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Clinical Data of the Diseased with Acute COPD Exacerbations (AECOPD)

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Abstract: ***COPD exacerbation is a major cause of morbidity and mortality, associated with impairment of quality of life, lung function decline and increased consumption of health care resources. Clinic features are studied of acute exacerbation of COPD with GOLD stage III and IV. The assessment of patients reporting symptoms and interpretation by the doctor remains important because diagnosis of COPD exacerbation is clinical diagnosis. The distinction of the variation of symptoms from day to day from symptoms as a result of the occurrence of clinical exacerbation remains a challenge. Resulted that patients with COPD exacerbations are predominantly older age and male gender, which is consistent with the frequency of COPD in these groups. They have increased incidence of coexistent diseases predominantly cardiovascular.Clinical manifestation of COPD exacerbation is dyspnea and mucoopurulent and purulent sputum. Resulted predominantly type I exacerbation by Antonisen. Based on the degree of dyspnea MRC and CAT score indicated that the level of impairment of patients health is high.***

Keywords: COPD AECOPD, dyspnea, cough, sputum, CAT, MRC

1. Background

Throughout the natural progression of COPD, most patients develop acute episodes of symptom worsening, which are separated from the symptom variations from day to day, and can require changes in therapy. These episodes are called exacerbations. The difference in symptoms has been usedto define exacerbations clinically (1), whereas the change in therapy (usually corticostereoid or antibiotic) has been used to define as an exacerbations in an operative manner in classical studies, or from the data in the database (2,3).

COPD exacerbations (AECOPD) are a major cause of morbidity and mortality, that are accompanied with damage in life quality, a decline of pulmonary functions and a very large consumption of medical care resources. Early and adequate recognition of acute exacerbations, as well as an individualized treatment is important tominimizing mortality, morbidity and the decline of pulmonary functions. Reporting symptons from patients, and interpretation from the doctor, might lean toward a subjective, unstable view, suggesting the need of more objective criteria for sickness activity. The diagnosis of AECOPD remains clinical. The difference between variation of symptoms from day to day, from symptoms as a conequence of exacerbations remains a clinical challenge Even so, somewhat ,''loose'' definitions of exacerbations have made it difficult to qualify and characterise episodes. (4)

In the end of the 1990s, it has been reported, that COPD exacerbations are an important definitive of life quality, related to health of patients with COPD and also, with the speed and gravity of disease progresion. Exacerbations are definedby a sudden worsening of symptoms and can be dangerous for living. Even then, if they are treated in the appropriate manner, patients can be returned to the original state of exacerbations, in relation to symptoms, as with physiological markers (5)

AECOPD can be represented through a wide spectrum, from worsening of symptoms, to hospitalization, respiratory insufficence and death. Around half of light reacuteisations are never reported by the patient. People with lower levels of pulmonary functions, have a tendency to go through more reacuteisations and on the other hand, frequent reacuteisation can lead to lower levels of pulmonary functions. It is known that exacerbations are an important part of natural disease progession. There is data that patients with higher frequency of exacerbations (more than three per year) have a more rapid decline in pulmonary functions. (6)

Anthonisen and bshp. (7) have determined COPD exacerbations based on the presence of three specific symptons: emphasising of dyspnea, increase in sputum volume, and purulence. In this sudy, there have been proposed 3 subtypes (I, II and III) depending on the representation of all, or some of the symptoms:

- Type I with all three symptoms:
- Type II with two of the symptoms;
- Type III with one of the symptoms, accompanied by at least one of the following pathologies:present infection of upper pulmonary pathway (fever), increase of coughing, wheezing, increase in respiratory frequency of cardiac frequency

The disadvantage of this definition stands in the fact that it is not widely applicable. Three of the above symptoms do not identify reacuteisations, in all of the diseased; this defintion does not define the patients, that demand oral corticotherapy, due to how it is based on subjective symptoms

A second definition, proposed by a work group in 2000, has been "a drawn out worsening of the patient's state, from a stable condition and further from normal day by day variations, which is, at the beginning, acute, and needs a change in regular medication of a patient with COPD". (8) This definition defines light and medium reacuteisations, that demand an increase of the bronchodilatator dosis. This definition looks to be good, in the analysis between populations of patients. The disadvantage of this definition is on the fact that it is difficult to apply to specific individuals, because of the many variations of symptoms and their different perception from one patient to another. The third definition is based on specific medical intervenation. According to this defnition: the reacuteisation of COPD is a worsening of respiratory symptoms, that demand medication with corticostereoids parenteral/peros, antibiotics, or both and/or hospitalisation due to respiratory symptoms. This definition does not stop the previous consensus definition. Even so this dfinition does change the focus from perception of symptom by the patient, to the actions taken by the patient and medical doctor. It is based on objective criteria; so the reacuteisations are easily evidenced and registered. It is good in studies that have a purpose of comparing medication, that can reduce or slow down reacutiseation of COPD. The main disadvantage of this definition is that medicating COPD reacuteisations may vary between different regions or countries, according to changes in referring or medication practices.

