Kushner & Ascheim et al., 2013).

According to Steg, James and Atar et al. (2012), cardiac makers are ordered immediately, followed by a 6-8 hour interval (after the first set) and may be done for 3-4 days. The physician's order determines the number of days. Overall, CK-MB and Troponin aid in confirming MI diagnoses (due to their capacity to confirm the presence of necrosed and damaged myocardial muscle cells (Mosca, Benjamin & Berra et al., 2011).

The top priority nursing diagnosis should be related to the decreased state of cardiac output. This diagnosis should be conducted in relation to alterations in the patient's cardiac contractility, as well as alterations in afterload and preload. Notably, myocardial contractility is associated with conditions such as cardiomyopathy, valve malfunction, and ischemia of the myocardium (that cause heart failure), and the effects of renin-angiotensin-aldosterone stimulation (in conjunction with the sympathetic nervous system) that arise in response to the decreased nature of the patient's cardiac output. In addition, myocardial contractility is associated with structural changes that may occur in the heart of the patient (such as a spherical shape, hypertrophy, and dilation); arising from neurohormonal adaptive responses' prolonged activation.

Initially, oxygen was supplied at the rate of three liters per minute. This decision was to aid in increasing oxygen supply to the patient's myocardium. To prevent myocardial ischemia and increase vasodilation, the first 48 hours would see an SL-NTG vasodilator used in conjunction with the administration of Nitroglycerin 1/150 gr. Similarly, NTG plays the role of enhancing the supply of oxygen to the patient's myocardium. After STEMI, possibilities of dysrhythmias are decreased by administering Amiodarone. With the latter forming an antidysrhythmic of choice, an argument in favor of its adoption avows that it poses the capacity to reduce death rates by 13 percent (following an MI). Another potent vasodilator concerns Morphine. Its crucial role lies in the ability to increase the supply of oxygen to the myocardium of the patient. According to Peberdy, Callaway and Neumar et al. (2010), Morphine is also preferred because of its ability to reduced anxiety and pain arising from acute MI.

Regarding the procedure of blood flow restoration to the affected tissues, the fibrinolytic therapy adopted is Retevase. This therapy enhances the stimulation of the lysis of the clot through the conversion of inactive plasminogen into plasmin. As asserted by Goff, Lloyd-Jones and Bennett et al. (2014), plasmin plays the critical role of degrading the fibrin. As such, the lysis of the clot leads to the re-opening of obstructed arteries. However, there is a need for early intervention to achieve the restoration of the supply of oxygen to the patient's myocardium. The implication is that candidates or patients expected to undergo fibrinolytic therapy ought to have had a chest pain onset within 12 hours. However, an increase in the risk of bleeding implies that the patient may be excluded from the aforementioned therapeutic approach. Thus, an alternative inotropic agent (Dobutamine) is used because it poses the capacity to increase the force and strength of contractility. In turn, the inotropic agent plays the role of oxygen supply increase to the patient's coronary arteries, as well as increasing the patient's cardiac input. According to O'Gara, Kushner and Ascheim et al. (2013), dobutamine and other inotropicagents reduce chances of damaging recently infracted areas and are, thus, recommended.

Wenchebach forms a second degree Mobitz Type I or AV block type I. With a progressively lengthening AV conduction time, it comes to a point when the P is no longer conducted. The eventuality is that QRS drops. The dropped QRS is followed by a progressively longer PRI while the former assumes normal width. With the P to P interval reported to be regular, each beat is followed by a shortening R to R. With the first beat witnessing the shortest PRI, the final wave experiences a failed conduction of the group's final P wave. Ultimately, QRS fails to follow the P wave.

In the presented case, any evidence of the presence of a stable clot such as that resulting from CVA, trauma, or a recent surgery implies that administering thrombolytic therapy is not ideal because this procedure causes disruption to the clot. Furthermore, thrombolytic therapy is not advisable for patients with NSTEMI or unstable angina. According to Steg, James and Atar et al. (2012), these conditions are caused by plaque ruptures in which partially occluded thrombi are formed. As such, thrombolytic causes an increase in thrombi through the break up of the clot. The secondary effect is that other vessels may be occluded. However, thrombolytic therapy that is conducted within 12 hours from

acute MI's Q-wave onset has been found to reduce mortality and the infarct in patients (Mosca, Benjamin & Berra et al., 2011). Despite this promising outcome, contraindications have been recommended in situations where acute MI is present. In addition, thrombolytic therapy has been discouraged in patients with an increased risk for severe uncontrolled hypertension, history of stroke, hemorrhagic stroke, those receiving oral anticoagulants, dementia, known bleeding disorders, and active bleeding (Peberdy, Callaway & Neumar et al., 2010). Overall, thrombolytic therapy poses the advantage of high-degree blood flow restoration in situations where infracted arteries are reported. Potential risks of thrombolytic include stroke, bleeding, and allergic reactions (Goff, Lloyd-Jones and Bennett et al., 2014). For the case of H.G., thrombolytic therapy could be administered because the description suggested that he was suffering from acute MI.

