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Obesity And Disease Severity In Iraqi Patients With Covid-19

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Abstract

Recently, many studies have shown an increasing global interest for COVID-19 pandemic, which has almost invaded the earth. However, the obesity is considered one of the risk factors for the exacerbation of the severity of respiratory diseases. However, it is unknown whether obese patients are also more prone to have a more severe COVID-19 condition. Accordingly, this study was aimed to find the link between obesity and illness severity for Iraqi patients infected with COVID-19. A total of 167 covid-19 positive patients were examined for their obtaining the virus via undergoing viral RNA detection using GeneXpert technique. For each of the study's groups, body max index (BMI) was calculated. In severe patients, the BMI level was significantly higher (P = 0.05) than in other patient categories. Conclusion: Covid-19 infection is most sever among obese patients.

Keyword: Covid-19, BMI, Obesity.

Introduction

The global epidemic of Corona virus is triggered by SARS-CoV-2. Malnutrition represented by lack and over nutrition has been related to a worse prognosis of viral illness since the 1918 "Spanish" influenza epidemic (Short et al., 2018). Obesity and diabetes were linked to increased mortality and a longer duration of illness in 1957-1960 and 1968 for Asian and Hong Kong influenzas respectively, even if there are no underlying chronic conditions that increase the risk of complications from influenza (Yang et al., 2013 and Moser et al., 2019). In 2009 during influenza A virus pandemic (H1N1), the obesity was related as a factor which increased the hospitalization, risk severity of disease and death (Morgan et al., 2010 and Honce et al., 2020). About half of the world's population suffers from an increase in obesity in developed countries, accompanied by an increase and a rise in the frequency of infection after each year. According to the criteria of the World Health Organization, about 34% of Americans individuals are overweight according to the most recent NHANES study (Caspard et al., 2018). About 160 to 210 million Americans were developed this illness during the year according to the CDC model. Moreover, according to CDC data which received from hospital inpatient capacity and mortality the number of deaths reached about 200,000 to 1.7 million (Ritchie et al., 2020). In this paper, we will discuss various theories and evidence about the role of obesity's high prevalence in the spread and perpetuation of COVID-19 epidemics.

Methods

Samples collection

From September to December 2020, 167 patients diagnosed with covid-19 with ages ranging from 20 to 71 years old. The clinical samples were distributed to 96 male and 71 female who attended to Imam Ali Hospital in Baghdad Governorate. Clinical signs such as fever, coughing, sneezing, productive sputum, chest tightness, sore throat, muscle soreness, and oxygen saturation drop were used to determine the severity of the condition. The universal formula was used to calculate the BMI for each patient.

Criteria of samples collection under study

1- All patients were asked a set of questions which include general information like family history for hereditary and chronic diseases beside covid-19 infections, height and weight, sex, age and any medical therapy were administered. Clinical symptoms such as fever, coughing, sneezing, and shortness of breath and oxygen saturation were recorded for each patient. Based on severity of clinical symptoms, the current of clinical samples were divided into three subgroups asymptomatic, mild and sever. The first category was divided into asymptomatic, the second category was represented as a mild symptoms like loss of smell or taste, the third category was classified as a severe explicit respiratory symptoms.
2- Nasal swabs were collected from all patients under study and transferred into viral transport media until used for molecular detection via GeneXpert laboratory method. The sample was added into reagent and then placed this mixed into GeneXpert machine, from this point all processes was automated and covid-19 infection was detected.
3- Divide an individual's weight in kilograms by the square of their height in meters (kg/m2) to calculate BMI. Subjects were separated into four categories based on their BMI levels: underweight (UW) (18-22 kg/m2), normal weight (NW) (23-26 kg/m2), overweight (OW) (27-30), and obese (> 30 kg/m2) (Andersen et al., 2016).

Exclusion criteria

Pregnant women, patients under the age of 19, smokers, and patients with additional disorders in addition to covid-19 were all excluded.

Molecular detection of viral infection

Molecular detection of viral RNA utilizing the GeneXpert technology was used to diagnose Covid-19 infection.

Statistical analysis

The data were examined by using the IBM SPSS version 25.0. The probability also examined by using Chi square. On the other hand, qualitative data were expressed as frequency and percentage.

Results

The results as shown in Table 1 illustrated highly significant differences between severity of covid-19 and their obesity as a risk factor for infection.

Table 1. The correlation between the patients with severity of Covid-19 illness and their obesity.