Not having a standarized metric or biomarkers for exacerbations, defining whether exacerbation is present or not, in individual patients, has bee done by being based in both the patient and the clinicist. The decision of the patient comes first, that in a basis of complaint worsening, heads to the medical doctor. The definition of the clinicist, is based on the data of thepatient, in relation to the above described signs, and every other piece of data, taken through physical examination or laboratoric tests, that can suggest alternative explanations for the change in the patient's health condition. The definition of gravity and timelapse (healing) of the exacerbation, has been left to the judgement of the clinicist and the patient, tha tmay have different thoughts. In a clear manner, there needs to be a collective definition and standarized tools, for evaluating COPD exacerbations. Due to the exacerbations being defined by their sign and symptoms experienced by the patient, and the episode itself has been recognized at first and then treated by the patient, in an independent manner, or with assistance from a medical doctor, they are included in the results reported by the patient. COPD exacerbations are heretogene in several levels. Etiology can be connected with viral or bacterial infection, or one with different causes. Most importantly, individual patients can react in different ways to exacerbations. It is possible for there to be fundamental genetic differences, that contribute to clinical reactions. Patients with COPD are also heterogene, in relation to their social support systems. Those with more capable medical doctors might have better overall care, especially when they have a worsening of their state of health. The best medical support can allow patients to survive for longer with more severe dieases Some individuals, when there is an exacerbation, may require a greater intensity of operation. Heretogenicity, can influence the evaluation of the exacerbation in several ways. Firstly, patients with accompanying diseases might have pathologies (e.g congestive heart insufficency) that complicates the diagnosis of the COPD exacerbation. Secondly, many coexisting pathologies can also stop the management of the exacerbations. Diabetes, for an example, can be worsened in patients that need systemic glucocorticoids. The presence of these accompanying diseases, can affect in a dramatic manner the influence of the exacerbation and influence itself, in the way in which the therapy for exacerbation is applied. Base gravity of COPD has a large influence in the nature of clinical operation needed for an exacerbation. An individual, that has a heavily damaged pulmonary function and needs suplementary nasal oxygen with high flux in the base state, can also need intubation, after a minimal lessesening of lung function. On the other hand, an individual with good lung function, can be able to tolerate the considerable damage and still be work capable. This heterogenicityin reaction, makes it hard for the level of medical care use to be an evaluator of exacerbation gravity.

Also problematic, is the timelapse of the exacerbation. As there isno consensus of definition for the beginning, there isn't one for healing and it is difficult to determine the timelapse. An important matter, which also remains unsolved, is the difference between resolution and healing.

2. Aim

Clinical characteristics in acute exacerbation of the diseased with COPD stages III and IV

3. Method

The abstract is a prospective study conducted in Regional Hospital "Fier". In the study, patients with stage III and IV of COPD in exacerbation stage, have been involved. Based on a protocol, anamnestic data of clinical and laboratoric examinations has been collected. Characteristics of patients in the study, are represented in table 1.

Table 1: Characteristics of the patients in the study

Characteristics of patients in the study	Average± Std. Deviation
Age (years)	69.3±7.06
Age of smoking initiation	18.3±9.8
Cigarettes/day	28.3±14.3
Time of smoking (years)	38.6±14.8
Packets/years	59.3±39.2
Alcohol quantity (ml)	97.3±194.8
Time with cogh (years)	9.4±5.8
Time with sputum (years)	7.96±5.52
Time with dyspnea (years)	7.2±5.26
Time of recovery (days)	7.05±1.86
Weight (kg)	68.94±12.32
Height (cm)	166.26±7.09
BMI	25.47±5.09
FVC (% of theoretical)	56.89±11.84
FEV1 (% of theoretial)	36.87±8.57
Tiffeneao Index	51.44±9.18
SaO2	90.34±3.78
CATscore	26.54± 6.8
MRC dyspnea	3.52± .87

