The airflow obstruction generally is progressive, may be accompanied by airway hyper-reactivity, and may be partially reversible [1]. Pathological changes in COPD occur in the large (central) airways, the small (peripheral) bronchioles, and the lung parenchyma [2].

Although primarily affecting the lungs, the chronic inflammatory process of COPD does have systemic effects [3]. There is increasing evidence that COPD is a multiorgan system disease. Skeletal muscle weakness and wasting and impaired exercise tolerance which are frequently occurring symptoms in advanced COPD appear to be linked to a systemic inflammatory response [4]. Several systemic inflammatory mediators such as TNF-**α** [5], the soluble TNF transmembrane receptor-75 (sTNF-R75) [6], soluble adhesion molecules [7], some interleukins, acute phase proteins (CRP, fibrinogen, lipopolysacceride-binding protein (LBP)) and leucocytes are increased in the systemic circulation of patients with COPD [4]. One of the markers of systemic inflammation that is consistently shown to be slightly increased in patients with COPD compared with healthy controls is CRP [8]. Exacerbation is a prominent feature of the natural history of COPD. Exacerbations are commonly considered to be episodes of increased dyspnea and cough and change in the amount and character of sputum. Exacerbations are more frequent as disease progresses and are most often triggered by respiratory infections, often with a bacterial component [9].

Studies have shown that an elevated CRP level is a useful indicator of exacerbation in cystic fibrosis, chronic bronchitis, and COPD [10-12].

Methods

Patients

Fifty Patients (age 65±6 years) with COPD on the basis of clinical history and pulmonary function test were enrolled into the study, were admitted to Al-Kadhimya Teaching Hospital because of clinical exacerbations of their condition with dyspnea and increased cough with expectoration of yellow-green sputum. All of them had baseline FEV1 of (0.7±0.2). Pneumonia was excluded by chest radiograph and clinical examinations.

Blood samples

Blood was withdrawn and serum was collected for measurement of CRP and full blood count together with sputum sample for microbiological examination (especially culture). CRP measured by using of Wellcotest Latex agglutination test (*Wellcome Diagnostics*) which is semi-quantitative method, the cutoff point of this test is ≤ 0.6 mg/dL, so all positive values were ≥ 1.2 mg/L.

Treatment and Follow up

All these patients with exacerbations were treated with antibiotics, bronchodilators, controlled low tension oxygen therapy, low dose diuretics for those associated with right sided heart failure with short course of steroids. Then blood sample for CRP and full blood count (Blood samples were collected for all patients before and after treatment. FEV1 is also repeated after 4-5 days after treatment mentioned above.

Statistical Analysis

The statistical analysis was done using *t test* and correlation coefficient (r). All the results are significant if the *p* value is < 0.005.

Results

CRP levels were elevated ≥ 1.2 mg/L in 27 patients who were positive for bacterial culture (group I), and 11 of the 23 patients with no clear bacteriological evidence of infection (group II); while those with CRP and culture negative were mentioned as group III (see Figure.1). The average elevated CRP level in group I was (11.2±6.8); while average