CLINICAL CASE
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COMPLETE LEFT BUNDLE BRANCH BLOCK AS PREDICTOR OF CARDIOGENIC SHOCK IN PATIENT WITH ACUTE MYOCARDIAL INFARCTION ON THE BACKGROUND OF DIABETES MELLITUS TYPE 2
Olena Makharynska, Nataliia Bogun, Irina Oktiabrova, Eke Miracle Chiamaka

Summary: Cardiogenic shock is the leading cause of death in patients with acute myocardial infarction, with high hospital mortality rates ~80 %. The incidence of cardiogenic shock among myocardial infarction patients is approximately 7 %. Cardiogenic shock patients are found that pre-existing diabetes is associated with an increased risk of cardiogenic shock and it worsens outcomes, with a longer hospital stay. Cardiogenic shock develops approximately twice as often among diabetics as among non-diabetic patients with acute myocardial infarction. Diabetes mellitus and left bundle branch block are predictors of cardiogenic shock complicating acute myocardial infarction. Except for bad prognosis, left bundle branch block can be a marker of a slowly progressing, degenerative, ischemic or non-ischemic cardiac disease, affecting not only the muscle but also the heart conduction system. Immediate diagnosis and management are required. In this article, a clinical case of acute posterior myocardial infarction complicated by complete left bundle branch block and cardiogenic shock in the patient with concomitant diabetes mellitus type 2 is demonstrated. This article emphasizes the priority of referring patients with left bundle branch block to primary percutaneous coronary intervention, usage of more specific ECG criteria for acute coronary syndrome, the role of myocardial infarction biomarker including sensitive assays for cardiac troponins, and bedside echocardiography which may improve diagnostic accuracy and result in timely intervention in such patients. This article also underlines the role of mechanical circulatory support, urgent reperfusion therapy, and strict control of glycemia in the acute phase of myocardial infarction which may contribute to clinical stability of patients with diabetes mellitus and myocardial infarction complicated by cardiogenic shock.

Key words: left bundle branch block, acute myocardial infarction, cardiogenic shock, diabetes mellitus type 2, mechanical circulatory support

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Cardio-vascular and endocrine disorders are two the biggest problems in the routine medical practice. On the one hand, cardiogenic shock (CS) is the most common cause of cardiovascular death in patients with acute myocardial infarction (MI) with mortality rate ~80 %. The incidence of cardiogenic shock among MI patients is approximately 7 %, and in the total infarct population, several important risk factors such as previous MI, infarct size, and location have been identified as predictors of cardiogenic shock, but the death from cardiogenic shock complicating MI is a main contributor to the in-hospital mortality of MI [1]. On the other hand, recent studies suggest that patients with diabetes mellitus are at increased risk of death after MI, but the presence of diabetes among patients with cardiogenic shock, the influence of diabetes on the risk of shock development in acute MI and the survival rate of diabetic patients with cardiogenic shock is less clear [2]. In the large study with 72,765 cardiogenic shock patients were found that pre-existing diabetes was associated with an increased risk of cardiogenic shock (5.8 % vs 5.2 %; adjusted odds ratio [aOR] 1.14) and it worsens outcomes (higher in-hospital mortality (37.9 % vs 36.8 %;
aOR 1.18), with a longer hospital stay (mean±SEM: 11.6±0.16 vs 10.9±0.16 days) [3]. Several studies in randomly sampled populations, sicker patients with hypertension, suspected or existing coronary artery disease, and post-myocardial infarction patients, have evaluated the impact of LBBB (left bundle branch block) on mortality in patients with isolated LBBB, and found the prognosis of LBBB patients is largely related to the type and severity of the underlying heart disease [4]. Left bundle branch block is an independent negative prognostic marker in acute myocardial infarction. A diagnosis of MI is especially difficult in the setting of LBBB because of the characteristic electrocardiography (ECG) changes caused by altered ventricular depolarization [5]. In the majority of MI, LBBB appearance are a result of involving a large portion of the distal conduction system including both fascicles with a discrete lesion just distal to the bundle of His leading to extensive myocardial damage causes LBBB. A new LBBB is caused by MI appeared usually in anterior or anteroseptal MI locations, involving a large myocardial damaged area. Inferior or posterior infarctions may uncommonly result in a new LBBB from involvement of the more proximal portion of the conduction system supplied by the atroventricular nodal artery. [6]. In the German study LBBB with acute chest pain was observed in a cohort of patients with high morbidity and mortality rates [7]. Except bad prognosis LBBB can be a marker of a slowly progressing, degenerative, ischaemic or non-ischaemic cardiac disease, affecting not only the muscle but also the heart conduction system [4]. In contemporary medical practice in most patients with suspected acute MI, the clinical utility of new or presumably new left bundle branch block (LBBB) as a diagnostic criterion equivalent to ST-segment elevation is not well established. Even with the introduction of modern intensive care units (ICUs), advanced medical treatment, and invasive devices, in-hospital death rates remain high at 40 %-50 %, despite advances in early revascularization and adjunctive pharmacotherapy [8].