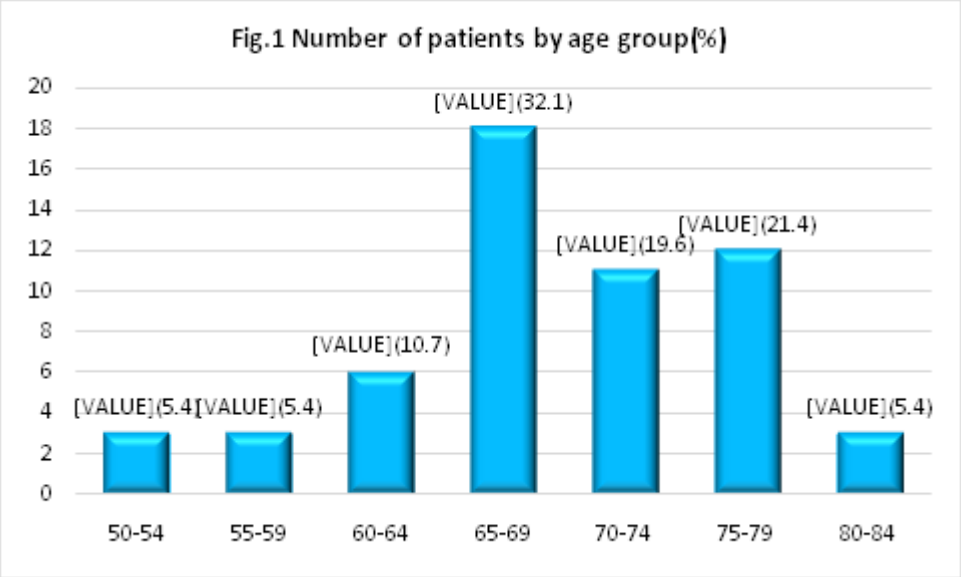
According to GOLD stage, 29 (52%) of the patients have been of the III stage and 27 (48%) of the IV stage, while according to cathegory, there have been 2(3%) –C3, 43(77%) D3 and 11(20%) –D4

Statistical analysis

All collected data was sent into the Microsoft-Excel program, from where they were exported to SPSS (Statistical Package for Social Sciences) 20.0 and Medstat, with which the statistical analysis of the data was conducted. For all cathegoric variables (nominal, including binary/dychotomic scale and ordinal),the absolute values and percentages were calculated. For all numerical variable, where the data was subjected to normal spread, arithmetic averages ±standard deviations were calculated. The presentation of the data was conducted via simple and compound tables, as well as graphics The values of $p \leq 0.05$ were considered significant.

4. Results

As can be observed in Fig 3.1.1 in the studied group of 56 patients with COPD exacerbations, most (41 cases-73.1%) are in the ages between 65 and 79, more often (29 cases-32.1%) between 65 and 69 years old.



In the study there is a predominance of male patients (54 – 96%) and mostly originating from rural areas (32-57%), whereas according to profession, 30 (53.6%) are worker. In the 56 patients taken, there have been registered 73 accompanying diseases, with cardiovascular ones (41 cases – 56.2%) predominate. Only 5(8.9%) of the patients did not have data on accompanying diseased; 8(14.4%) of the patients had 2 of them and 7(12.6%) had up to three accompanying diseases. In relation to the data on the family anamnesis, there have been 6(10.7%) patients with data that indicated presence of COPD in their families and 3(5.4%) of pulmonary diseases, where in 6(10.7%) of cases it has been the father, in 2(3.6%) the brother, and in 1(1.8%) the mother.

In the studied cases, the most common material used both for heating and cooking has been wood, respectively 38(67.9%) cases and 35(62.5%); gas has been used in 9(16.1%) cases for heating and 15(26.8%) cases for cooking. The disease has begun with coughing in 36(64.3%) cases, with dyspnea – 18(32.1) and with cough and dyspnea - 2(3.6%).years -20(35.7%), 10-14 years -16)28.6%), 15-19 year -4(7.1%) and >19 years -4(7.1%). Theappearance of sputum has resulted with ≤ 4 year in 13(23.2%) cases, 5-9 years – 25(44.6%), 10-14 years -11(19.6%), 15-19 years – 3(5.4%) and >19 years -4(7.1%). The symptom of dyspnea has been present for ≤ 4 years in 13 (23.2%) cases, 5-9 years -29(51.8%), 10-14 years -10 (17.6%), 15-19 years -2(3.6%) and >19 years -2(3.6%)

The nature of sputum in 5(9%) of the patients has been mucose, in 37 (66%) –mucopurulent and in 14 (25%) purulent. Dyspnea in 1(1.8%) patient has resultet in struggling, in 9(16.1%) in upwards advance, and in 15 (26.8%) in flat advance, in 26(46.6%) because of dyspnea, the need for breaks during the advance arose, and in 5(8.9%) there has been dyspnea even in a state of stillness.

According to CAT, 52 (93%) of the patients are 10+ and all with MRC2+.
Rezulton se sipas CAT 52(93%) paciente jane 10+ dhe te gjithe me MRC 2+.

