The Effect of Acute Bronchitis on Blood Pressure

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Abstract

Background: Acute bronchitis is one of the top 10 conditions for which patients seek medical care. Acute bronchitis is a clinical term implying a self-limited inflammation of the large airways of the lung. A possible link between inflammation and elevated blood pressure has been suggested by several cross-sectional and longitudinal studies.

Objective: To assess the effect of acute bronchitis on the blood pressure.

Patients and Methods: The cross sectional study was carried out in in Baghdad. 100 patients with acute bronchitis, their age group of 17-76 years were included in the study. Blood pressures (systolic and diastolic) for the patients were taken in sitting position on the right arm during the attack and after improvement (five to seven days later).

Results: 100 patients with acute bronchitis were enrolled in the study. Mean age was 45.51±15.46. Female were 54 (54%), and 46 (46%) were male. There was 42 (42%) have history of hypertension. Only 15 patients (15%) have history of smoking. Mean arterial pressure (MAP) during attack was normal in 58 (58%), and high in 42 (42%). MAP after improvement was normal in 84 (84%), and 16 (16%) remain high. About clinical features, 100% have cough, 71% sputum, 36% dyspnea, and 6% hemoptysis. Systolic, diastolic blood pressure, and mean arterial pressure during the attack were higher than after improvement with highly statistically significance (P value < 0.001). There was no statistically significant effect for the history of hypertension and smoking on blood pressure during the attack and after improvement (p value 0.441, 0.309 respectively).

Conclusion: Systolic, diastolic blood pressure, and mean arterial pressure during the attack of acute bronchitis were higher than after improvement.

Key words: *Acute bronchitis, systolic blood pressure, diastolic blood pressure, mean arterial pressure.*

Introduction

Acute bronchitis is one of the most common diseases. [1] It affect approximately 5% of adults per year, and although they occur throughout the year, the incidence is higher in the autumn and winter [2]. The usual causes of acute bronchitis are viral infections of the upper airways including influenza A and B, parainfluenza, coronavirus (types 1-3), rhinovirus, respiratory syncytial virus, and human metapneumovirus [3]. However, reports indicate that more than 60 to 90 percent of patients with acute bronchitis who seek care are given antibiotics [9, 10].

Cough constitutes the most prominent manifestation of acute bronchitis[6]. It is a self-limiting upper respiratory infection that takes about two to three weeks to resolve, however, a cough can last to a maximum of 6 weeks [7]. Initially, the cough is non-productive, but after about a week there is an increase in mucus production, and in the second week, the colour of the sputum often changes from grey-white to purulent. Despite being a self-limiting condition, most patients with acute bronchitis seek medical advice, mainly because of bothersome cough [8].

Acute bronchitis is thought to reflect an inflammatory response to infections of the epithelium of the bronchi.

Epithelial-cell desquamation and denuding of the airway to the level of the basement membrane in association with the presence of a lymphocytic cellular infiltrate have been demonstrated after influenza a tracheobronchitis. [9]

In patients with acute cough and sputum production suggestive of acute bronchitis, the absence of the following findings reduces the likelihood of pneumonia sufficiently to eliminate the need for a chest radiograph: 1- heart rate > 100 beats/min; 2- respiratory rate > 24 breaths/min; 3- oral body temperature of > 38°C; and 4- chest examination findings of focal consolidation, egophony, or fremitus [10].

High blood pressure is a major public health epidemic in the United States. There are 76.4 million adult Americans with hypertension as defined by a blood pressure "140/90 mm Hg or greater [11]. The WHO's 2016 Global Health Observatory (GHO) data estimated that high blood pressure would cause 7.5 million deaths, about 12.8% of the total of all deaths [12].

Within Europe, high blood pressure is particularly an issue as it has been shown to have an increased prevalence of 60% when compared with the U.S. and Canada – two prominent non-European developed countries [13]. Angiotensin converting enzyme (ACE) is an endogenous regulator of blood pressure that is highly expressed in the lung and has been linked to risk for acute respiratory distress syndrome (ARDS) [14].

The mean arterial pressure (MAP) is a term used to describe an average blood pressure in an individual. It is defined as the average arterial pressure during a single cardiac cycle [15]. MAP may be used similarly to Systolic blood pressure in monitoring and treating for target blood pressure. Both have been shown advantageous targets for sepsis, trauma, stroke, intracranial bleed, and hypertensive emergencies [16].

Patients and Methods

Setting and study design:

The cross sectional study was carried out in in Baghdad. 100 patients with acute bronchitis (history of cough for two weeks or less with or without sputum, dyspnea, hemoptysis), their age group of 17-76 years were included in the study. From May 2018 to May 2019.

