Name	Year	Biomarker(s) measmeasured	Study group	inclusion criteria	Exclusion criteria	Mean age	Endpoints included	Total duration of follow-up	Number of patients	Total deaths in study	Assay used	Threshold for elevation.

BAILLARD	2003	cardiac troponin I, assayed in blood samples obtained on admission and 24 hours later	71 consecutive patients at Two intensive care units at two french university hospitals	patients admitted with severe exacerbation of COPD	Patients with evidence of pulmonary embolism (PE) or Q-wave myocardial infarction were not included. Exclusion of PE was based on clinical signs and symptoms, laboratory tests (blood gas analysis and D-Dimer tests) at admission. When the diagnosis of PE was suspected, it was ruled out or confirmed by high probability lung scan and Doppler echography examination of the lower limbs, followed by spiral computed tomography scan when doubts remained. The diagnosis of COPD was made according to American Thoracic Society criteria. Severe exacerbation was defined as an acute increase in dyspnoea requiring ICU admission and likely to require ventilatory support.	71	in-hospital mortality	in-hospital mortality	n=71	18	(Stratus II immunoassay analyser, Dade In- ternational)	Levels above 0.5 ng/ml were considered positive.

ABROUG	2006	NT-proBNP	All consecutive patients admitted to the medical ICU between October 2001 and March 2005 for severe AECOPD were considered for inclusion	consecutive patients hospitalised in ICU for the first time for an AECOPD and requiring non-invasive or conventional mechanical ventilation. AECOPD defined as increased in cough and dyspnea nad as a change in sputum abundance and purulence. Severity of AECOPD was defined	patients with an obvious cause of exacerbation (pneumonia or oneumothorax on CXR or PE diagnosed on CT). Patients experiencing cardiac arrest before ICU admission and patients with persistent haemodynamic instability requiring inotropic or vasoactive support. Patients with acute renal failure (calculated creatinine clearance <15ml/min) or who were nonechogenic on echocardiographic evaluation.	median 68	LV dysfunction	In hospital - at diagnosis	n=148	?	NT-proBNP and cardiac troponin T were determined by quantita- tive electrochemiluminescence assay (Elecsys proBNP and Elecsys Troponin; Roche Diagnostics, Indianapolis, IN) on an Elecsys 2010 analyzer (Roche Diagnostics)	A cutoff of 1,000 pg/ml was accurate to rule out left-heart involvement in AECOPD (sensitiv- ity, 94%; negative predictive value, 94%; negative likelihood ratio, 0.08). A cutoff of 2,500 pg/ml had the best operating characteristics to rule in the diagnosis (positive likelihood ratio, 5.16).

BREKKE	2008	Troponin T	patients discharged after treatment for COPD exacerbation from Akershus hospital between 2000-2003. Followed up until 2005.	cases identified using the hospital's patient database. Patients 40 yrs or older who were admitted between January1 2000 and December 31 2003 and were discharged with a primary diagnosis of COPD exacerbation with ICD codes J44.0, J44.1 or COPD as an underlying diagnosis combined with pneumonia as the main diagnosis were included.	patients with a previous diagnosis of sarcoidosis, interstitial lung disease or neuromuscular disease were excluded.	70.9	Mortality following hospital discharge	median 1.9 years		312	Elecsys® Troponin T STAT (Roche Diagnostics GmbH)	cTnT >/=0.04 mug.L(-1)
STOLZ	2008	B-type Natriuretic Peptide (BNP)	208 consecutive patients presenting to the ED of University Hospital Basel with AECOPD from November 2003 to March 2005	COPD as diagnosed by two physicians based on clinical history, physical examination and spirometric criteria as determined by the GOLD guidelines	patients with cystic fibrosis, active pulmonary TB or infiltrates on chest radiographs on presentation. Severely immunocompromised patients also excluded	70	need for intensive care, short-term mortality, long-term mortality	2 years		46	fluorescence immunoassay (Biosite Diagnostics; La Jolla, CA).	none used