Table 2: Patients classified according to the MRC scale of dyspnea

MRC scale of dyspnea			
Scale	Scale of dyspnea in relation to activity	Nr. of cases	%
1	Without dyspnea except in emphasized efforts	0	0
2	Dyspnea when accelerating or ascending small heights	8	14.3
3	Walks slower than others on a flat level due to dyspnea, or needs to rest while walking with his own rhythm	17	30.4
4	Stopping after walking for 100 meters or after a few minutes in a flat level	25	44.6
5	Has a lot of dyspnea if he exists house, or has dyspnea while dressing or undressing	6	10.7
Total		56	100.0

From the anamnesis, it results that patients taken into study, 14 (25%) have gone through one exacerbation per year, 31 (554%) -2 exacerbation per year, 9 (16.1%) -3 exacerbations per year and 2(3.6%) -4 exacerbations per year. COPD exacerbations have been initial in 55(98%) o the patients and in 1(2%) recidivant.

Subjectively, patients have refered to feeling themselves restored in their original state after 5 days in 17(30.4%) cases, after 6 days in 4(7.1%) of cases, in 7 days in 18(32.1%) of cases, after 8 days in 5(8.9%) of cases, after 9 days in 2(3.6%) of cases, after 10 days in 9(16.1%) of cases and after 12 days in 1(1.8%) case

Table 3: Data on the management of COPD patients prior to exacerbations

Treatment of COPD patients prior to exacerbation	*Cases (%)*
Patients in medical care: yes/no	53/3 (94.6/5.4)
Regular/irregular medication: yes/no	48/8 (85.7/14.3)
Antibiotics medication: yes/no	55/1 (98.2/1.8)
Short time oral corticoid: yes/no	54/2 (96.4/3.6)
Inhaled corticoid: no/yes short/yes lengthy	8/12/36 (14.3/21.4/64.3)
Beta mimetics: no/SABA amd LABA	1/55 (1.8/98.2)
Euphiline: yes/no	9/47 (16.1/83.9)
Parasimpaticholitic: yes/no	9/47 (16.1/83.9)
Vaccination: yes/no	6/50 (10.7/89.3)

The data on managing patients in a stable condition and during COPD exacerbations have been included respectively in tables 3 and 4.

Table 4: Data on the management of patients with COPD exacerbations

Treatment of COPD patients during exacerbations	Cases (%)
Ambulatory/hospital treatment	26/30 (46.4/53.6)
Antibiotics medication: yes/no	56/0 (100/0)
Systemic corticoid	56 (100)
Inhaled corticoid: yes/no	42/14 (75/25)
Beta mimetics: SABA and LABA	56 (100)
Euphiline: yes/no	3/53 (5.4/94.6)
Parasimpaticholitic: yes/no	9/47 (16.1/83.9)
Oxygenitherapy: yes/no/non invasive ventilation	50/6/4 (89.3/10.7/7.1)

Not referred for hospitalisations: 5(8.9%) of patients, whereas a hospitalisation has been had in 32 (57.1%), two in -17 (30.4%), four -1(1.8%) and five hospitalisations in one case – 1(1.8%)

5. Discussion

In the studied group AECOPD has been predominant in the age from 65 to 79, more often in the ages 65-69 years; males predominate and more often those of rural origin, and of the worker profession. This is in accordance to many studies on how males predonimate on AECOPD, which is related to smoking as well. Furthermore, in studies, to identify risk factors on exacerbations and hospitaliations of COPD, a more advanced age has been identified as a risk factor. (9, 10)

In relation to the influence of sex in COPD, there are contrasting opinions, where actuald ata suggests different risk of COPD for different communities. Amidst different possible explanations observed among sexes, are differences in pulmonary morphology, fumation, hormonal factors, the difference between genderin inflammatory reaction and professional factor interveners. (11) In our country (12) the data of depistations has a male predomination, where the frequency of males has resulted to be double that of females.

In the study, there appears to be an expressed presence of accompanying diseases, cardiovascular ones predominating. As a result of the aging of the population and the fact some chronic diseases have common risk factors, the accompanying diseases are more common in COPD patients. (13) As a consequence, management of patients with complex accompanying diseases has become a problem for most medics, that handle treatment of pulmonary diseases. The accompanying disease has to be distinct from the base disease, in this case, COPD exacerbations. In a pragmatic manner, an accompanying disease is defined, as any disease that can have an additional or synergic influence with morbidity or mortality that derive from COPD exacerbations.

COPD is oft accompanying with one or more comorbidities and/or systemic effects. So, in many patients, COPD can be considered only the pulmonary component of multimorbidity, that is characterized by accompanying chronic diseases (e.g hypertension, artherosclerosis, chronic heart insufficiency, lung cancer, osteoporosis and depression) and systemic effects (e.g weight loss, muscular atrophy) that cannot be entirely explained by aging or other common risk factors (e.g smoking, diet, inactivity and life style). (14 15 16) Chronic comorbidities are key contributor to the clinical gravity of patients with COPD, since they often influence important markers of the patients.