Our 72-year-old patient was delivered by ambulance with complaints of general weakness and dyspnea on minimal physical exertion next day after her chest pain appeared. Intensive chest pain bothered patient on 11-dec-2017 at 9pm for which the patient has consecutively taken 6 tablets of Nitroglycerin. On admission moment patient denies chest pain as complain. In her past medical history remarkable were: diagnosis of Diabetes mellitus type II was established in 1995, patient constantly received treatment with “Lantus” 40 IU/day and Glybenclimide 5 mg/day. She suffers from Arterial hypertension from 1999, with max number 220/1000 mm Hg, controlled by constant antihypertensive treatment with Ca-channel blocker and β-blocker. Retrosternal chest pain first episode was noticed in 2010 and diagnosis of Coronary artery disease: stable angina IV class according to NYHA was established. In 2011 performed coronarography revealed a diffuse atherosclerosis of coronary arteries. Right coronary artery - critical occlusion before bifurcation with stenting (arrow), TIMI -1 (penetration without perfusion) before stenting, in proximal segment – stenosis 70 %, in middle segment – 50 %. Left coronary artery - prolonged atherosclerotic plaque with sub-occlusion in left anterior descending branch, atherosclerosis of diagonal branches, diffuse stenosis of circumference branch – 60%-70%. Percutaneous coronary intervention (PCI) of the left coronary artery in left anterior descending branch and also of the right coronary artery with drug - eluting stents “Endeavor Resolute” was done. No chest pain or physical exertion intolerance after PCI was observed by her cardiologist. Subsequently, symptoms re-appeared elementarily in 2016.

Objective examination of this patient in the emergency department (ED) during hospitalization revealed lethargic passive patient in the severe state, skin and mucosa pale, visualized acrocyanosis and cyanosis of lips. Patient was overweight (body mass index – 32 kg/m²). SpO₂ – 75 %. Vital signs were: blood pressure (BP) - 110 / 60 mm/Hg, heart rate (HR) = pulse rate (PR) – 90 bts in min., respiratory rate (RR) – 18 in min. During lung auscultation were found: decreased breath sounds over inferior and lateral parts of lungs, wheezing over both lung fields and rales below both scapular angles. Left border of the relative heart dullness displaced 1.5 cm away from midclavicular line, heart tones during auscultation found rhythmic, muffled. Abdomen of normal size, symmetric, no tenderness, but liver was enlarged +2cm with no tenderness. Also were observed pitting edemas.
ECG of the patient in ED showed: sinus rhythm, HR – 83 bpm, left axis deviation, LBBB (QRS - 0.12s), paired supraventricular extrasystoles in V1, posterior myocardial infarction (ST-segment elevation greater than or equal to 0.1 mV (1 mm) in leads with a positive QRS complex, and ST depression greater than or equal to 0.1 mV (1 mm) in leads V1 through V3, in leads with a dominant S wave. Negative T in 1 and aVL, Q wave start of formation).

Fig. 1. ECG of our 72 y.old patient during admission in ED

Before patient was referred in ED, previous ECG findings from 2014 were: sinus rhythm, HR 80 in min., normal heart axis. Relative signs of left ventricle (LV) hypertrophy. Repolarization alternation in anterior-lateral LV wall. Preliminary diagnosis of Acute coronary syndrome (ACS) with ST-elevation was established.

Fig. 2. Previous patient’s ECG from 2014 after PCI was performed in 2011
In the complete blood count during admission clinically significant changes were: mild hypochromic anemia (hemoglobin - 80 mg/l (N 120-140 mg/l), red blood cells count – 3.5 (N 3.9-4.7 * 1012), color index – 0.75 (N 0.85 – 1.15)) and leukocytosis 14.7 *109 (N 4 – 9 *109) despite erythrocyte sedimentation rate (EST) remains in normal ranges. Glucose profile was 26.6 (admission) – 15.0 – 13.2 - 10.2 mmol/l on the background of insulin therapy prescribed. Troponin I was elevated till 0.84 ng/ml (N till 0.5).

