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Clinical Significance of bundle Branch Block Complicating Acute Myocardial infarction at Hospital admission

Authors
Dr Hanan K. Altalhi[1], Dr Ali Areef Fadhlullah[2], Dr Asgad A. Abdalgbar[3]
[1,2]Assistant Prof, [3]Associated Prof
Dept of Internal Medicine, Faculty of Medicine University of Omar El Mukhtar, Albayda –Libya
Email: *[1]hnangk@yahoo.com [2]ali.aref67@yahoo.com, [3]aabdalgbar@yahoo.com*
Corresponding Author
Dr Ali Areef
Dept of Internal Medicine, Faculty of Medicine University of Omar El Mukhtar, Albayda –Libya
Telephone: 00201090064146

ABSTRACT
Background /Aim: *Studies of patients with myocardial infarction and Bundle branch block have reported high mortality rate and poor prognosis. To document the frequency of Bundle branch block and their influence on prognosis in patients with acute myocardial infarction.*
Patients & Methods: *This is case –control study of 42 patients with acute myocardial infarction and bundle branch block (case subject) and 42 patients with acute myocardial infarction and with out block (control subject) .Patients admitted to the coronary care unit were searched to identify those with documented acute myocardial infarction complicated by the presence of bundle branch block. Conduction defects were classified as follow: complete left bundle branch block (LBBB); right bundle branch block (RBBB); right bundle and left anterior fascicular block (RBBB+LAFB); right bundle and left posterior fascicular block(RBBB+LPFB). And all patients treated with Thrombolytic therapy (Streptokinase or t-PA tissue –type plasminogen).*
Results: *In 42 patients with acute myocardial infarction complicated by bundle branch block, most common types of block were LBBB (38%) and RBBB+LAFB (33.4%).*
Patients with RBBB + LPFB had a higher mortality than patients with other intra ventricular conduction defect (42% VS 26%,p<0.025). Hospital mortality was directly related to the degree of heart failure only ,(8%) of patients with class I-II heart failure died, compared to (47%) of Patients with class III-IV heart failure (p < 0.001). The hospital mortality were higher in patients with bundle branch block than in those without block . (26 % VS. 12% p>0.001).
Conclusion: *The occurrence of Bundle branch block in acute myocardial infarction indicate that infarction may be extensive and may result in cardiac failure or death.*
Keyword: *Electrocardiography (ECG), Myocardial infarction (MI), Left bundle branch block (LBBB), Right bundle branch block (RBBB).*

Introduction

Before the wide spread of thrombolytic therapy up to 35% of patients with acute myocardial infarction presented to the hospital with bundle branch block or developed it after admission (Dubois C, et al. 1988; Hindman MC, et al. 1978; Lie KI,

et al.1974). Thrombolytic therapy has been shown to reduce mortality in acute myocardial infarction by restoring antegrad coronary flow in the infarction -related artery and reducing the extent of myocardial injury. However, it is not known whether this has produced parallel reduction in the incidence and severity of bundle branch block. To address this equation we have examined the incidence of bundle branch block and their influence on survival in patients with myocardial infarction who underwent coronary care unit and treatment with thrombolytic therapy .

Aim of the work

We investigated the outcome for patients with acute myocardial infarction who subsequently developed bundle branch block. In relation to those who maintained normal intra ventricular conduction throughout their hospital stay.

Patients & Method

All patients of both sexes sustaining acute ST elevation myocardial infarction were including in this study . Patients with old established conduction defect based on their old medical records, patients with advanced heart failure, renal failure, and patients with permanent pacemaker inserted were excluded from the study

Acute myocardial infarction (AMI) was diagnosed on the basis of recently adopted definition of AMI by ACC/AHA/ESC/WHF task force (Thygesen K, et al .2007). ST elevation myocardial infarction was defined as typical rise and fall in CK-MB (usually twice the level of upper reference limit) and at least one mm ST rise in two contiguous limb leads or 2mm rise in two contiguous chest leads. LBBB was defined as the QRS duration of≥0.12 s ; a Q S or r S complex in lead V1 or R –wave peak time of ≥ 0.06s(often with a notched R –wave) in lead I , AVL .V5, or V6 associated with the absence of a Q – wave in the same lead. (Sgarbossa EB, et al.1996). Right bundle branch block was defined as a prolonged QRS duration o≥ 0.12s or an rsr, rs R, orr SR pattern in lead V1 or V2 . If this was not present , the R –wave in lead V1 had to be notched with prolonged R - wave peak time of 0.05s in lead V1 and normal peak time in lead V5 and V6. Lead V6 and I had to show a QRS complex with a wide S- wave (S duration>R duration or > 0.04s. (Willems JL, et al. 1985).Left anterior fascicular block required a left ward shift of the QRS axis ≤-30 and left posterior fascicular block required a right ward shift to ≥120. (Rosenbaum MB, et al. 1970).

