ISSN: 2753-7919
British Journal of Healthcare and Medical Research; Vol. 13 No. 02 (2026) (324-328)
https://doi.org/10.14738/bjhr.1302.20284

Systemic Bactericidal Complement in Human Cardiomyopathies: Revisiting Approach

Ibrahim M S Shnawa[1], Mohamed M Abdurazak[2], Baha H H Alameidi[3]

1. Department of Medical Biotechnology, College of Biotechnology, AL-Qasim Green University and Department of Dental Technology, College Health an Medical Technology-University of Hilla. Babylon IRAQ
2. Consultant Medical Internist-Babylon Board of Health/IRAQ
3. College of Dentistry, University of Babylon ,Babylon/IRAQ

Abstract: Cardiac diseases are of common prevalence among age human beings. In a clinical setting, the cardiologist of working team at Hilla Mergan Teaching Hospital, diagnose 23 cardiomyopathy test patients whom complained inflammatory-infectious manifestations in continuum with ageing processes. Five normal aged and five normal adulthood were the controls. The test and control groups were enrolled in blood collection with heparin for bactericidal assay. The heparinized one in tenth diluted blood samples were inoculated with fresh diluted E coli culture suspensions. The inoculated blood samples were incubated for 10,20 and 40 minutes at 37C.Then streaked onto nutrient agar plates and incubated for an overnight period in 37C.To score bactericidal activity onto the streak line; confluent growth means no action, weak growth means hypoactive and no growth means active bactericidal complement. Complement bactericidal activity patterns in cardiomyopathy patients were as; inactive, hypoactive and active. The nature of this bactericidal activity was found to be dependent on the initial blood-bacteria interaction time at 37C as the time passed from 10 to 40 min. Nine out of the 23(39.13%) test patient have shown inactivity up to 40 min incubation. Fourteen out of the 23(60.7%) were expressing various grades of bactericidal activity. Complement inactivity can be attributed to; consumption of complement in immune-pathogenesis of the cardiac disease, ageing and/ or infection induced complement suppressive effects. Complement hypoactivity may be due to the cardiomyopathy and /or ageing.

Keywords: Blood, bactericidal effect, cardiac disease cardiomyopathy, complement, hypo-activity.

INTRODUCTION

The systemic complement system is a serum protein system that composed of about 30 components. Each component is different from the other in chemical, molecular structure, electrophoretic mobility and immunogenicity. Human electro-phoro-gram seen have shown that complement proteins are located at alpha, beta and gamma globulin but complement is different from the immunoglobulin in chemical nature and in immune features. Complement proteins are thermolabiabl at 56C for half hour. Complement is activated in a cascade like pathways in which the early activated component resulting in a molecule integrates with others into a functional unite that help in cell lysis, opsonization and release of active inflammatory peptides. Complement system are of fluid, membrane and complosom phases. The activation of complement system encompasses three activation phases as; initiation, augmentation and membrane attack, cell lysis. The activation pathways are; classical, lectin and properdin. Properdin is involved in in innate immunity,

classical and lectin pathways are involved the adaptive immunity [1-4]. Complement system plays a key role in the immune-pathogenicity of ischemic heart disease. Autopsy samples taken from heart in cases myocardial infarcted patients demonstrated selective complement deposition in areas of infarction[5]. Complement mediated inflammation are found important in a variety of heart diseases [6]. The complement fractions, C5b-9 may function as a possible biomarker for myocardial tissue damage in the acute coronary syndrome[7]. Complement factor D is associated with cardiovascular and metabolic diseases [8]. The objective of the present work was to map systemic complement bactericidal activity in cardiomyopathy patients.

MATERIALS AND METHODS

Based on the clinical examination, electrocardiography and laboratory investigation the study patients were diagnosed by the team cardiologist as; ischemic heart disease, acute myocardial infarction, heart failure, myocardial infarction with plural effusion and myocardial infarction with lung consolidation, ei.cardio and cardiomyopathy patients. In addition to five adulthood normal control and five aged normal controls,Table-1.

Table-1: Cardiac and cardiomyopathy test patients and normal controls

Test Groups	Disease	Number :Total ;%
Pateints	Ischemic heart disease	2/23,8.6%
	Acute myocardial infarction	9/23
	Heart failure	2/23,8.6%
	Old myocardial infarction	3/23,13.0%
	Cardiovascular atherosclerosis and heart failure	1/23,4.4%
	Heart failure	3/23,13.0%
	Heart failure and pneumonia	3/23,13..0%
Controls	Normal adulthood control	5
	Norma aged control	5

Heparinized blood samples were collected from both test and control groups. Heparinized blood samples were diluted one in tenth with sterile saline and inoculated with fresh diluted one in tenth two hours E.coli culture. The inoculated blood samples were incubated at 37C for 10;20; and 40 minutes at 37C. Then after these incubation periods a drop from each incubation period were streaked onto nutrient agar plates and incubated overnight at 37C. Bactericidal power were scored as; confluent growth at streak line means no activity, mildly reduced growth means hypoactivity and evident reduced mean active complement [9-10]. ESR, HB and White cell counts were made as in [11].