Echocardiographic findings of this patient were represented by dilation of left heart chambers, LV hypertrophy and diffuse contractility decline. LV FDD – 59 mm (N – 35 – 55mm), LV FSD – 48 mm (N – 23 – 38 mm), LV FDV – moderately increased – 174 ml. EF – 35 % (N - 55 – 78 %). Stroke volume – 62 ml – increased Posterior wall thickness in diastole – 13 mm (N – 6 –13mm). Mild hypertrophy of LV wall. Intraventricular wall thickness in diastole – 12 mm. Mitral regurgitation II stage. Right ventricle parameters were in normal ranges. Left atrium as right one, both, were dilated (45 mm in diameter (N – till 39 mm) and 37 mm in diameter (N – 25-37) respectively).

Despite prescribed according to the current guideline’s treatment and previous continuous therapy both Arterial hypertension and Diabetes mellitus with routine dual therapy with aspirin + ticagrelor after PCI was performed deterioration of our patient state continued to develop. After 2 hours from admission, dyspnea, exacerbated by horizontal position and in minimal exertion appeared, RR – 26 in min. Acrocyanosis and cyanosis of lips remain present. SpO2 – 82 %. Crackles in lower lung fields during auscultation noticed by ICU department physician. Heart sounds are muffled, rhythmic. HR- 68 bpm. BP-90/60 mm/Hg on dopamine infusion. Diuresis by catheter 8 - 10:00 is 10 ml. On ECG was seen progression of acute MI on the background of developing cardiogenic shock; sinus rhythm, HR-84 bpm. Left axis deviation. LBBB (QRS - 0,12s). Positive Q wave, posterior myocardial infarction (ST-segment elevation greater than or equal to 0.1 mV (1 mm) in leads with a positive QRS complex in III and aVF, and ST depression greater than or equal to 0.1 mV (1 mm) in leads V1 through V3, ie, leads with a dominant S wave. Reciprocal negative T in 1 and aVL, Q=0.02 sec, 4mm), negative dynamics comparing with previous. Chest X-ray data showed congestive changes, probable pulmonary edema.

In 3.5 hours from admission clinically deterioration of the patient’s state was represented by signs of cardiogenic shock complicated by pulmonary edema. BR became 30 in min. SO2 continue to decrease – 63 %. HR=Ps=66 in min. BP 85/60 mm Hg on dopamine infusion background. Pitting edemas. Diuresis by catheter 8-14:00 is 20 ml. On behalf of pulmonary edema treatment was added Sol. Morphini hydrochloride 1 % - 1ml in 10 ml 0,9 % NaCl solution bolus, Furosemid 60 mg intravenously, venous tourniquets placement, O2 inhalation.

Continues therapy didn’t show any positive response in patient’s state.

Fig. 3. Acute posterior myocardial infarction progression on ECG 2,5 hours after admission. Positive Q wave, posterior myocardial infarction (ST-segment elevation greater than or equal to 0.1 mV (1 mm) in leads with a positive QRS complex in III and aVF
In 16 hours after admission patient become unconsciousness, pupils were wide, no respiration observed, no BP or pulse on main vessels found. On ECG: idioventricular rhythm seen (Fig. 4.).

Fig. 4. Idioventricular rhythm in patient with ACS with ST-elevation

Therapy with cardiovascular resuscitation performance, artificial lung ventilation in CMV regimen, on the background of adrenalin 0,18 % – 1 ml and atropine 0,9 % – 10 ml injections weren’t successful. Patient remained being unconsciousness with wide pupils and no photoreactions. Respiration was absent, no BP or pulse on main vessels observed. On ECG was seen isoline. Biological death was established.


Among high mortality rate factors of cardiogenic shock in patients with MI modern scientists define right and left bundle branch block (an independent negative prognostic marker in acute Myocardial infarction (AMI) (30 % vs. 19 %, p = 0.012, OR 1.57)), advanced age (75 years and more), large myocardial involvement, severe left ventricular dysfunction, severity of end-organ injury. The glucose level at admission is a strong independent predictor for mortality also as a present co-morbidity: STEMI, Dyslipidemia, Stroke or Diabetes mellitus. Our patient according to this list had enormously high risk of death from MI despite continuous medical therapy of her medical problems including PCI performed. She had 7 among 11 mortality factors present.