With the MI affirmed to target the inferior wall, a PRI = 0.24 suggests an AV Heart Block. In most cases, an AV block is reported in patients experiencing MI of the inferior wall. It can also be inferred that H.G.'s CCU heart rate and blood pressure were low. This inference is informed by the presence of a low cardiac output, a thread and weak pulse, as well as symptomatic hypotension. The description indicates further that H.G. was started on a dobutamine drip with the objective of increasing his heart's contractility. Furthermore, H.G.'s arrival to the emergency room was characterized by experiences of ventricular dysrhythmias that saw him undergo defibrillation twice (300j and 200j) before being started on an Amiodarone drip.

One of the other diagnostic tests that could be conducted involves Serial ECG's. This test is important because it aids in monitoring PRI, St-segment, and changes in conduction systems (O'Gara, Kushner & Ascheim et al., 2013). With Type I heart block perceived to be present, Serial ECG's becomes ideal. Another potential test involves Echocardiogram- TEE or TTE. The role of echocardiograms is to determine the size and thickness of the heart, the ejection fraction, and the level of the heart's function. Furthermore, echocardiograms check for the ischemic disease and clots (Steg, James & Atar et al., 2012; Mosca, Benjamin & Berra et al., 2011). There is also a need to draw arterial blood gases to determine if H.G.'s acute MI has affected his respiration.

Conclusion

Through angioplasty or angiogram tests, H.G. could be exposed to a cardiac catheterization with the aim of visualizing both the recanalization of the artery and the occlusion of his coronary arteries. An additional test entails a chest X-ray to evaluate the potentiality of aortic dissection or heart failure. Similarly, myocardial perfusion scanning could be done to determine the pattern of the flow of blood to the walls of the heart. Lastly, a CT-scan could be administered to determine the extent to which the myocardium may have been destroyed.

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Adult Respiratory Distress Syndrome Intervention

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Abstract

Adult Respiratory Distress Syndrome (ARDS) is a severe lung condition. The condition occurs when the patient's lungs' air sacs are filled with fluid. The number of organs involved and associated illnesses form additional determinants of ARDS mortality rate. For example, cases in which three organs are involved and that the situations last for more than one week have led to invariably fatal outcomes. Notably, an increase in the rate of mortality correlates directly with the state of disease severity. For the survivors, physical, psychologic, and cognitive morbidity is common. Upon discharge from ICUs, effects such as cognitive dysfunction, psychiatric illnesses, short-term compromise of lung function, family stress, poor quality of life, minor imaging abnormalities, and persistent physical disabilities and abnormal exercise endurance may be experienced.

Introduction

Adult Respiratory Distress Syndrome (ARDS) is a severe lung condition. The condition occurs when the patient's lungs' air sacs are filled with fluid. According to Caironi, Cressoni and Chiumello et al. (2010), too much fluid leads to a significant reduction in the amount of oxygen contained in one's bloodstream. As such, it can be inferred that ARDS accounts for the prevention of organs from receiving the oxygen needed to function. The eventuality is that ARDS causes organ failure (Gattinoni, Carlesso & Taccone et al., 2010).

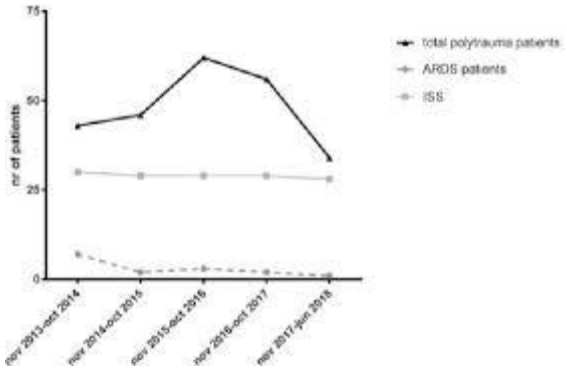
Clinical indications appear between one to three days from the onset of the trauma or injury. ARDS's indicators include mental confusion, a fast pulse rate, fever, dry and hacking coughs, as well as discolored nails or skin. Similarly, conditions such as low blood pressure, general weakness and muscle fatigue, and, labored and rapid breathing suggest the presence of ARDS (Esan, Hess & Raoof et al., 2010).