At cardiac care unit, a brief history was obtained from each patient presenting with chest pain including presence of risk factors like diabetes, smoking and hypertension and previous history of ischemic heart disease. Clinical examination was done with emphasis on signs of cardiac failure. Standard 12 leads Electrocardiography (ECG)was done at cardiac care unit and blood samples were sent to laboratory for cardiac enzymes and base line biochemical profile. All patients were considered for thrombolytic therapy (injection streptokinase 1.5 million units over one hour) in the absence of all contraindication and management according to standard treatment protocol. All patients under went continues ECG monitoring for at least 48 hour on admission to cardiac care unit and daily during hospital stay .The worst class of heart failure for each patients obtained by review of the clinical record, these were designated classes I-V as defined by killip and Kim ball (Killip T, et al. 1967): class I,no heart failure; class II, mild heart failure manifested by basilar ales and/or an S3 gallop; class III, pulmonary edema, determined by the presence of dyspnoea and S3 gallop, pulmonary rales, and chest X-ray finding compatible with pulmonary edema; and class IV, Carcinogenic shock manifested by hypotension (systolic pressure < 90mmHg), Oliguria (< 20ml /hr) , and poor perfusion to skin.

Result

Table 1:- Shows characteristics and variation of study subjects.

84 patients with acute myocardial infarction were included in the study, 42 patients with bundle branch block (case subjects) and 42 patients without bundle branch block (control subjects). There is significant difference in mean age between case group and control group being older in case group ($p<0.006$) , peak total creatinine kinas was higher among case subjects ($p<0.001$) and number of diabetic patients increased among case subjects($P<0.01$).

Most patients with bundle branch block at hospital admission had anterior wall infarction 34(80.9%), inferior or posterior wall 8 (19.1%). Patients without bundle branch block at hospital admission had anterior wall infarction 22 (52.3%), inferior or posterior in 20 (47.7%).

Table 1- Shows Characteristics and Variation of Study Subjects

variable	Patients with BBB(n=42)	Patients without BBB(n=42)	P value
Age(year)	65±12 year	61±12 year	0.006
Men	30(71%)	25(60%)	0.001
SBP(mmHg)	127(110-140)	126(110-189)	0.31
DBP(mmHg)	75(64-96)	72(62-88)	0.39
HR(beat/min)	78(64-90)	72(62-88)	0.07
Peak CK(U/L)	1.964(717-2.900)	1.557(642-2.736)	<0.001
CK-MB(U/L)	256±143	167±75	<0.001
Hypertension	18(43%)	19(46%)	0.29
Diabetes	10(23%)	7(17%)	0.01
Current smoker	12(28%)	31(73%)	0.23
Anterior-indeterminate wall MI	34(80.9%)	22(52.3%)	<0.001
Inferior-posterior wall MI	8(19.1%)	20(47.7%)	<0.01

Date presented are median (lower, upper quartiles).mean value ±SD or number (%) of patients .CK = Creatine kinase ; DBP = diastolic blood pressure; SBP = systolic blood pressure; HR=Heart failure.

Table 2:-Shows the incidence of the various type of bundle branch block.

Left bundle branch block was observed in 16 patients (38%) and right bundle branch block in 5 patients (12%) and right bundle branch block + left anterior fascicular block 14patients (33.4%) and right bundle branch block + left posterior fascicular block 7 patients (16.6%).

Table 2: Shows the incidence of the various type of bundle branch block.

Type of BBB	Number	(%)
LBBB	16	38%
RBBB	5	12%
RBBB+LAFB	14	33.4%
RBBB+LPFB	7	16.6%

Abbreviations: LBBB = left bundle branch block ;RBBB = right bundle branch block; LAFB = left anterior fascicular block ;LPFB = left Posterior fascicular block

Table 3: Determinate of Hospital Mortality in patients with Acute Myocardial Infarction and Bundle Branch Block

Patients with acute inferior or posterior had a (12.5%) hospital mortality, while patients with acute anterior or indeterminate location infarction had a (29%) hospital mortality.

Patients with RBBB + LPFB had a higher mortality than patients with other intra ventricular conduction defect (42%). Hospital mortality was directly related to the degree of heart failure only, (8.6%) of patients with class I-II heart failure died, compared to (47%)of Patients with class III-IV heart failure ($p < 0.001$).

Table 3: Determinate of Hospital Mortality in patients with acute myocardial infarction and bundle branch block.