Inclusion criteria: 1. Adult patients 2. With clinical features of acute bronchitis

Exclusion criteria: 1. Patients complaint of fever as it can cause tachycardia which may increase blood pressure. 2. Heart rate > 100 beats/min; 3. Respiratory rate > 24 breaths/min; 4. Chest examination findings of focal consolidation, because not all patients did chest x ray [10], and 5. Patients who did not come for fellow up.

Definitions of the outcomes:

Self-administered questionnaires on age, respiratory symptoms (cough, sputum, dyspnea, and hemoptysis) were performed. History of hypertension, smoking were assessed by self- report for all patients.

Blood pressures (systolic and diastolic) for the patients were taken in sitting position on the right arm, by Mercury Sphygmomanometer.

The patients had two visits, during the attack and after improvement (five to seven days later). With each visit asked about respiratory symptoms, and blood pressure measured.

MAP can be estimated from measurements of the systolic pressure P sys and the diastolic pressure P dias. MAP =P dias + 0.33(P sys – P dias) [18]

Statistical Analysis

The statistical analysis of this prospective study performed with the statistical package for social sciences (SPSS) 21.0 and Microsoft Excel 2013. Categorical data formulated as count and percentage. Chi-square test describe the association of these data. Numerical data with normal distribution were described as mean and standard deviation, independent sample t-test used in comparison between two groups. The lower level of accepted statistical significant difference is bellow or equal to 0.05.

Results

100 patients with acute bronchitis were enrolled in the study. Mean age was 45.51±15.46. Female patients were 54 (54%), and 46 (46%) were male. There was 42 patients (42%) have history of hypertension, while 58 (58%) have not. 15 patients (15%) have history of smoking, while 85 (85%) have not. Mean arterial pressure (MAP) during attack was normal in 58 (58%), and high in 42 (42%). MAP after improvement was normal in 84 (84%), and high in 16 (16%). As in table 1.

Table 1: Characteristics of patients

		Bronchitis patients N=100
Age (years)		45.51±15.46
Gender type	Female	54 (54%)
	Male	46 (46%)
Hypertension	Yes	42(42%)
	No	58 (58%)
Smoking	Yes	15 (15%)
	No	85 (85%)
MAP during attack	Normal	58 (58%)
	High	42 (42%)
MAP after improvement	Normal	84 (84%)
	High	16 (16%)

About clinical features of patients, all patients were have history of cough, 71% have sputum, 36% have dyspnea, and 6% have hemoptysis. As in table 2.

Table 2: Clinical features of patients.

		Bronchitis patients N=100
Cough	Yes	100 (100%)
	No	0 (0%)
Sputum	Yes	71 (71%)
	No	29 (29%)
Dyspnea	Yes	36 (36%)
	No	64 (64%)
Hemoptysis	Yes	6 (6%)
	No	94 (94%)

For the relation between disease activity and blood pressure, the systolic blood pressure, diastolic blood pressure, and mean arterial pressure during the attack were higher than after improvement with highly statistically significance (P value < 0.001). As in table 3.

Table 3: Relation between disease activity and blood pressure.

	during attack	after improvement	P value
SBP	151.15±20.30	137.65±22.20	<0.001*
DBP	86.50±9.99	80.20±12.47	<0.001*
MAP	108.05±12.07	99.35±13.75	<0.001*

*P value < 0.001 is highly statistically significant

The mean arterial pressure outcome after improvement showed that 55 patient were normal mean arterial pressure during the attack and only 3 patients (5.2%) were higher than after improvement. 29 patient were high mean arterial pressure during the attack and only 13 patients (31%) were normal after improvement, with highly statistically significance (P value < 0.001). As in table 4.

Table 4: Mean arterial pressure (MAP) outcome after improvement.

MAP		During attack		Total
		Normal	High	
		Count	Count	
After improvement	Normal	55	29	84
	%	94.8%	69.0%	84%
	High	3	13	16
	%	5.2%	31.0%	16%
	Total	58	42	100
p value		0.001**		

*P value < 0.001 is highly statistically significant

The effect of history of hypertension and smoking on blood pressure outcome, showed no statistically significant effect for the history of hypertension and smoking on blood pressure during the attack and after improvement (p value 0.441, 0.309 respectively). As in table 5

Table 5: Relation of history of hypertension and smoking on blood pressure outcome.

‘			Outcome		Total	p value
			Improved	Not improved		
Hypertension	Yes	Count	13	29	42	0.441
		%	31.0%	69.0%	100.0%	
	No	Count	16	42	58	
		%	27.6%	72.4%	100.0%	

Smoking	Yes	Count	3	12	15	0.309
		%	20.0%	80.0%	100.0%	
	No	Count	26	59	85	
		%	30.6%	69.4%	100.0%	
Total		Count	29	71	100	
		%	29.0%	71.0%	100.0%	

Discussion

In this study patients with history of hypertension was 42%, this was higher than that of Cathleen study [19] in which the overall age-adjusted prevalence of hypertension among persons aged ≥18 years was 29.6%, however in the later study there is race /ethnicity difference which showed that Hispanic blacks had a higher rate of hypertension (41.3%) than non-Hispanic whites (28.6%), so prevalence of hypertension may be different from country to other depend on factors like race, and our study took small number if compared that study.

