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Association Study of Streptokinase with Cardiovascular Disease

Mahavir Yadav[1], Pushpendra Singh[2] and Atul Pandey[3]

[1] School of Biomolecular Engineering and Biotechnology, Rajiv Gandhi Proudyogiki Vishwavidyalaya, Bhopal, India.
[2, 3] Department of Pathology, GMC Shahdol (M.P.), India.

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Corresponding Author:
Mahavir Yadav

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ABSTRACT

Cardiovascular diseases are the main cause of death on the planet. In 2004, approximately 32% of all women and 27% of all men died (WHO Report 2016) because of this disease. The real reasons for cardiovascular disease are understood as raised circulatory strain, raised plasma lipids, raised plasma glucose, and smoking. Physical inertia and focal weight are common risk factors, others such as smoking, diabetes, hyperlipidemia, and hypertension are also responsible according to some studies. Streptokinase (STK) has been widely used in the treatment of acute myocardial infarction (AMI) by intravenous mixtures in Europe. These preliminary STK treatments have yielded positive results. Streptokinase is an extracellular, single-chain, non-enzymatic, monomeric protein consisting of 440 amino acids, including a 26-amino acid N-terminal peptide that is cleaved during secretion to yield the mature 414 amino acid protein residues of 47 k Da. Streptokinase is naturally produced by Streptococci spp. bacteria, which use this enzyme activator to break up blood clots so that they can spread from the initial site of infection. SK contains three consecutive spaces.

Introduction

Streptokinase is a thrombolytic medication and enzyme. As a medication, it is used to break down clots in some cases of myocardial infarction, pulmonary embolism, and arterial thromboembolism. Streptokinase is a polypeptide derived from Lancefield group C bacteria's beta-hemolytic streptococci. It forms a complex with plasminogen, which then converts to the proteolytic enzyme plasmin. This process results in a cascade that ultimately leads to the lysis of fibrin clots. Streptokinase causes a systemic thrombolytic state that usually resolves within 48 hours (about 2 days) of administration. Streptokinase can be useful in the management and treatment of acute ST-segment myocardial infarction, deep vein thrombosis, pulmonary embolism, arterial thrombosis or embolism, and arteriovenous cannula occlusion. It is in the thrombolytic class of medications.

This investigation studied the indications, action, and contraindications of streptokinase as a valuable agent in the treatment of thrombotic and embolic disorders. A healthy homeostatic system suppresses the development of blood clots in normal circulation but reacts extensively in the event of vascular injury to prevent blood loss. The consequences of disrupted homeostasis cause blood coagulation (thrombus) in the circulatory framework and can cause vascular blockage, resulting in genuine outcomes such as stroke, pneumonic embolism, deep vein thrombosis, intense myocardial localized necrosis, and even death. COVID-19-infected patients have an increased risk for thromboembolic complications, which can cause many organ dysfunctions that lead to premature patient mortality. Our patient was admitted because of sudden chest pain and shortness of breath. To establish the cause of hemodynamic instability, urgent bedside echocardiography was performed, which showed indirect signs of pulmonary embolism with right ventricular dysfunction. The patient was successfully treated with fibrinolysis. The PCR analysis was positive for COVID-19. The molar mass of streptokinase is 47 kDa and is comprised