Conclusion

Despite the present scientific data clearly shows approximately two-fold increased risk of cardiogenic shock for diabetic MI patients compared to nondiabetic patients, if shock has developed, outcomes and survival is similar in both groups. Probable causes of MI after PCI performance in DM patients are: re-stenosis after PCI, progression of a separate untreated plaques, or the development of new ones with acceleration of negative remodeling owing to neointimal proliferation after PCI and increased platelet aggregation, small distal vessels microangiopathy and reduced collateral blood flow. The current management of patients with acute myocardial infarction complicated by cardiogenic shock is associated with a high rate of mortality, despite widespread regional implementation of rapid transfer to percutaneous coronary intervention-capable centers for prompt infarct-related artery reperfusion. In all patients with cardiogenic shock after MI in the TRACE registry received ACS noninvasive treatment results were associated with a poor short and long-term outcome of cardiogenic shock after MI [1]. In selected patients as our patient is who was hemodynamically unstable, there might be a benefit associated with early institution of mechanical circulatory support before revascularization. Unloading the left ventricle during MI to decrease LV wall stress, stroke work, and myocardial oxygen demand might limit myocardial cellular loss and decrease the extent of infarction. The major clinical utility of short-term mechanical circulatory support is the reversal of shock by the restoration of cardiac output for
ПОВНА БЛОКАДА ЛІВОЇ ГІЛКИ ПУЧКА ГІСА ЯК ПЕРЕДВІСНИК КАРДІОГЕННОГО ШОКУ У ХВОРИХ НА ГОСТРІЙ ІНФАРКТ МІОКАРДУ НА ФОНІ ЦУКРОВОГО ДІАБЕТУ 2 ТИПУ

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Резюме: Кардіогенній шок є основною причиною смерті у пацієнтів з гострим інфарктом міокарда з високим рівнем гострітальної смертності ~80 %. Частота кардіогенного шоку серед хворих на інфаркт міокарда становить приблизно 7 % У пацієнтів з кардіогенним шоком виявлено, що навіть цукровий діабет поєднуєтись з підвищеним ризиком кардіогенного шоку і поширює наслідки з більш тривалим перебуванням у лікарні. Кардіогенній шок розвивається приблизно однією часткою серед діабетиків, ніж серед недіабетичних хворих з гострим інфарктом міокарда. Цукровий діабет...
ПОЛНАЯ БЛОКАДА ЛЕВОЙ НОЖКИ ПУЧКА ГИСА КАК ПРЕДИКТОР КАРДИОГЕННОГО ШОКА У ПАЦИЕНТА С ОСТРЫМ ИНФАРКТОМ МИОКАРДА НА ФОНЕ САХАРНОГО ДИАБЕТА 2 ТИПА

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Резюме. Кардиогенный шок является основной причиной смерти у пациентов с острым инфарктом миокарда, с высокими показателями госпитальной смертности ~80 %. Частота возникновения кардиогенного шока среди пациентов с инфарктом миокарда составляет примерно 7 %. У пациентов с кардиогенным шоком обнаруживается, что ранее существовавший сахарный диабет связан с повышенным риском кардиогенного шока и ухудшает исходы с более длительным пребыванием в стационаре. Кардиогенный шок развивается примерно в два раза чаще у диабетиков, чем у недиабетических пациентов с острым инфарктом миокарда. Сахарный диабет и блокада левой ножки пучка Гиса являются предикторами кардиогенного шока, осложняющего острый инфаркт миокарда. За исключением плохого прогноза, блокада левой ножки пучка Гиса может быть маркером медленно прогрессирующего, дегенеративного, ишемического или неишемического заболевания сердца, поражающего не только миокард, но и проводящую систему сердца. Требуется немедленная диагностика и лечение. В этой статье подчеркивается приоритетность направления пациентов с блокадой левой ножки пучка Гиса к первичным центрам чрескожного коронарного вмешательства, использование более специфических критериев ЭКГ для острого коронарного синдрона, роль биомаркеров инфаркта миокарда, включая чувствительные анализы на сердечные тропонины, и прикроватную эхокардиографию, которая может улучшить диагностическую точность и результат своевременного вмешательства у таких пациентов. В этой статье также подчеркивается роль механической поддержки кровообращения, неотложной реперфузционной терапии и строгого контроля гликемии в острой фазе инфаркта миокарда, что может способствовать клинической стабильности пациентов с сахарным диабетом и инфарктом миокарда, осложненным кардиогенным шоком.

Ключевые слова: блокада левой ножки пучка Гиса, острый инфаркт миокарда, кардиогенный шок, сахарный диабет 2 типа, механическая циркуляторная поддержка
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