Covid-19 patients		BMI classification [No (%)]				
		UW	NW	OW	Obese	Total
Severity classification [No (%)]	Asymptomatic	2 (1.2%)	2(1.2%)	8 (4.8%)	8 (4.8%)	20
	Mild	5 (2.9%)	8 (4.5%)	20 (11.97%)	36 (21.6%)	69
	Sever	13 (7.78%)	11 (6.6%)	9 (5.4%)	45 (27%)	78
	Total	20	21	38	89	167
Statistical test					Probability	
Chi-Square test					P = 0.01	

(No: number),(*: significantly different), (NW: normal-weight), (OW: over-weight), (BMI: body mass index), (UW: under-weight).

Discussion

Obesity changes numerous stages of the innate and adaptive immune responses, resulting in a low-grade inflammatory disease. This route plays a role in systemic metabolic dysregulation, which is linked to obesity-related diseases (Ouchi et al., 2011). Obesity may be linked to an increase in the severity of the disease because patients have higher levels of pro-inflammatory cytokines like monocyte chemoattractant protein-1 (MCP-1), alpha-TNF-alpha and IL-6 in the basal state, which is often produced through visceral and subcutaneous adipose tissue which lead to deficiency of innate immunity (Ahn et al., 2015). Chronic inflammation associated with increased obesity causes reduced activation of macrophages and pro-inflammatory cytokines when an antigen was presented (Karlsson et al., 2016). On the other hand, the poor vaccination success of obese patients is explained by lower macrophage activation once an antigen is given (Xue et al., 2017); the rise of vaccine escape variations and antiviral-resistant in the obese individuals are explained by this distinct obesogenic milieu (Zhang et al., 2013). The incidence of severe Covid-19 infection can be increased by obesity and has been estimated to be approximately sixfold in infected persons with metabolic-associated fatty liver disease, according to a recent study from China (Zhang et al., 2013).Moreover, obesity is a strong predictor of severe COVID-19 in children and adolescents (Zhang et al., 2013).

COVID-19 may also increase the risk of infection, hospitalization, clinically serious disease, mechanical ventilation, and mortality in obese individuals (Cai et al., 2021). COVID-19 and its complications are more likely in people who are overweight or obese, therefore health-care providers should be aware of this (Louie et al., 2009).

In obese and obese-diabetic patients, the responses of T and B-cells are also compromised, resulting in greater vulnerability and a delay in the clearance of viral infection. Leptin plays an important role as an essential regulator of B-cell maturation, development and function. Similarly, (Zhang et al. 2013) suggested that leptin resistance is a cofactor for pandemic influenza (H1N1) in 2009 (Park et al., 2018). Obese patients may also have changes in the number and function of lymphocytes, which might affect memory T cell responses and vaccination efficacy (Honce et al., 2019). Obesity inhibits T-cell response to viruses and antibody response to seasonal influenza vaccination, and perhaps underperformance of macrophage cells in terms of their inappropriate maturation, which plays a role in poor response to

vaccine (O'Brien et al., 2012). The severe lung lesions seen in victims of the influenza pandemic are due to a dysregulated pro-inflammatory response. In order to guard against new influenza virus strains and subtypes, a strong CD8 + T cell response is required. Several studies in OB mice with IAV infection have found increased illness severity, secondary bacterial infections, and vaccination effectiveness (Klinkhammer et al., 2018). Physical inactivity is another major issue among obese people. Obese patients, in comparison to thin individuals, are more sedentary or engage in less physical activity. Physical activity is reduced in several stages of the immune response such as suppression of inflammatory cytokines (proinflammatory) and activation of macrophages (O'Brien et al., 2012). Besides, adipose tissue was also associated with an increase in the severity of infection, as the adipocytes secrete many factors represented by hormones such as leptin and adiponectin, which affect many organs, including the lungs (Zheng et al., 2015 and Reidy et al., 2019). Whereas, Leptin, as a pro-inflammatory cytokine, can alter both innate and adaptive immunological responses by promoting the production of interleukin 2 (IL-2) and tumor necrosis factor-alpha (TNF-α) while decreasing the secretion of IL-4 and IL-5 (Wong et al., 2019). Adiponectin, on the other hand, is an anti-inflammatory adipokine that inhibits (TNF-α, IL-6, and nuclear factor kappa) and promotes (IL-1 and IL-10 receptor antagonists) (Wong et al., 2019). Obesity cause increase levels concentrations of leptin on the contrary, obesity causes a decrease in adiponectin concentrations (Fuster et al., 2016 and Wong et al., 2019).

Conclusion

Obesity plays a significant influence in the severity of the disease and its close association, according to the study, and adipose tissue raises the rate of lectin formation, which stimulates inflammatory cytokine production.

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