Available Online at http://www.journalajst.com

ASIAN JOURNAL OF SCIENCE AND TECHNOLOGY

ISSN: 0976-3376

Asian Journal of Science and Technology
Vol. 10, Issue, 10, pp.10276-10277, October, 2019

RESEARCH ARTICLE

LIPOPROTEIN (A) ASSOCIATED MYOCARDIAL INFARCTION IN A 21- YEAR- OLD FEMALE

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ARTICLE INFO

Article History:
Received 15th July, 2019
Received in revised form
29th August, 2019
Accepted 17th September, 2019
Published online 30st October, 2019

Key words:
Lipoprotein (a), Atherosclerosis, Coronary artery Disease, Autosomal Codominant trait, Athero- Embolic Stroke, Statins.

ABSTRACT

Lipoprotein (a) (Lp(a)) excess is an independent risk factor of coronary artery disease (CAD) and have shown wide ethnic variations.Approximately 25% of Indians and other South Asians have elevated Lp(a) levels (≥50 mg/dl). Many studies have pointed out that Lp(a) levels may be a risk factor for cardiovascular diseases (Bandara, 2016). Female sex, family history of CAD, high concentrations of total cholesterol (TC) and low density lipoproteins(LDL) were reported to be associated with high concentration of Lp(a) (Bandara, 2016). We present a 21-year-old female who presented to a tertiary care hospital with typical features of a non ST elevated myocardial infarction (NSTEMI) as a result of a thrombus in left main coronary artery (LMCA) and confirmed witha marginally high low density lipoprotein (LDL) and lipoprotein (a) with a borderline high risk for CAD. All other causes for CAD was excluded. She gives a family history of CAD in her paternal uncle and cousin who died at the age of 28 years after a major coronary infarction. She is currently managed with lipid lowering drugs and dual antiplatelet drugs (DAPT)

***Citation: Kariyawasan, C.C. and Buddhadasa, M.S.* 2019.** "Lipoprotein (a) associated myocardial infarction in a 21- year- old female", *Asian Journal of Science and Technology*, 09, (05), xxxx-xxxx.

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INTRODUCTION

Lipoprotein (a) (Lp (a)) excess is an independent risk factor of coronary artery disease (CAD) and have shown wide ethnic variations.Approximately 25% of Indians and other South Asians have elevated Lp(a) levels (≥50 mg/dl). Many studies have pointed out that Lp (a) levels may be a risk factor for cardiovascular diseases (Bandara, 2016). Female sex, family history of CAD, high concentrations of total cholesterol (TC) and low density lipoproteins(LDL)were reported to be associated with high concentration of Lp(a) (Bandara, 2016). Further, lipid parameters used in assessment and management of risk factors for (CAD) may not reflect accurately the disease or severity if the patients are on pharmacological treatment when compared to Lp(a) (Atukorala, 2002). Cardiovascular diseases (CAD) is the leading cause of mortality in the world (Smith, 2004; Mendis, 2015). Spectrum of disease in CAD includes atherosclerosis related coronary heart disease (CHD), stroke and peripheral vascular disease. The deaths and disease burden due to CAD is 80% and seen in developing countries (Mendis, 2015). The prevalence and mortality rates of CVD are expected to double from 1990 to 2020, and > 80% of this increase is predicted to be in developing countries (Okrainec, 2004).

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Sri Lanka is a South Asian developing country in epidemiological transition. The prevalence of CHD in Sri Lanka is estimated to be 9.3%, with the prevalence of stroke in the Colombo district being 1.04% (Ranawaka, 2016; Katulanda, 2010). CHD and stroke together account for 23% of hospital deaths in Sri Lanka (http://www.health.gov.lk/en).

Case report: A 21 - year - old female was admitted to a tertiary care hospital, with symptoms of chest pain radiating to the neck and left hand and associated with sweating typical of a "heart attack". She gives a history of chest pain 6/12 previously and was treated for dyslipidaemia. Her childhood and adolescence have been uneventful. Her family history reveals the death of her paternal uncle with a coronary heart disease and the death of her paternal cousin at 28 years of age with a STEMI followed by a sudden cardiac arrest. Her physical examination was not significant.Bio chemistry revealed a total cholesterol of 200mg/dl and aLDL value of 105 mg/dl. The other parameters of the lipid profile were normal. The angiogram revealed normal coronaries with LMCA thrombus. She was managed with Abciximab infusion, IV clexane 40mg bd and (DAPT) by the cardiology team and discharged on lipid lowering drugs, Other causes for arterial thrombosis was excluded and lipoprotein (a) level was analyzed and was increased with a level of 28.9mg/dl,which denoted a borderline high risk for coronary artery disease.

DISCUSSION

Lipoprotein(a) (Lp(a)) is a highly atherogenic and heterogeneous lipoprotein that is inherited in an autosomal codominant trait. Lipoprotein(a) (Lp(a)) is an LDL-like molecule consisting of an apolipoprotein B-100 (apo(B-100)) particle attached by a disulphide bridge to apo(a). Lp(a) acts by inhibiting the activation of transforming growth factor (TGF) and contributes to the growth of arterial atherosclerotic lesions by promoting the proliferation of vascular smooth muscle cells and the migration of smooth muscle cells to endothelial cells. It also inhibits plasminogen binding to the surfaces of endothelial cells and decreases the activity of fibrin-dependent tissue-type plasminogen activator. Lp(a) is thought to act as a proinflammatory mediator that augments the lesion formation in atherosclerotic plaques. Elevated serum Lp(a) is an independent predictor of coronary artery disease and myocardial infarction. (Bandara, 2016) Furthermore, Lp(a) levels is considered a marker of restenosis after percutaneous transluminal coronary angioplasty, saphenous vein bypass graft atherosclerosis, and accelerated coronary atherosclerosis of cardiac transplantation. Finally, the possibility that Lp(a) may be a risk factor for ischemic stroke has been assessed in several studies.

Lp(a) levels were found to be associated with increased ischemic stroke risk, primarily among individuals without AF but not in those with AF. (Konstantinos, 2017) since Lp(a) promotes atherosclerosis, elevated Lp(a) levels might be primarily related to athero - embolic stroke, rather than cardioembolic stroke (Konstantinos, 2017). Recent findings suggest that Lp(a) lowering therapy might be beneficial in patients with high Lp(a) levels. A future therapeutic approach could include apheresis in high-risk patients in order to reduce major coronary events. (Lubitz, 2018). There is currently not a consensus for Lp(a) screening. The European Atherosclerosis Society consensus panel recommended screening for anyone at an intermediate or high risk of CVD/CHD with an Lp(a) goal level of < 50 mg/dl (Lubitz, 2018). The panel also advised using niacin as pharmacotherapy for meeting that goal.Currently there is no standard treatment to reduce Lp(a), and no studies have been conducted to assess the impact therapeutic Lp(a) reduction on CAD. The main goal of treatment is to address other known risk factors for CAD, including aggressive LDL lowering. Statins do not lower Lp(a) (Lubitz, 2018). However, statin therapy is essential for patients with increased Lp(a) to mitigate additional CAD risk.It is vitally important to educate about the excessive risk associated with this lipoprotein and the need to avoid the acquisition of other lifestyle-related risk factors such as smoking, excess weight, and physical inactivity to preserve more ideal cardiovascular health in adulthood.

No conflict of Interest.

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