In patients suffering from ARDS, common risk factors include histories of alcoholism, the chronic lung disease, smoking cigarettes, and age (such as adults over 65 years old). Similarly, the condition has been reported to pose more adverse effects for people who have liver failure and toxic shock (Ware, Koyama & Billheimer et al., 2010). Head or head injuries such as during contact sports and car wrecks, overdosing on tricyclic antidepressants, developing severe lung infections (that include pneumonia), the development of severe blood infections, and inhaling toxic substances that include smoke, chemicals and salt water form addition and common agents leading to ARDS (Sud, Sud & Friedrich et al., 2010). In the case presented, the patient's conditions experienced and forming predictors of ARDS risk factors include receiving 50% 02 via venturi mask and endotracheal tube placement; suggesting that he had already been hospitalized for another condition.

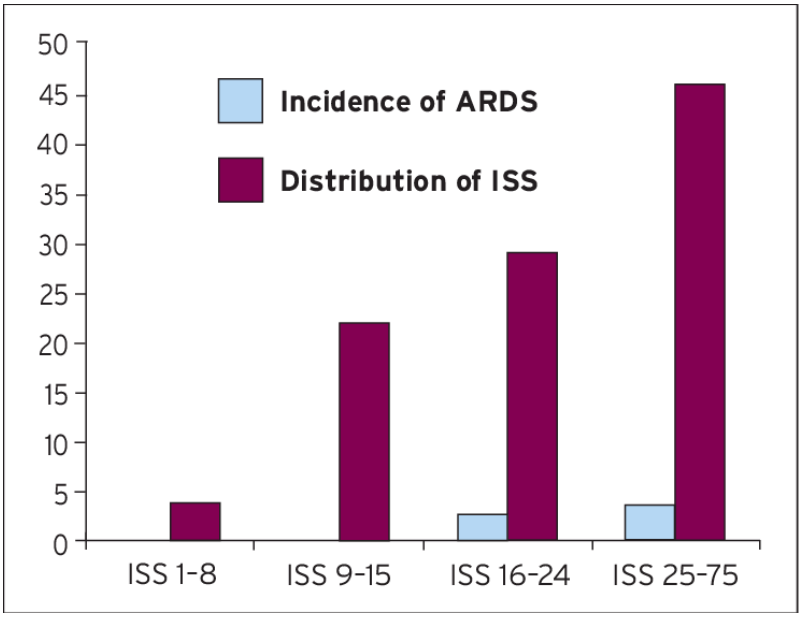
Methods

Lung ventilation/perfusion scans are applied and play the role of measuring the blood and air flow in the lungs. The central drive necessitating this test is the need to rule out sudden blockages in the lung arteries, as well as pulmonary embolism. As such, V:Q refers to a ventilation/perfusion ratio. In patients diagnosed with ARDS, V:Q mismatch is perceived to be that which contributes to increased physiologic dead space. The latter arises due to the inhibition of blood flow to well-ventilated alveoli, with shunt resulting when ARDS causes blood flow's failure to participate in gas exchange (Gattinoni, Carlesso & Taccone et al., 2010).

Results



Regarding pathophysiological alterations, ARDS is associated with an increase in capillary permeability in the condition's hallmarks. Furthermore, the condition causes damage to the alveolar epithelium and capillary endothelium as the process of fluid removal (from the alveolar space) is impaired, causing an accumulation of protein-rich fluids. The eventuality is that diffuse alveolar damage complements pro-inflammatory cytokines. According to Hough, Steinberg and Rubenfeld et al. (2009), ARDS causes inflammation when neutrophil is activated in the condition's pathogens. Overall, ARDS causes widespread and diffuse inflammation of the lung tissue, low level of blood oxygen (hypoxemia), flooded microscopic air sacs of the lungs, and a partial collapse of the lungs. Furthermore, ARDS causes damage to surfactant-producing type II cells while alveolar space's presence of protein-rich fluid causes disruptions to the function and production of pulmonary artery surfactant, leading to impaired gas exchange and microatelectasis. The ultimate effect is that which entails ventilation/perfusion and regional variations in pulmonary perfusions, as well as increased alveolar-arterial gradient and blood shunting through unventilated alveoli (Caironi, Cressoni & Chiumello et al., 2010). Additional effects include alveolar remodeling, differentiation and proliferation of type II alveolar cells, regeneration of epithelial cells, and the resorption of alveolar edema.