BREKKE	2009	Cardiac Troponin T	patients admitted with COPD exacerbation in 2000-03 were identified. 441 had measurement of cTnT performed. Levels of cTnT > or = 0.04 microg/l were considered elevated. Clinical and historical data were retrieved from patient records, hospital and laboratory databases. Odds ratios for cTnT elevation were calculated using logistic regression.	exacerbation of COPD on 2000-2003 who had cardiac troponin T measured	patients with a previous diagnosis of sarcoidosis, interstitial lung disease or neuromuscular disease were excluded	72.2					Elecsys Troponin T STAT	Levels of cTnT ≥ 0.04 µg/l were considered elevated.

FRUCHTER	2009	troponin I	The records of 182 patients with acute exacerbation in whom troponin I levels were sampled during their hospitalization were reviewed retrospectively. Receiver operator curve was used to determine the cut-off level for troponin I that discriminated survivors and non-survivors, and predictors for all-cause mortality were tested in a multivariate analysis.	"Patients were included if the following criterion was met: diagnosis of COPD according to the criteria set by The Global Initiative for Chronic Obstructive Lung Disease (GOLD) (21). AECOPD was defined by the presence of an increase in at least two of three symptoms—dyspnea, cough, and sputum purulence - severe enough to warrant hospital admission without concomitant evidence of pneumonia. " and cTnI measured (this left to discretion of ED physician	"patients with chronic renal failure, defined as calculated serum creatinine level of more than 1.5 mg% (normal <1.1 mg%) for 3 months or more, were excluded. Patients with other conditions known to af- fect troponin levels (9) such as sepsis, pulmonary embolism, myocarditis, cardiomyopathy, and chest contusion were also excluded. "	71.2	Mortality following hospital discharge	3-83 months, median 35		66	cTnI assay used by the hospital labora- tory was AxSYM troponin-I ADV	0.03 ng / L (determined using ROC)

MARTINS	2009	troponinI	analysis of admissions for acute exacerbation of COPD with cardiac tropninI being obtained within the first 48hours of admission. 173 patients of which 05 male and 68 female.previous medical condidtions didnt vary according to sex tough women were more prone to usign beta blockers and diuretics and men to o2 therapy. patients with cardiac troponinI greater than 99th percentile were significantly older.	"Cases were identified by consulting the electronic records for all admissions to the hospital during the year 2007, with primary discharge coding diagnosis of COPD exacerbation. "	exclusion criteria included: marked renal failure (eGFR <15ml/min), persistent haemodynamic instability requiring inotropic or vasoactive support, pulmonary embolus, MI and cardiac arrest prior to admission(diagnoses made by attending physician)	median 77 years	in-hospital death, 18-month survival (for patients with a valid contact number)	18 months		5.9% in-hospital mortality, 21.1% post-discharge	chemiluminescence's microparticle immunoassay, using the ARCHITECT STAT system	0.012 ng/ml.

CHANG	2011	NTproBNP	unselected patients admitted to hospital with physician diagnosed COPD without evidence of acute cardiac disease over 1 year.	admitted to hospital with physician diagnosed COPD	no evidence of acute cardiac disease	71.7	all-cause mortality at 30 days and 1 year	1 year		42	quantitative electrochemiluminescence assay (Elecsys proBNP and Troponin; Roche Diagnostics Corporation, IN, USA)	NT-proBNP >220pmol/L and troponin T > 0.03μg/L are considered abnormal
GARIANI	2011	BNP	Retrospective medical records analysis of all patients hospitalised between January 2003 and May 2009 with the final diagnosis of acute exacerbation of COPD, and who had undergone BNP dosage at admission followed by an echocardiography	hospitalised between January 2003 and May 2009 with a final diagnosis of acute exacerbation of COPD who had undergone BNP analysis at admission followed by and ECHO. Over 18yrs old	patients with a known history of heart failure	75	LV dysfunction	n/a		?	not stated	500 pg/ml (also looked at below 110 pm/ml to rule out LV dysfunction.