RESULTS

Inflammatory Response:

The patients laboratory biology analysis have shown inflammatory, as elevated ESR and leukocytosis- Respiratory and urinary infectious responses in continuum with ageing processes, Table-2;

Table 2: Laboratory Biology Analysis of the test Patients

Analytical features	Positive/Total	Percentages
Ageing	23/23	100%
Concurrent respiratory infection	14/16	87.5%
Concurrent urinary tract infection	2;16	12.5%
Hb% 11-18	18/19	94.7%
Elevated ESR	12/19	63.2%
Leukocytosis	10/19	52.63%

Bactericidal Power

The patients active bactericidal complement were found as; 1/23 4.3%,3/23,13.01% and 10 /23,43.5% for the incubation period 10,20 and 40 min., respectively. Patient hypoactive bactericidal complement were; 6/23, 6.01%, 11/23, 47.8%, 4/23, 17.4% for the incubation period 10, 20, and 40 minutes respectively. The inactive bactericidal complement were nine for both 20 and 40 minutes, incubation., Table-3.The general trend of bactericidal activity was incubation time dependent as passing from 10 t0 40minutes.

Table-3: Complement bactericidal activity among patients and controls.

Test Groups	Bactericidal effect	10 minutes. incubation	20 minutes. incubation	40 minutes. incubation
Patients	Active Hypoactive Inactive	1/23,4.3% 6/23,26.1% 16/23,69.6%	3/23,13.0% 11/23,47.885 9/23,39.1%	10/23,42.5% 4/23,17.4% 9/23,9.1%
Aged Control	Active Hypoactive	0/5,0.0% 5/5,100%	3/5,60% 2/5,40%	5/5,100% 0/5,0.0%
Adulthood control	Active Hypoactive	0/5,0.0% 5/5,100%	5/5,100% 0/5,0.0%	5/5,100% 0/5,0.0%

DISCUSSION

Normal aged controls were showing 60 % and 100% bactericidal activity at 20 and 40 minutes incubation respectively. This may mean that ageing does not influence complement bactericidal activity suppression. Normal adulthood controls were showing 100% complement bactericidal activity at 20 and 40 minutes incubation. Thus, both aged and adulthood controls were showing 100% complement bactericidal activity at 40 minutes incubation. Thus, the scientific holdings goes to the conclusion that aging does not implicated in complement bactericidal hypo or inactivity as control results tell us. Ageing at 20 found as hypoactive affect bactericidal complement function so that appeared active at 40 minutes. Complement bactericidal hypo-activity was higher in 20 than in 40 minutes. The complement bactericidal hypo-activity at 40 minutes incubation can be attributed to; myopathy, comorbidities complement suppressive effect [12-13].Complement bactericidal inactivity 9/23 can be due either of the followings; consumption in immune-pathogenesis of the disease [6,14,15], myopathy[16], and/or possibly ageing [17,18]. The results, Table-3 appeared to be still novel as compared to current achievements, Table-4.

Table 4: Systemic complement bactericidal activity in cardiomyopathy compared to current update.

Achievement	Reference
Complement deposition in an infarcted myocardial tissue	[5]
High serum levels of the terminalC5b-9 in cases of congestive heart failure	[14]
Chronic immunoglobulin induction of complement activation in the myocardium may contribute to the progression of cardiomyopathy via C5b-9 induced TNF alpha expression in cardio-myocytes	[15]
Complement activation appeared to be involved in various heart diseases	[6]
The complement fraction C5a and its receptors played a key role in cardiac dysfunction during sepsis	[19]
Complosome ,Intracellular complement system	[1]
Complement factor D in pathogenesis of cardiovascular and metabolic diseases	[8]
All the three pathways of complement system activation are implicated in developing cardiac damage	[20]
The terminal C5a-9 may function as a possible biomarker for myocardial tissue damage in acute coronary syndrome	[7]
Systemic complement bactericidal hypo-activity and inactivity induced by cardiomyopathy	This study

Immune Complement Features of Cardiomyopathy;

1. Immunosenescence
2. leukocytosis
3. Elevated ESR
4. Inactive bactericidal Complement
5. Hypoactive bactericidal complement
6. Active bactericidal Complement
7. The complement bactericidal activity was dependent on the initial blood-bacteria interaction –time.