In ARDS, the intrapulmonary shunting of blood causes hypoxemia. The shunting results from airspace collapse or filling. With findings documented to include tachypnea and dyspnea, diagnoses are achieved through chest x-rays and ABGs. According to Esan, Hess and Raoof et al. (2010), the treatment procedure for ARDS' hypoxemia involves mechanical ventilation – especially in cases where high-flow O_2 records a saturation falling below 90%.

With lung compliance defined as a measure of the ability of the lungs to expand and stretch, static compliance entails the change in volume upon pressure application. Therefore, the clinical significance of static pressure cannot be overemphasized. For example, static compliance enables care providers to monitor elastic resistance, playing a lung indicative role of plateau pressure. Similarly, static compliance alerts clinicians to exhibit closer patient monitoring or investigate potential problems (Ware, Koyama & Billheimer et al., 2010). In the case presented, a decrease in static compliance suggests that the patient's lungs do not reveal significant changes when pressure is applied. This outcome is evidenced by continued deterioration in the arterial blood gas (ABG).

At the emergency department, the patient's pH was 7.48. Later, the pH rose to 7.50 while undergoing treatment at the ICU. This level can be inferred to be highly alkaline or basic. A decrease in patient's arterial carbon dioxide ($PaCO_2$) partial pressure is likely to have arisen from alveolar hyperventilation. The eventuality is that the decrease leads to the increased state of ratio of bicarbonate concentration to $PaCO_2$. In the end, the pH level is expected to increase; yielding respiratory alkalosis.

In clinical practices, PEEP refers to positive end-expiratory pressure. This technique of ventilation in exhalation is achieved through mechanical impendence when valves are introduced within circuits (Sud, Sud & Friedrich et al., 2010). According to Hough, Steinberg and Rubenfeld et al. (2009), PEEP is likely to prevent the return of intrathoracic pressure to atmospheric pressure during expiration. When levels are deemed sufficient, PEEP could diminish the patient's cardiac output in the respiratory cycle's entirety. With the ability to lower the LV volume and limit venous return, PEEP may improve the cardiac output of the patient by maintaining the lung volume; attributed to the maintained level of alveolar patency.

A study by Caironi, Cressoni and Chiumello et al. (2010) suggested that the primary role of PEEP is to stabilize or recruit lung units while improving oxygenation among patients found to

experience hypoxemic respiratory failure. On the one hand, beneficial effects include minimized potential for lung injuries that are ventilator-induced and improved lung compliance. On the other hand, adverse effects include interference with hemodynamic pressure assessment, decreased cardiac output, worsened gas exchange, and barotraumas (Gattinoni, Carlesso & Taccone et al., 2010). In the case presented, PEEP could redistribute fluid in the alveoli while reducing intrapulmonary shunting. It is also worth noting that PEEP is likely to reduce risks of oxygen toxicity and FIO2 requirements while improving arterial oxygenation (PaO2). By recruiting and stabilizing lung units, PEEP is also projected to prevent alveolar collapse and increase functional residual capacity; improving lung compliance. It is further affirmable that PEEP could shift tidal deflections and prevent repetitive collapse of lung units which, in turn, minimizes potential for injuries that are ventilator-induced.

Given that PEEP decreases venous return and that it accounts for a significant increase in pressure levels of the intraluminal central venous, the right and left ventricular preload is expected to reduce. Furthermore, an increase in the right ventricular afterload is expected to be followed by a decrease in the cardiac output. This decline is attributed to the occurrence of organ hyperfusion and the aspect of hypotension. In addition, the decrease in cardiac output is associated with lower blood pressure in the presence of hypovolemia. Overall, PEEP is likely to reduce the ventricular compliance, decrease the afterload in the left ventricle, and increase the patient's intracracial pressure – due to experiences of increased pressure in the central venous.

Upon admission to the ICU, the primary nursing diagnosis for this patient should be that which involves impaired gas exchange related to decreased lung compliance, interstitial edema and alveolar-capillary permeability – as evidenced by breathing failure at the river, respiration 28/minutes, heart rate 120/minute, and blood pressure 122/80 (accompanied by an audible expiratory wheeze and a bilaterally diminishing breath with crackles).