of 414 amino acid residues (AA). The gem structure reveals that SK contains three consecutive spaces, specifically aminos 1 to 150le agent in the treatment of thrombotic and embolic disorders. A healthy homeostatic system suppresses the development of blood clots in normal circulation but reacts extensively in the event of vascular injury to prevent blood loss. The consequences of disrupted homeostasis cause blood coagulation (thrombus) in the circulatory framework and can cause vascular blockage, resulting in genuine outcomes such as stroke, pneumonic embolism, deep vein thrombosis, intense myocardial localized necrosis, and even death. COVID-19-infected patients have an increased risk for thromboembolic complications, which can cause many organ dysfunctions that lead to premature patient mortality. Our patient was admitted because of sudden chest pain and shortness of breath. To establish the cause of hemodynamic instability, urgent bedside echocardiography was performed, which showed indirect signs of pulmonary embolism with right ventricular dysfunction. The patient was successfully treated with fibrinolysis. The PCR analysis was positive for COVID-19. The molar mass of streptokinase is 47 kDa and is comprised of 414 amino acid residues (AA). The gem structure reveals that SK contains three consecutive spaces, specifically aminos 1 to 150, aminos 151 to 287, and aminos 288 to 411, from the amino to the carboxyl ends, connected by adaptable circles. Fibrinogen, the forerunner of fibrin, was the primary blood coagulation factor. The plasma fibrinogen concentration was linked to coronary disease as well as cerebrovascular and peripheral vascular disease. Thrombosis, the blockage of blood vessels with clots, can lead to acute myocardial infarction and ischemic stroke, both leading causes of death. Other than surgical interventions to remove or bypass the blockage or the generation of collateral vessels to provide a new blood supply, the only treatment available is the administration of thrombolytic agents to dissolve the blood clot.

This article describes a comprehensive review of streptokinase (SK). We discussed the biochemistry and molecular biology of SK, describing the mechanism of action, structures, confirmational properties, immunogenicity, chemical modification, and cloning and expression. The production and physicochemical properties of this SK are also discussed.

Objective of the paper: To confirm at the authors level the existing knowledge of Streptokinase.

Methods

Epidemiological Study population

This study looked at a population of 210 insignificant subjects, including 100 cardiovascular disease patients and 110 ethnically coordinated controls from the focal Indian population. Cases included patients who sought care at the Department of Medicine, GMC Shahdol, SSMC Rewa, and district hospital Shahdol, Sidhi. The World Health Organization (WHO Expert Board 2003) criteria were used to diagnose cardiovascular pain.

Hematologic and Biochemical Analysis:

Biochemical parameters accompanying cardiovascular disease were estimated for both cases and controls subjects. Measurement of Serum levels of Triglycerides (TG), HbA1c, High Density Lipoprotein-cholesterol (HDL-C), Low Density Lipoprotein -cholesterol (LDL-C). Based on spectrophotometric adjustment application and automatic analytic allure analyzer Cobas Integra 400 addition (Roche Diagnostics, Mannheim, Germany), urea was absent. Systolic and diastolic claret pressures were abstinent alert in the adapted arm in sitting position afterwards comatose for at atomic 5-minute application an accepted sphygmomanometer and the boilerplate of the two account was used. Separate serum from the corresponding samples was separated by centrifuging the tubes at 3000 rpm for 12 min. at 4˚C.

Results

Epidemiological Study population-

The descriptive data and comparison of anthropometric and biochemical parameters of cardiovascular disease versus controls are displayed in Table no. 1. The age, sex, BMI, WHR were the parameters. As expected, the cardiovascular disease had notably more elevated amounts of weight of ladies (P=0.0013**), Men (P=0.0016**). BMI of Women (P=0.0172*) and men (P=0.0240*) were significantly associated with cardiovascular disease. Waist circumference in women (P=0.5037) and men (P=0.3260) in like manner WHR in Women (P=0.5263) and Men (P=0.1169) were not related. (See Table no. 1).

Table 1: Comparison of anthropometric parameters of cardiovascular disease and Healthy population

Characteristics	Case (CVD patient) N=100 (75/35)	Control (Healthy population) N=110 (85/40)	P-value
N (Men/Women)	100 (75/35)	110 (85/40)	
Age (years)	50.5 ± 12.5	55.0 ± 14.2	0.7100
Height (m)	160.50 ± 13.40	162.2± 12.000	0.1815
Weight (Kg)			
Women	68.5 ± 5.20	64.8 ± 4.50	0.0013**