The history of smoking in this study was 15%, while according to the WHO 2015 [20] data, there was 21.5 % in Iran, 19.9% in United Kingdom, and in United States of America 19.5%, but no data available from Iraq. However again the number in our study is small to detect prevalence.

In this study all patients were have history of cough, 71% have sputum, 36% have dyspnea, and only 6% have hemoptysis. In Boldy study [21] the cardinal symptom was the acute onset of cough (100%), usually productive (90%), wheezing was noted by 62% of patients. Transient bronchial hyper responsiveness appears to be the predominant mechanism of the bothersome cough of acute bronchitis [22]. Acute purulent bronchitis is characterized by infection of the bronchial tree with resultant bronchial edema and mucus formation. Because of these changes, patients develop a productive cough and signs of bronchial obstruction, such as wheezing or dyspnea [23].

In this study the systolic blood pressure, diastolic blood pressure, and mean arterial pressure during the attack of acute bronchitis were higher than after improvement with highly statistically significance (P value < 0.001). This may be explained by fact that inflammation of the bronchial epithelium in acute bronchitis, this inflammation causes the bronchial and tracheal mucosa to thicken as well as epithelial-cell desquamation and denuding of the basement membrane airway [9, 24]. Inflammatory processes are important participants in the pathophysiology of hypertension and cardiovascular disease [25]. Tissue expression and plasma concentrations of inflammatory markers and mediators are increased in patients with cardiovascular disease, those molecules include CRP [26]. Furthermore, the CRP level is positively associated with systolic blood pressure, pulse pressure, and hypertension [27]. Other inflammatory mediators are increased in patients with essential hypertension and in experimental models of hypertension include, interleukin (IL)-6,IL-1 [28], tumor necrosis factor-a(TNF-a) [29], monocyte chemoattractantprotein-1(MCP- 1), intercellular adhesion molecule-1(ICAM-1) and vascular cell adhesion molecule-1(VCAM-1) (26), and have been linked to the activation of the nuclear factor kappa B (NF-kB) system [30] . So in both acute bronchitis and hypertension inflammation play important role in the pathogenesis.

There is a strong association between smoking and cardiovascular disease, acute tobacco consumption is only associated with a temporary rise in blood pressure per cigarette consumed – a rise which subsides after 30 minutes. [31]. However, chronic tobacco consumption causes arterial stiffness that can persist for years after

smoking cessation [32]. Further, heavy consumers have increased incidence of hypertension [33]. In this study there no statistically significant effect for the history of hypertension and smoking on blood pressure during the attack and after improvement, this indicate that the effect of acute bronchitis on blood pressure is independent from history of hypertension and smoking.

Conclusion

During the attack of acute bronchitis blood pressure (systolic, diastolic blood pressure, and mean arterial pressure) may be elevated, and may decreased after improvement of the bronchitis without need for antihypertensive drugs. So just need fellow up the blood pressure during the attack and after improvement.

Conflict of Interest: No

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Ethical Clearance: Not Required