Høiseth	2011	Troponin T	Patients were included from 3 January 2005 to 30 November 2006 and followed until 31 December 2008 or death. All patients admitted with assumed AECOPD were eligible for preliminary inclusion in the emergency room, prior to the emergency physicians' knowledge of any blood tests. The research fellow contacted the patient on the ward within a day to retrieve written informed consent and medical history.	The diagnosis of copd, as defined by the British Thoracic Society in 2004,24 was later verified by two study doctors by independent review of the hospital records, blinded for the result of the troponin analysis. In case of disagreement, the diagnosis was settled by consensus. Mortality data were gathered from the National Population Registr	Exclusion criteria were: age <50 years, metastatic cancer and ECOG (Eastern Cooperative Oncology Group) performance status grade ?2, neuromuscular disease with respiratory failure and non-cooperability.	71.5	mortality up until end of study	1.9 years (median)		57	(cobas e 411 immunoanalyser, Roche Diagnostics, Mannheim, Germany)	> 14 ng/l, with a third tertile at 40 ng/l

Høiseth (BMC pulm med)	2012	hs-cTNT	All patients admitted with AECOPD during 23 months between 2003 and 2006. 97 patients included. Mean age at inclusion was 71.5 years and 47% were female.	All patients admitted with AECOPD during 23 months between 2003 and 2006 were eligible	PE	71.5	n/a	n/a	n=97		hs-cTnT (cobas e 411 immunoanalyser, Roche diagnostics)	14 ng/L
Høiseth (Respir red)	2012	NT-proBNP				71.5	Mortality following hospital discharge	1.9 years (median)	n=99, 217 admissions	57	(Roche Diagnostics, Mannheim, Germany)	NT-proBNP tertile limits were 264.4 and 909 pg/m
MARCUN	2012	NT-proBNP	patients admitted for an acute exacerbation of COPD	– age over 35 years old with AECOPD stage II-IV, with residence within the geographical area linked to the study hospital in Slovenia. Able to communicate by telephone.	diagnosis of cognitive impairment, unstable of terminal disease other than COPD, death during hospitalisation.	70	Mortality following hospital discharge, re-hospitalisation	6 months		17	Elecys 2010 (Roche Diagnostics) using Electrochemiluminescence immunoassay (ECLIA)	n age and gender adjusted 95-percentile values for NTproBNP (ng/L) and a single value of 0.012 ng/L for TnT.

OUANES	2012	NT-proBNP	all patients consecutively admitted with severe AECOPD	diagnosis of COPD was based on clinical history and assessment of respiratory function, when available. AECOPD defined according to GOLD guidelines. Severe AECOPD were defined according to clinical findings of respiratory fatigue.	Patients with an obvious cause for AECOPD (pneumonia, pneumothorax and PE) and patients who had cardiac arrests were excluded.	67	LV dysfunction	none		n/a	NT-proBNP levels were determined by quantitative electrochemiluminescence assay (Elecsys proBNP; Roche Diagnostics, Indianapolis, IN, USA) on an Elecsys 2010 analyzer (Roche Diagnostics	The threshold NT-proBNP value with the highest diagnostic accuracy was greater in the setting of renal dysfunction (2000 pg/mL; sensitivity 71%, specificity 82%, compared with 1000 pg/mL in patients with normal renal function; sensitivity 94%, specificity 82%

SOYSETH	2013	Troponin T	consecutive admissions to participating units(a teaching hospital and a pulmonary rehabilitation clinic) for the years 2010-2011 meeting opbjective, standardised criteria for AECOPD and stable COPD. Index group – patients hospitalised for AECOPD at Akershus University hospital Feb2010 – Dec2011. Referrences recruited at lung rehabilitation hospital.	– all the patients had COPD confirmed by spirometry in their stable state within the last five years. All patients between 40 and 79 years old with cumulative tobacco consumption of 10 pack years or more. Current and former smokers included.								