Determinant infarction location	Number of patients	Hospital Mortility (%)
Ant - ind	34	29%
Inf - post	8	12.5%
Type BBB		
LBBB	16	19%
RBBB	5	20%
RBBB + LAFB	14	29%
RBBB + LPFB	7	43%
Heart failure		
Killip class I + II	23	8,6%
Killip class III +V	19	47%

Ant – Ind = anterior or in determinant location infarcts ; Inf-Post =inferior-posterior.

Table 4:- Comparison of Hospital Mortality during acute myocardial infarction in subgroups of patients with and without bundle branch block.

The hospital mortality was higher in patients with bundle branch block than in those without blocks (26% VS 12% $p< 0.0001$) but the mortality

associated with the development of power failure was similar for patients with and without bundle branch block, regardless of infarction location (47% VS 50% p NS)Although low mortality in patients with bundle branch block but no power failure (8.6%).was higher than in patients with neither bundle branch block nor failure (2%) p <0.001.

Table 4: Comparison of Hospital mortality during acute Myocardial infarction in sub groups of patients with and without bundle branch

	Bundle branch block		No bundle branch block		
	N	Hospital mortality	N	Hospital mortality	p
Total PATIENTS	42	26%	42	12%	P<0.0001
Ant –Ind MI	34	29%	22	18%	0.0001
Inf – Post MI	8	12.5%	20	5%	0.01
Killip class I -II	23	8.6%	34	2%	0.001
Killpclass III-V	19	47%	8	50%	NS

Discussion

Bundle branch block has been reported to be present at sometime during hospitalization in 13% of patients with acute myocardial infarction (Mullin CB, et al. 1976; Killip T, et al. 1967; Bigger JT, et al.1977).

The frequency of occurrence of the different types of bundle branch blocks in this study is similar to previous reports as reviewed by Mullins and Aktins (Mullin CB, et al. 1976) LBBB and RBBB+ LAFP are the most common, occurring at about the same frequency, and isolated RBBB and RBBB+LPFB are less common.

The hospital mortality of myocardial infarction complicated by bundle branch block ,average 15-20 % (Moss A,et al. 1964).The 26% mortality rate in the 42 patients with bundle branch block is significantly higher than the 12% mortality for control subjects without bundle branch block; however, .this mortality rate is lower than the 44% mortality (range 19%-74%) for bundle branch block during acute myocardial infarction reported in the literature (Hunt D,et al. 1969; CollJJ, et al. 1972)

The wide range of mortality figures and the difference between this study and those previously reported probably reflects different cardiac care unit population

The specific types of bundle branch block have been noted in the literature to influence hospital mortality, but the results have been variable. Some studies have demonstrated a lower mortality in patients with LBBB than in patient with isolated RBBB or bifascicular block involving the right bundle branch block; (Gould L, et al. 1973; Coll JJ, et al.1972). however, other studies, including this one, have demonstrated equal or highermortality with LBBB ,often associated with larger area of infarction ((Gould L, et al. 1973; Coll JJ, et al.1972)). In their review of the literature, Mullins and Atkins (Mullins CB, et al. 1976) found that mortality rates were similar for the various block (44-57%) and was highest for the small number of reported patients with RBBB +LPFB, this is similar to the result of this study.

When bundle branch block complicate acute myocardial infarction ,the site of infarction is usually anteroseptal (Godman MJ, et al.1970; Roos JC, et al.1970; Lichstein ,et al.1973;Rizzon P, et al. 1974; Nimetz AA, et al .1975.).In this study , 34%of the infarction which could be localized were anterior. The relatively small number of patients with inferior or posterior infarction had a lower risk of dying during hospitalization than patients with anterior or in determinant location infarcts, and although the incidence of power failure was similar for the different infarction locations , mortality was lower in patients with inferior or posterior infarctions and power failure than patients with anterior or in determinant location infarction and power failure. The fact that patients with bundle branch block, have a high incidence of power failure and die as a result of progressive and irreversible hemodyn-amic deterioration has been stressed in the literature (Hunt D,et al.1969;Coll JJ, et al. 1972)

This study confirms the common occurrence of pulmonary edema and cardiogenic shock in patients with bundle branch block during acute

myocardial infarction, as the incidence of power failure in this study is significantly higher than incidence in a control group during infarction.

Conclusion

The occurrence of bundle branch block in acute myocardial infarction is important because its indicate that infarction may be extensive and may result in heart failure or death. Such patients should be closely observed and monitored.

In patients with bundle branch block and a typical presentation its important first to think about a possible acute myocardial infarction and in the absence of contraindications, administration of thrombolytic therapy is highly indicated incase with strong clinical suspicion .

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