As is with our study, heart diseases are an especially common comorbidity, contributing in the worsening of health and functional state (17), risk increase for a lengthier exacerbation (17), more pronounced dyspnea (17) and lower survivability (18). COPD< as well, is accompanied by an increased incidence of lung cancer (19, 20) and diabetes prevalence, in some studies and after smoking check. In the study “Toward a Revolution in the treatment of Chronic obstruction (TORCH)” where atients with moderate to severe obstruction have been registered, it was shown that 26% of deaths were because of cardiovascular causes, 21% were due to cancer and only 35% were connected directly to COPD> (21) In patients with mild obstruction, cancer and cardiovascular diseases are calculated respectively for 50% and 20% of deaths. The latest evidence supports the coexistence of several certain comorbidities with COPD (22), suggesting possible common pathobiological mechanisms for these diseases. There is also evidence in rising that acute exacerbation of respiratory symptoms in patients with COPD can be caused by the extrapulmonary mechanisms and the exacerbation of accompanying chronic diseases such as systemic arterial hypertension, acute heart decompensation, atrial fibrillation and pulmonary embolia. (23) On the other hand, COPD exacerbations must influence the risk of cardiovascular events. (24) Even if the acute

exacerbation of respiratory symptoms appears more commonly in COPD patients, it also happens with considerable frequency in smokers without COPD, suggesting they are not COPD specific.(25) COPD patients have a similar prevalence to sleep apnea as in the general population. When this syndrome of overlapping exists, patients have been treated with cPAP , because this has indicated a reduction of mortality (26) In relation to the data on the family anamnesis, there have been 6 (10.7%) patients with COPD data in their family. There have been studies to determine whether family ancestry has anamnestic data for COPD as well as to etermine whether ancestral predisposition is connected to the habit of smoking, increases the likelihood of COPD in comparison with other risk factors, mentioned separately. The conclusion is that in ancestors known for COPD, lies a significant risk factor for it's development. Ancestors known for COPD and smoking, increase the likelihood of COPD development in comparison to the risk factor taken separately (27)

The mucus hypersection has been moderated with the asthma pathogenesis, whereas in the bronchitis pathogenesis it is the predominating factor. Main COPD symptoms are dyspnea in effort, cough and sputum. (28) Other present symptoms, even if presumed not to be as important, include wheezing and toracal discomfort. Edemas in the feet speak for right heart disfunction and is an indicator of gravity. (28)

Many researchers in the years 1970 and 1980 considered coughing and sputum as the most important characteristics of the COPD exacerbation. One of the first studies to use clear criteria with the symptoms, was was the one of McHardy and bshp. who thought the more important symptoms were cough, sputum and wheezing. (29) Even then, by Anthonisen and beyond, dyspnea was considered a key symptom of the exacerbations. The London study of COPD has considered as more significant symptoms of the exacerbation, the increase of dyspnea, the increase of sputum volume and the increase of sputum purulence. (30) COPD exacerbations have been evidenced, based on the changes on the symptoms for over 30 years. The results of some studies have unveiled important information using this method. This has lead to the discovery of certain different phenotypes of COPD exacerbations; the characterised exacerbation mainly by dyspnea without changes of sputum, exacerbations that cannot be recovered and recurrent exacerbations. Further studies have evidenced an increase in the inflamatory marker levels and in the bronchial pathways of the patients, which have slowed down the healing due to disease exacerbations. For those patients that do not heal, there are no clear predicting factors and there are no epidemiological studies of the disease burden of those that cannot recover. Even then, it can be adviced that patients with COPD exacerbations, must be seen in a rutine manner in around five weeks after the exacerbation, to determine the status of recovery. Even if there is no consensus in how exacerbation failure should be manages, the randomized, treatment-controlled and exacerbation recidive studies as well as the Perera and bshp. study reccomend further treatment with systemic stereoids. (48)

Clinical markers of COPD exacerbations are very varying and it is difficult to predict the impending beginning of an exacerbation. Changes in the pulmonary function immediately before the exacerbation are small and not too useful in predicting worsening of COPD symptoms. In fact, the decrease of PEFR or FEV1 are also of little sensitivity on the discovery of the beginning of the exacerbation, even when these two parameters are measued daily. Thi change can be due to the individual changeability, which is greater than the change during the exacerbation. (31) On the other hand, emphasiing that the considerable decine of PEFR is related to the level of dyspnea during the exacerbation as well as with the time spent hospitalized. (32, 33)

COPD exacerbations are related with the acute worsening of pulmonary function markers, including expirating flux. (34) Even so, the use of functional pulmonary tests or spirometry in diagnostiaction, management and predition of COPD exacerbations is still uncertain. Different from asthma, where the flux markers can be sed to direct management of patients, the differences in spirometric variables measured with hospitalised patients with COPD exacerbations are generally small and not neccessarily correlating with an improvement of the symptoms (35); so, a considerable interest has been expessed in the use of other spirometric variables (such as inspiratory capacity) in the field of COPD exacerbation. (34) Even if spyrometry at the patient's bed is becoming ever more disposed, the more detailed evaluation of the pulmonary functions (like measurements of the static lung volume or respiratory mechanics) demand the use of stable equipment and a level of cooperation from the patient, that is often not possible during accute clinical worsening.