In ARDS cases, one of the nursing interventions involves ensuring adequate humidification. The role of this procedure is to help in liquefying tenacious secretions. Another intervention involves monitoring the patients' level of mental sluggishness, noting confusion, and levels of consciousness as precautionary measures to predict potential adversities or increasing severity. Lastly, there is a need monitor and record the nature of the patient's response to medication, ideal for determining the effectiveness of ongoing medication and forming a platform from which recommendations for alternative medication could be made.

In patients with ARDS, prognosis outcomes suggest an overall mortality that ranges between 50-75 percent, varying with the individuals' ages and causative agents. For example, causes arising from trauma are rated to account for a mortality rate of 38 percent while ARDS arising from pneumonia has an 86-percent rate of mortality (Esan, Hess & Raoof et al., 2010).

Conclusion

In conclusion, the number of organs involved and associated illnesses form additional determinants of ARDS mortality rate. For example, cases in which three organs are involved and that the situations last for more than one week have led to invariably fatal outcomes. Notably, an increase in the rate of mortality correlates directly with the state of disease severity. For the survivors, physical, psychologic, and cognitive morbidity is common. Upon discharge from ICUs, effects such as cognitive dysfunction, psychiatric illnesses, short-term compromise of lung function, family stress, poor quality of life, minor imaging abnormalities, and persistent physical disabilities and abnormal exercise endurance may be experienced.

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Hypertension Management Effectiveness

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Abstract

Based on the scholarly insights about the prevalence, incidence, and management of hypertension, it is evident that variations exist relative to predictive factors such as age, gender, and racial background. For national and local healthcare systems, a major intervention at their disposal involves community needs assessment and family education. The education needs to target issues such as the causes, signs, symptoms, risk factors, statistics about the incidence and prevalence, and some of the secondary effects of hypertension on population and community or family and individual productivity. By complementing such efforts with diet education, it is predicted that the incidence and prevalence of hypertension will be minimized, eventually restoring the economic productivity of the affected communities.

Introduction

Emerging as one of the leading conditions that account for the increasing incidence of psychological stress in patient populations, hypertension entails abnormally high blood pressure. According to Buford (2016), most of the hypertensive patients' arteries are the zones that experience high blood pressure-related persistent elevations. In a related observation, Freeman, Vinh and Widdop (2017) stated that several complications arise from the presence of hypertension and. Some of these complications include heart disease, stroke, and the risk of death. As such, it can be inferred that hypertension is a risky condition due to its associated comorbidities that translate into increased mortality rates and reduced life expectancy. The central purpose of this research paper is to examine the prevalence and incidence of hypertension around the world, as well as some of the practices responsible for managing the condition, hence improved health care outcomes. The motivation is to gain insight into some of the various scholarly affirmations that have investigated the selected subject.

Methods

Several studies have pointed out the criticality of embracing or implementing strategies aimed at managing hypertension. For instance, the investigation by Mueller, Purnell, Mensah and Cooper (2015) indicated that effective management approaches are those that emphasize coordinated healthcare interventions and communications at the population level. In another study by Shamsi, Dehghan and Esmaeili (2017), the main objective was to unearth the prevalence of hypertension in relation to the racial backgrounds of the affected patients, families, or communities. Similar to the affirmations by Singh, Shankar and Singh (2017), the study established that in a developed region such as the U.S., ethnic minority community members (such as African-Americans) are more likely to experience hypertension. In reference to such insights, Solomon, Seixas and Ogedegbe et al. (2015) asserted that the statistical outcomes are important to national healthcare systems because they inform the extent to which treatment and control efforts continue to lag in such communities, pointing further to the need to consider socio-cultural background as a factor that plays a predictive role in the incidence

and prevalence of hypertension, upon which disease management programs could be tailored to the needs of such communities (or designed in a manner that proves responsive to social, cultural, and economic needs of the affected populations). This secondary study employs a content analysis technique to gain critical insight into the selected medical subject of hypertension, as well as how several moderating factors shape the effectiveness of disease intervention.