Men	71.2 ± 5.60	68.6 ± 3.1	0.0016**
BMI (kg/m^2)			
Women	27.6 ± 3.1	26.1 ± 4.3	0.0172*
Men	28.5 ± 4.7	27.1 ± 3.1	0.0240*
Waist circumference (cm)			
Women	85.5 ± 6.2	84.5 ± 6.7	0.5037 ns
Men	90.0 ± 7.0	89.0 ± 6.0	0.3260 ns
Hip (cm)			
Women	96.8 ± 5.0	96.2 ± 6.0	0.6409 ns
Men	91.0 ± 4.0	90.5 ± 5.5	0.5124 ns
WHR			
Women	0.90 ± 0.05	0.89 ± 0.08	0.5263 ns
Men	0.96 ± 0.05	0.95 ± 0.03	0.1169 ns

(N – Number of individuals in study group.); (-Denotes level of notable change between cardiovascular cases and healthy controls.)*

Hematologic and Biochemical Analysis:

Biochemical test performed in the blood test for various clinical parameters were performed including Post-Prandial Glucose, HbA1C (%), HDL-C(mmol/L), LDL-C (mg/dL), TG (mg/dL), Systolic BP (mmHg), Systolic BP (mmHg), Diastolic BP (mmHg), Blood Urea(mg/dL) Pulse pressure. Measurable investigation was finished by utilizing understudy's t test and p worth acquired recommends the degree of noteworthy changes here. The expressive information and correlation of biochemical parameters of cardiovascular disease versus healthy controls are introduced in Table no.2. True to form the cardiovascular disease had particularly more elevated amounts of LDL-C (P=0.0006***) and TG (P=0.0192*). Huge relationship with cardiovascular infection were seen in Systolic BP (P<0.0001***) and Diastolic BP (P<0.0001***) when this parameter was contrasted with that of healthy control subject. Nominal difference was seen in Pulse weight (P=0.0024**). Postprandial Glucose, blood urea level, HbA1C, and HDL-C level was not significantly different between two groups and all the clinical test outcomes are organized in table no. 2 (See Table no. 2).

Table 2: Comparison of Biochemical and clinical findings of Cardiovascular patients and controls

Characteristics	**Case (CVD patient) N=100 (75/35)**	**Control (Healthy population) N=110 (85/40)**	**P-value**
Post-Prandial Glucose (mg/Dl)	126.7 ±12.4	125.5 ± 10.1	0.4095 ns
HbA1C (%)	6.9 ± 0.8	6.7 ± 0.9	0.0725 ns
HDL-C (mmol/L)	112.2 ± 14.8	109.8 ±11.6	0.1606 ns
LDL-C (mg/dL)	43.1 ± 4.3	41.3 ± 3.7	0.0006***
TG (mg/dL)	131.1 ± 13.2	126.9 ± 14.2	0.0192*
Systolic BP (mmHg)	132.20 ± 8.1	128.8 ± 4.7	P<0.0001***
Diastolic BP (mmHg)	87.1 ± 5.8	82.5 ± 3.0	P<0.0001***
Blood Urea (mg/dL)	9.1 ± 1.6	8.8 ± 1.8	0.1773 ns
Pulse pressure	66.7 ± 18.5	61.1 ± 8.7	0.0024**

(*N – Number of individuals in study group); (*-Denotes level of notable change between cardiovascular cases and healthy controls.)*

Discussion

Cardiovascular Disease (CVD) is a major public health problem in India. The epidemiological alteration plays out abnormally in adapted regions of India because of assorted bread-and-butter development. Disparate relationships amid CVD risk factors are axiomatic in regions that are at adapted stages of epidemiological alteration (Prabhakaran, D et. al. 2016). In this study, we analyzed the association of anthropometric and biochemical parameters with cardiovascular disease. The three above protein Streptokinase are associated with cardiovascular disease. Our work compared with individuals with an accustomed BMI (defined as a BMI of 18.5 to 24.9), lifetime risks for adventure CVD were higher in middle-aged adults in the ample and adipose groups. A portion of middle-aged men and women have aggressive risk ratios for adventure CVD when compared to normal weight. Our Biochemical and clinical findings suggest LDL-C, TG, Pulse pressure; Systolic BP and Diastolic BP were associated with cardiovascular disease. Biochemical assays were performed in the Blood samples

afterward for all the clinical parameters and the findings were tabulated. A statistical assay was done by application student's T test and p values acquired suggest the level of coherent changes.