CAT (the COPD assessment test) is an instrument projected to gather information in a simple and credible measure, of the health state in COPD and to help patients and medics in the quantitative defining of the influence of COPD in the patient's health.. (38) In our study, in 7.1% of the patients it was <10 and in most, 92.9% at 10+. These results of CAT show that the scale of health damage in the studied patients with COPD exacerbations was of the high stage (26.54 on average) and only 7.1% have resulted with health damage of a lower scale.

The strongest prediction of whether the COPD patient will go through future exacerbations, is based on the previous COPD exacerbation. (39)

COPD is related to periodic exacerations, characterised by the acute worsening of chronic dyspnea symptom, cough and sputum. Hospitalisations due to acute exacerbations are an imortant part of care of COPD patients. Exacerbations are linked to further damage of the health state (40), increase of mortality (41) and high costs (42). Rehospitalisations are common and happen in up to 60% of patients within a year of the last exacervation. Exacerbation frequency influences the patient's health state as well as quality of life. Donaldson and bshp emphasized that the frequency of exacerbation plays a part in the natural history of the disease. In reality, they showed for the first time,t hat the acute exacerbation frequency is an important factor, that contributes in the long time decline of lung function in COPD. (6) Furthermore, patients that go through frequent exacerbations in a year, are likely to have a higher frequency of exacerbations in the following year (43) and those who suffer from severe COPD

have a greatter likelihood of developing grave exacerbations, characterised by acute respiratory insufficence. (44)

From our data it result that after a week of medication, most patients have subjectively improved. According to Spencer and bshp, recovery from an infective exacerbation can be split into two phases: the phase of quick improvement during the first four weeks, and the phase of slow improvement that lapses several months (45) According to the ambulatory treatments/in hospital, respectively it has resulted in 26/30(46.4%/53/6%) patients. COPD exacerbation can have a wide set of representations from the worsening of the symptoms, to hospitalisation, respiratory insufficciency and death. (46) So, definition of the exacerbation as for clinical purposes as well as for research studies has been arguable. (47) Due to the identification of exacerbations possibly being difficult and costly, many epidemiological studies have used hospitalization due to COPD exacerbations as an estimate of eacerbations. Even so, it must be kept in mind that "a hospitalisation due to COPD exacerbation" is not precisely equivalent to "a COPD exacerbation" Firstly, only severe COPD exacerbations need hospitalisation. In fact, it has been reported that patients seek medical care only in half of their exacerbation cases. (33) Secondly, hospitalisations can be caused by factors, that are unrelated to the gravity of the exacerbation, mainly those related to access in hospital care. Thirdly, there are some limitationswhe hospitalisations are taken from administrative databases, such as the need for diagnosis evaluation and the need to consider rehospitalisations, calculated as a hospitalisation when they have happened in a very short period of time (e.g less than 14 days). (2) Lastly, it is likely that new schemes for managing exacerbations, such as creation of hospital-like conditions at home, necessitate a change in the identification of COPD exacerbations, from the medical care databases. Not conidering the above mentioned limitations, many intervening and observing studies have used hospitalisations to investigate the defining means of COPD exacerbations. One of the advantages of this method, is that even if validity always needs to be tested, the information from databases is not a subject to patient criteria (as is a symptom journal) and usually it includes a medical diagnosis. So it is less likely to give a wrong classification. Furthermore, acceptance to be hospitalised is an important event in the progress of COPD, as for the patient as well as for the medical sevice. Lastly, this is a practical method, clear and free to idenfity the exacerbation.

6. Conclusion

In COPD exacerbations, predominate patients of an old age, of the male gender, which fits with the COPD frequency in these groups; have accompanying diseases, with the predomination of cardiovascular ones. In COPD exacerbations, mucropurulent and purulent sputum with emphasized expressions of dyspnea has predominated, as well as type I of exacerbation due to Antonisen. Based on the MRC scale of dyspnea and CAT score, it results that the scale of health damage in the studied patients it's high.