Results

Scholarly investigations have focused on some of the factors accounting for disparities in the incidence and prevalence of hypertension among the affected communities. In one of such studies, Whelton, Einhorn and Muntner et al. (2016) found that some of the forces responsible for the disparities include physiological, cultural, and socio-economic differences, with the worst-hit communities being those with resource constraints. From a statistical perspective, which qualifies the existence of race-based disparities in the prevalence and incidence of hypertension, Yoon, Carroll and Fryar (2015) stated that in African-Americans, the average percentage incidence of hypertension is 39.1%, proving high compared to other ethnic groups; including the case of non-Hispanic whites. In a related examination, Buford (2016) found that the modes of implementing the awareness and treatment programs operate similarly when groups such as African-Americans and non-Hispanic whites are compared but efforts seeking to control hypertension prove lower in the African-American community. Specifically, the statistical outcomes reported by Freeman, Vinh and Widdop (2017) suggested that the average percentage of control among African-Americans stands at about 28.9% while the case of the whites reveals a rate of control of about 35.4%. Based on these scholarly outcomes, it is worth inferring that the prevalence of hypertension in most of the ethnic minority communities accrues from lower rates of disease control; with the case of hypertension unexceptional. A question that arises is what are some of the feasible approaches through which hypertension might be managed, hence reduced incidence?

Figure 1. Prevalence of hypertension among adults aged 18 and over, by sex and age: United States, 2015–2016

[1]Men significantly different from women in the same age group.
[2]Significant increasing trend by age.
NOTES: Estimates for age group 18 and over are age adjusted by the direct method to the 2000 U.S. Census population using age groups 18–39, 40–59, and 60 and over. Crude estimates for age group 18 and over are 32.1%, total; 31.8%, men; and 32.4%, women. Access data table for Figure 1 at: https://www.cdc.gov/nchs/data/databriefs/db289_table.pdf#1.
SOURCE: NCHS, National Health and Nutrition Examination Survey, 2015-2016.

Relative to the mortality rate accruing from hypertension and its associated comorbidities, most of the recent literature points to similar trends as the case of the rate of disease control; with disparities reported from one community to another. According to Yoon, Carroll and Fryar (2015), most of the ethnic minority communities experience a higher

prevalence of hypertension. With the results concurring with those that had been reported by Mueller, Purnell, Mensah and Cooper (2015), the study indicated that in a group such as the African-American community, the incidence of hypertension is not only high but also reveals gender-based disparities. In particular, it was affirmed that the mortality rate in men stands at an average of 66.4% compared to their female counterparts, who were associated with an average mortality rate of 40.3%. Also, the study by Shamsi, Dehghan and Esmaeili (2017) indicated that the mortality rate for hypertension stretches beyond aspects of racial background and the parameter of gender to vary relative to the age of the affected population. In particular, the latter investigation highlighted that among the whites, the rate of hypertension and pre-hypertension as about 17.5%, proving lower than children from an ethnic minority community such as the African-American group, whose rate of hypertension and pre-hypertension among children was documented to stand at about 24.5%. As avowed by Singh, Shankar and Singh (2017), these trends are worrying and call for greater attention and early intervention by relevant healthcare authorities; especially those that seek to curb disparities in access to health care services.

Regarding some of the studies that have investigated the major factors responsible for hypertension prevalence and the rates of disease treatment and control, mixed outcomes have been reported. For example, Solomon, Seixas and Ogedegbe et al. (2015) stated that some of the predictive forces responsible for the perceived (and racial-based) disparities in the treatment and control of hypertension include variations in the affected individuals' physiological and hemodynamic determinants. In particular, the studies highlighted that most of the members belonging to ethnic minority communities (such as African-Americans) have a high prevalence of obesity (at an average of about 51%), yet this condition (obesity) is a predictor of hypertension. Whelton, Einhorn and Muntner et al. (2016) stated further that most of the ethnic minority community members are likely to experience a high prevalence of hypertension due to higher rates of sodium retention, hence a higher incidence of hypertension and pre-hypertension in the community.

Salt sensitivity has been observed further to predict the higher incidence and prevalence of hypertension and pre-hypertension in most of the ethnic minority communities. As documented by Yoon, Carroll and Fryar (2015), salt sensitivity is about 50% in communities such as African-Americans, arising from high-salt diets, a factor accounting for a high incidence of hypertension in this group. Overall, it can be inferred that socio-economic constraints and inadequate access to quality health care exacerbate the burden of hypertension and pre-hypertension, coming at a time when most of the ethnic minority communities have low treatment and control rates.

Conclusion

Based on the scholarly insights about the prevalence, incidence, and management of hypertension, it is evident that variations exist relative to predictive factors such as age, gender, and racial background. For national and local healthcare systems, a major intervention at their disposal involves community needs assessment and family education. The education needs to target issues such as the causes, signs, symptoms, risk factors, statistics about the incidence and prevalence, and some of the secondary effects of hypertension on population and community or family and individual productivity. By complementing such efforts with diet education, it is predicted that the incidence and prevalence of hypertension will be minimized, eventually restoring the economic productivity of the affected communities.

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