Revisiting Updates

The work cited below; Shnawa et.al.1999.The bacterio-lytic complement and immune-senescence. Baby. Uni. Journal . Pure and Appl.4(3):485-488. It was revisited as; The abstract, introduction, materials and methods , results ,discussion were reframed , updated and cardiomyopathy oriented. Conclusion and immune complement feature new items were added. Compared to the relevant literature it is still of novel importance.

CONCLUSION

The systemic bactericidal complement activity in cardiomyopathy patients have shown to be dependent on the initial blood-bacteria interaction time and the action profile was; in active, hypoactive and active at 37C.

REFERENCES

1. West,E.E.;Kemper,C.2023.Complosome-Intreacellular complement system. Nat. Rev. Nephrol.19,426-439.
2. Mathern,D.R.;Heeger,P.S.2015.Molecules great and small,Clin.J.Am.Nephrol .10(9):1636-1650.doi.10.2215/CJN.062306.
3. Shnawa,I.M.S.2014.Immunology.Ishtar&Dijla, Publishers,Iraq-Jordan[Arabic],30-40.
4. Sarma,J.V.;Ward,P.A.2010.The complement System.Cell Tissue Res.343(1):227-235.doi.10.1007/s0041-010-1034-0.
5. Sherman,S.K.;Collard,C.D.2001.Role of the complement system in ischeamic heart disease;potentials fo pharmacological intervention.Biodrug15(9):595-607.doi.2165/00063030-2001.15090-00004.
6. lappegard K.T.;Garred ,P.;Jonasson,L.et.al.2014.A vital role for complement in heart disease.Mol.Immunol.61(2):126-134.doi.10.1016/j.molimm.2014.06.036.
7. Chiorescu,R.M.;Mocan,M.;Iacobescu,M.et al.2025.Behavior of complement system effectors in chronic and acute coronary artery disease.JCM14(11):1-30.doi.10.3390/jcm14113947.
8. Kong,Y.;Wang,N.;Tong,Z.et.al.2024.Role of complement factor D in cardiovascular diseases. Front.Immunol.15:1453030.doi. 10.3389/fimmu 2024.1453030.
9. Wardlow,A.C.1982.Bactericidal activity of human blood.In,Primose,S.B. ed.Sourcebook of Experiments for Teaching of Microbiology,Academic Press ,paris,493-496.
10. Shnawa,I.M.S.;Mohammed M.Abdul-Razak,Al-Ameidi,B.H.H.1999.The bacteriolytic complement and immune-senescence .Bay.Uni.J.Pure.Appl.Sci.4(3):485-488.
11. Barbara J. ;Bates ,I.2016.Dacie and Lewis. Practical Hematology,12th ed .ElSevier.
12. Muri,L.;Schubart,A.;Thorburn,C.et.al.2022.Inhibition of different complement pathways has varying impacts on the serum bactericidal activity and opsonopha-gocytosis against Haemophilus influenza type b.Front.Immuol.13:1020580.doi .103389 /fimmu.2022.1020580.
13. Sarikonada,G.;von Herrath,M.G.2010.Immunosuppressive mechanisms during viral infectious diseases.Meth.Mol.Biol.667:431-447.doi.10.1007/978-1-60761-869-027.
14. Clark ,D.J.;Cleman,M.W.;Pfau,S.E.2001.Serum complement in congestive heart failure. Am. Heart J.141(4):684-690.doi.1967/mhi.2001.113758.
15. Zwaka ,T.P.;Manolova,D.;Ozdemir,C.et.al.2002.Complement and dilated cardiomyopathy;a role of sublytic terminal complement complex-induced tumor necrosis factor-alpha synthesis in cardiac myocytes.Am.J.Pathol.161(2):449-457.doi.10.1016/s0002-9440(10)64201-0.
16. Perkovic ,V.;Barratt,J.;Rovin,B.et.al.2024.Alternative pathway inhibition with Iptacopan in IgA nephropathy.N.E.J.Med.392(6):531-543.doi.10.1056 /NEJMoa 2410316.
17. Zhao,D.Wang,Y.;Wong,N.D.et.al.2024.Impact of ageing on cardiovascular diseases: from chronological observation to biological insight.FamilySeries. JACC.Asia.4(5):345-358.
18. Zhou,M.;Zhao,G.;Zeng ,Y.et.al.2022.Ageing and cardiovascular disease; Current status and Challenges.Rev.Cardiovasc.Med.23(4):135.doi.j.rcm234135.
19. Fattahi,F.;Ward,P.A.2016.Complement and sepsis-induced heart dysfunction. Mol.Immunol.84:57-64.doi.10.1016/jmolimm.2016.11.012.
20. Hok,K.D.;Rich,H.E.;Shadid,A.et.al.2025.Functional role of the complement immune system in cardiac inflammation and hyperatrophy. Int.J.Mol.Sci.26(20),9931.doi.10.3390/ijms.26209931.