References

[1] Anthonisen NR, Manfreda J, Warren CP, Hershfield ES, Harding GK, Nelson NA. Antibiotic therapy in exacerbations of chronic obstructive pulmonary disease. Ann Intern Med 1987;106:196–204.
[2] Burge S, Wedzicha J. COPD exacerbations: definition and classifications. Eur Respir J 2003;41:43S–53S.
[3] Effing T, Kerstjens H, van der Valk P, Zielhuis G, van der Palen J. (Cost)-effectiveness of self-treatment of exacerbations on the severity of exacerbations in patients with COPD: the COPE II study. Thorax 2009;64:956–962.
[4] Celli BR, Barnes PJ. Exacerbations of chronic obstructive pulmonary disease. Eur Respir J 2007;29:1224–1238.
[5] Wedzicha J. A., Martizez F.J. Chronic Obstructive Pulmonary Disease Exacerbations, 2009 by Informa Healthcare USA, Inc.
[6] Donaldson GC, Seemungal TA, Bhowmik A, Wedzicha JA. Relationship between exacerbation frequency and lung function decline in chronic obstructive pulmonary disease. Thorax 2002;57:847–52.
[7] Anthonisen NR, Manfreda J, Warren CP, Hershfield ES, Harding GK, Nelson NA: Antibiotic therapy in exacerbations of chronic obstructive pulmonary disease. Ann Intern Med 1987, 106(2):196-204.
[8] Ram FS, Rodriguez-Rozin R, Granados-Navvarate A, e bshp. Antiobiotics for exacerbations of chronic obstructive pulmonary disease. Conhrane Database Syst Rey 2006:2:CD004403.
[9] Miravitlles M, Guerrero T, Mayordomo C, Sánchez-Agudo L, Nicolau F, Segú JL. Factors associated with increased risk of exacerbation and hospital admission in a cohort of ambulatory COPD patients: a multiple logistic regression analysis. Respiration 2000;67:495–501.
[10] Chatila W. M., ThomashowB.M., MinaiO.A., CrinerG.J., MakeB.J. Comorbidities in Chronic Obstructive Pulmonary Disease. Proc Am Thorac Soc. May 1, 2008;5(4): 549–555.
[11] Xu X, Weiss ST, Bijcken B, Schouten JP. Smoking, changes in smoking habits, and rate of decline in FEV1: new insight into gender differences. Eur Respir J 1994; 7: 1056–1061.
[12] Bushati J. Semundja mushkerore obstruktive kronike (SMOK) (Nderlidhje epidemiologjike-klinike-paraklinike). Disertacion UT Tirane, 1991.
[13] Soriano JB, Visick GT, Muellerova H. e bshp. Patterns of comorbidities in newly diagnosed COPD and asthma in primary care. Chest 2005; 128(4):2099-2107.
[14] Drazen JM, Fabbri LM. Ageing and multimorbidity. Eur Respir J 2014; 44: 557.
[15] Faner R, Cruz T, López-Giraldo A. e bshp. Network medicine, multimorbidity and the lung in the elderly. Eur Respir J 2014; 44: 775–788.
[16] Divo MJ, Martinez CH, Mannino DM. Ageing and the epidemiology of multimorbidity. Eur Respir J 2014; 44:1055–1068.
[17] Patel AR, Donaldson GC, Mackay AJ. E bshp. The impact of ischemic heart disease on symptoms, health status, and exacerbations in patients with COPD. Chest 2012; 141: 851–857.