Conclusion

It can be concluded from table 1 and 2 that most of the anthropogenic and biochemical parameters of the cardiovascular disease patients increase as compared to the healthy controls. So logically all the medications and treatments should be such, which lower these parameters to the healthy level.

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References:

Adinarayana, K., Ellaiah, P. and Prasad, D.S., 2003. Purification and partial characterization of thermostable serine alkaline protease from a newly isolated Bacillus subtilis PE *AapsPharmscitech*, *4*(4), pp.440-448.

Arnesen, H.A.E.B.E., Heilo, A., Jakobsen, E., Ly, B. and Skaga, E., 1978. A prospective study of streptokinase and heparin in the treatment of deep vein thrombosis. *ActaMedicaScandinavica*, *203*(1-6), pp.457-463.

Banerjee, A., Chisti, Y. and Banerjee, U.C., 2004. Streptokinase—a clinically useful thrombolytic agent. *Biotechnology advances*, *22*(4), pp.287-307.

Chadha, S.L., Radhakrishnan, S., Ramachandran, K. and Gopinath, N., 1989. Epidemiological study of coronary heart disease (CHD) in rural population of Gurgaon district (Haryana State). *Indian Journal of Community Medicine*, *14*(4), pp.141-147.

Chadha, S.L., Radhakrishnan, S., Ramachandran, K., Kaul, U. and Gopinath, N., 1990. Epidemiological study of coronary heart disease in urban population of Delhi. *The Indian journal of medical research*, *92*, pp.424-430.

Edwards, Z. and Nagalli, S., 2020. Streptokinase.

Edwards, Z. and Nagalli, S., 2020.Streptokinase.

Kim, D.M., Lee, S.J., Kim, I.C., Kim, S.T., and Byun, S.M., 2000. Asp41-His48 region of streptokinase is important in binding to a substrate plasminogen. *Thrombosis research*, *99*(1), pp.93-98.

Kolev, K., Skopal, J., Nagy, Z. and Machovich, R., 2001. Is streptokinase responsible for the endothelial injury and the platelet activation during fibrinolytic therapy? *Journal of Internal Medicine*, *249*(5), pp.475-476.

Kunamneni, A., Abdelghani, T.T.A. and Ellaiah, P., 2007. Streptokinase—the drug of choice for thrombolytic therapy. *Journal of thrombosis and thrombolysis*, *23*(1), pp.9-23.

Kwong, T.C., Fitzpatrick, P.G. and Rothbard, R.L., 1984. Activities of some enzymes in serum after therapy with intracoronary streptokinase in acute myocardial infarction. *Clinical chemistry*, *30*(5), pp.731-734.

Mitevska, I., Grueva, E., Kandic, E. and Bosevski, M., 2021. Successful treatment of massive pulmonary embolism with cardiogenic shock as a first manifestation of COVID-19 infection. *Macedonian Journal of Anaesthesia*.

Nicolaides, A.N., 2020.Prevention of venous thromboembolism. *Jornal Vascular Brasileiro*, *1*(2), pp.133-170.

Prabhakaran, D., Jeemon, P. and Roy, A., 2016. Cardiovascular diseases in India: current epidemiology and future directions. *Circulation*, *133*(16), pp.1605-1620.

Wang, X., Tang, J., Hunter, B., and Zhang, X.C., 1999. Crystal structure of streptokinase β-domain. *FEBS letters*, *459*(1), pp.85-89.