References

1. Tackett, KL; Atkins, A. "Evidence-based acute bronchitis therapy.". Journal of pharmacy practice. (December 2012)25 (6): 586–90.
2. Woodhead M, Blasi F, Ewig S, et al. Guidelines for the management of adult lower respiratory tract infections--full version. Clin Microbiol Infect 2011;17(Suppl.6):E1–59.
3. Falsey AR, Erdman D, Anderson LJ, Walsh EE. Human metapneumovirus infections in young and elderly adults. J Infect Dis 2003; 187:785.
4- Evertsen J, Baumgardner DJ, Regnery A, Banerjee I. Diagnosis and management of pneumonia and bronchitis in outpatient primary care practices. Prim Care Respir J 2010; 19:237.
5- Kroening-Roche JC, Soroudi A, Castillo EM, Vilke GM. Antibiotic and bronchodilator prescribing for acute bronchitis in the emergency department. J Emerg Med 2012; 43:221.
6. Tackett KL, Atkins A. Evidence-based acute bronchitis therapy. J Pharm Pract 2012;25:586–90.
7- Harris A, Hicks L, Qaseem A. Appropriate Antibiotic Use for Acute Respiratory Tract Infection in Adults: Advice for High-Value Care From the American College of Physicians and the Centers for Disease Control and Prevention. Ann Intern Med . 2016 Mar 15;164(6):425-34.
8- Chalmers JD, Hill AT. Investigation of "non-responding" presumed lower respiratory tract infection in primary care. BMJ 2011;343: 5840.
9- Walsh JJ, Dietlein LF, Low FN, Burch GE, Mogabgab WJ. Broncho tracheal response in human influenza. Type A, Asian strain, as studied by light and electron microscopic examination of bronchoscopic biopsies. Arch Intern Med. 1961; 108: 376 -388.
10- Braman SS. Chronic cough due to acute bronchitis: ACCP evidence-based clinical practice guidelines. Chest. 2006; 129(suppl):95S–103S
11- Roger VL, Go AS, Lloyd-Jones DM, et al. Heart and stroke statistics – 2012 update: A report from the American Heart Association. Circulation 2012;125:e12-e230.
12- Word Health Organisation. (2016). Global Health Observatory (GHO) data: Raised blood pressure. http://www.who.int/gho/ncd/risk_factors/blood_pressure_prevalence_text /en /. Accessed 24 Feb 2016.
13- Wolf-Maier K, Cooper R, Banegas J, Giampaoli S, Hense H, Joffres M, et al. Hypertension prevalence and blood pressure levels in 6 European countries, Canada, and the United States. JAMA. 2003; 289(18): p. 2363-9.
14- Evangelou E, Warren HR, Mosen-Ansorena D, et al. Genetic analysis of over 1 million people identifies 535 new loci associated with blood pressure traits. Nat Genet 2018;50 (10) :1412-25.
15- Zheng L, Sun Z, Li J, et al.. "Pulse pressure and mean arterial pressure in relation to ischemic stroke among patients with uncontrolled hypertension in rural areas of China". Stroke. (July 2008) 39 (7): 1932–7.
16- Magder SA "The highs and lows of blood pressure: toward meaningful clinical targets in patients with

shock.". Crit Care Med. (2014). 42 (5): 1241–51.

18- Klabunde, RE (2007). "Cardiovascular Physiology Concepts – Mean Arterial Pressure". Retrieved 2008-09-29. Archived version 2009-10-03.

19- Cathleen D., Kimberly A. Prevalence of Hypertension and Controlled Hypertension — United States, 2007–2010. Centers for Disease Control and Prevention (CDC). Supplements November 22, 2013 / 62(03); 144-148

20- WHO Prevalence of tobacco smoking: Age-standardized prevalence of current tobacco smoking among persons aged 15 years and older (%), 2015.

21- Boldy DA; Skidmore SJ; Ayres JG. Acute bronchitis in the community: clinical features, infective factors, changes in pulmonary function and bronchial reactivity to histamine. Respir Med. 1990; 84(5):377-85.

22- Ralph G., Merle A.Uncomplicated Acute Bronchitis. Ann Intern Med. 2000; 133 (12):981-991.

23- Perlman PE, Ginn DR. Respiratory infections in ambulatory adults. Choosing the best treatment. Postgrad Med. 1990;87(1):175–84

24- Wenzel R, Fowler A, 3rd (16 November 2006). "Clinical practice. Acute bronchitis.". NEJM. 355 (20): 2125; 187: 785.

25- Carmine S, Ernesto L. Inflammation in hypertension. Current Opinion in Nephrology and Hypertension 2006, 15:152–158

26- Blake GJ, Ridker PM. Novel clinical markers of vascular wall inflammation. CircRes2001; 89:763–771.

27- Engstronm G, Janzon L, Berglund G, et al. Blood pressure increase and incidence of hypertension in relation to inflammation-sensitive plasma proteins. ArteriosclerThrombVascBiol2002;22: 2054–2058.

28- Biasucci LM , Liuzzo G,Fantuzzi G. et al. Increasing levels of interleukin(IL)-1 Raand IL-6 during the first 2 days of hospitalization in unstable angina are associated with increased riskofin-hospitalcoronaryevents.Circulation1999;99: 2079–2084

29- Ballantyne CM, Entman ML . Soluble adhesion molecules and the search for biomarkers for atherosclerosis. Circulation 2002; 106:766–767.

30- Ruiz-Ortega M, Ruperez M, Lorenzo O. et al. Angiotensin II regulates the synthesis of pro nflammatory cytokines and chemokines in the kidney. Kidney International 2002; 62: S12–S22.

31- Groppelli A, Giorgi D, Omboni S, Parati G, Mancia G. Persistent blood pressure increase induced by heavy smoking. J Hypertens. 1992; 10(5): p. 495.

32- Jatoi N, Jerrard-Dunne P, Feely J, Mahmud A. Impact of smoking and smoking cessation on arterial stiffness and aortic wave reflection in hypertension. Hypertension. 2007; 49(5): p. 981.

33- Bowman T, Gaziano J, Burning J, Sesso H. A prospective study of cigarette smoking and risk of incident hypertension in women. J Am Coll Cardiol. 2007; 50(21): p. 2085.