[18] Mannino DM, Thorn D, Swensen A. e bshp. Prevalence and outcomes of diabetes, hypertension and cardiovascular disease in COPD. Eur Respir J 2008; 32: 962–969.
[19] de Torres JP, Marín JM, Casanova C. e bshp. Lung cancer in patients with chronic obstructive pulmonary disease — incidence and predicting factors. Am J Respir Crit Care Med 2011; 184: 913–919.
[20] Wilson DO, Leader JK, Fuhrman CR. e bshp. Quantitative computed tomography analysis, airflow obstruction, and lung cancer in the Pittsburgh lung screening study. J Thorac Oncol 2011; 6: 1200–1205.
[21] Calverley PM, Anderson JA, Celli B. e bshp. TORCH investigators. Salmeterol and fluticasone propionate and survival in chronic obstructive pulmonary disease. N Engl J Med 2007; 356: 775–789.
[22] Vanfleteren LE, Spruit MA, Groenen M. e bshp. Clusters of comorbidities based on validated objective measurements and systemic inflammation in patients with chronic obstructive pulmonary disease. Am J Respir Crit Care Med 2013; 187: 728–735.
[23] Roca M, Verduri A, Corbetta L. e bshp. Mechanisms of acute exacerbation of respiratory symptoms in chronic obstructive pulmonary disease. Eur J Clin Invest 2013; 43: 510–521.
[24] Donaldson GC, Hurst JR, Smith CJ. E bshp. Increased risk of myocardial infarction and stroke following exacerbation of COPD. Chest 2010; 137: 1091–1097.
[25] Tan WC, Bourbeau J, Hernandez P. e bshp. Exacerbation-like respiratory symptoms in individuals without chronic obstructive pulmonary disease: results from a population-based study. Thorax 2014; 69: 709–717.
[26] Marin JM, Soriano JB, Carrizo SJ. E bshp. Outcomes in patients with chronic obstructive pulmonary disease and obstructive sleep apnea: the overlap syndrome. Am J Respir Crit Care Med 2010; 182: 325–331.
[27] Battle A. Z. Chronic Obstructive Pulmonary Disease (COPD): Evaluation of Familiar Antecedents as a Risk Factor and Its Relationship with Smoking Habit. Chest. 2003;124(4_MeetingAbstracts):229S. doi:10.1378/chest.124.4_Meeting Abstracts. 229S-a.
[28] Global Initiative for Chronic Obstructive Lung Disease. Global Strategy for the Diagnosis, Management, and Prevention of COPD. Summary of GOLD recommendations, with citations from the scientific literature. Revised 2006.
[29] McHardy VU, Inglis JM, Calder MA. e bshp. A study of infective and other factors in exacerbations of chronic bronchitis. Br J Dis Chest 1980; 74:228-238.
[30] Seemungal T.A.R. Symptom Changes at COPD Exacerbation. Në Wedzicha J. A., Martizez F.J. Chronic Obstructive Pulmonary Disease Exacerbations.2009 by Informa Healthcare USA, Inc.
[31] Papi A, Luppi F, Franco F. e bshp. Pathophysiology of exacerbations of chronic obstructive pulmonary disease. Proc Am Thorac Soc 2006; 3:245-251.
[32] Garcia-Aymerich J, Monsoo E, Marrades RM. Risk factors for hospitalization for a chronic obstructive pulmonary disease exacerbation. Am J Respir Crit Care Med 2001; 164:1002-1007.
[33] Seemungal TA, Donaldson GC, Bhowmik A. e bsh. Time course and recovery of exacerbations in patients with chronic obstructive pulmonary disease. Am J Respir Crit Care Med 2000; 161:1608-1613.
[34] O'Donnell DE, Parker CM. COPD Exacerbations. 3: Pathophysiology. Thorax 2006; 61:354-361.
[35] Bhowmik A, Seemungal TAR, Sapsford RJ et al. Relation of sputum inflammatory markers to symptoms and lung function changes in COPD exacerbations. Thorax 2000; 55:114-120.
[36] Mahler DA. Dyspnea assessment and pharmacological manipulation in COPD. Program and abstracts of the American Thoracic Society 100th International Conference; May 21-26, 2004; Orlando, Florida.
[37] Gross NJ. Outcome measures for COPD treatments; a critical evaluation. COPD. 2004;1:41-57.
[38] Jones PW, Harding G, Berry P e bshp Development and first validation of the COPD Assessment Test. Eur Respir J 2009, 34, 618- 654.
[39] Thomsen M. e bshp. Inflammatory Biomarkers and Exacerbations in Chronic Obstructive Pulmonary Disease. JAMA 2013; 309(22):2353-2361.
[40] Seemungal TAR, Donaldson GC, Paul EA, Bestall JC, Jeffries DJ, Wedzicha JA. Effects of exacerbation on quality of life in patients with chronic obstructive pulmonary disease. Am J Respir Crit Care Med 1998;157:1418–1422.
[41] Connors AF Jr., Dawson NV, Thomas C, e bshp. Outcomes following acute exacerbation ofsevere chronic obstructive pulmonary disease. Am J Respir Crit Care Med 1996; 154:959-967.
[42] Anderson F, Borg S, Jansson SA. e bshp. The costs of exacerbations in chronic obstructive pulmonary disease. Resp Med 2002;96:700–708.
[43] Ball P, Harris JM, Lowson D. e bshp. Acute infective exacerbations of chronic bronchitis. QJM1995; 88:61-68.
[44] Stevenson NJ, Walker PP, Costell RW e bshp. Lung mechanics and dyspnea during exacerbations of chronic obstructive pulmonary disease. Am J Respir Crit Care Med 2005; 172:1510-1516.
[45] Spencer S., Jones P.W. Time course of recovery of health status following an infective exacerbation of chronic bronchitis. Thorax, 58 (2003), 589–593.
[46] Celli BR, Macnee W, and committee members of the ATS/ERS Task force. Standards for the diagnosis and treatment of patients with COPD: a summary of the ATS/ERS position paper. Eur Respir J 2004; 23:932-946.
[47] Pauwels R, Calverley P, Buist AS e bshp. COPD exacerbations: the importance of a standard definition. Respir Med 2004; 98(2):99-107.
[48] Perera WR, Hurst JR, Wilkinson TM, Sapsford RJ, Mullerova H, Donaldson GC, Wedzicha JA. Inflammatory changes, recovery and recurrence at COPD exacerbation. Eur Respir J. 2007, 29(3):527-34.