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Assessment of Lipid Profiles in Patients with Down Syndrome in Sudan

MUSAB SHOKRY SALIH¹

Department of Clinical Chemistry Faculty of Medical Laboratory Sciences, Alneelain University Khartoum, Sudan

GADALLAH MODDAWE

Biochemistry Department Faculty of Medicine, Omdurman Islamic University Omdurman, Sudan

SUHAIR ABDALRAHMAN AHMED ABDELKARIM A. ABDRABO

Department of Clinical Chemistry Faculty of Medical Laboratory Sciences, Alneelain University Khartoum, Sudan

Abstract:

Background: Recent researches suggest that children with Down syndrome (DS) have an increased risk of mortality from ischemic heart disease and cerebrovascular disease compared with the normal children.

Objectives: to estimate the concentration of lipid profiles (Total Cholestrol, triglyceride, low density lipoprotein, and high density lipoprotein) in patients with Down syndrome.

Methodology: This is a case control study conducted in Khartoum state during the period from January to May 2015. Blood sample were obtained from 35 patients with DS and 35 apparently healthy as control, with age ranged between 6 to 34 year with matching age and sex with patients. The samples analyzed for lipiDS profile by full automated chemistry analyzer COBAS Model C311.

Result: the mean± SD of serum TC, TG, HDL, LDL in DS respectively were (126.54±35.6), (104.83±35.3), (37±13.5), (76.8±33.05),

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while in control were $(97.7\pm11.4)(83\pm10.8)$, (62.14 ± 7.6) , (21 ± 10.9) . P.values < 0.05.

Conclusions This study confirmed the previously published data which is, significantly increase lipiDS were observed in children with DS compared to non-DS.

Key words: Lipid profiles, Down Syndrome, Khartoum, Sudanese.

Introduction:

Down's syndrome, also known as trisomy 21, is a genetic disorder caused by the presence of all or part of a third copy of chromosome 21.^[1] It is typically associated with physical growth delays, characteristic facial features, and mild to moderate rome are educated in typical school classes, while others require more specialized education.^[7] Some individuals with DS graduate from high school and a few attend post-secondary education.^[8] In adulthood, about 20% in the United States do paid work in some capacintellectual disability.^[2] The average IQ of a young adult with DS is 50, equivalent to the mental age of an 8- or 9-year-old child, but this varies widely.^[3]

DS can be identified during pregnancy by prenatal screening followed by diagnostic testing, or after birth by direct observation and genetic testing. Since the introduction of pregnancies with the diagnosis screening, are terminated.[4][5] Regular screening for health problems common in DS is recommended throughout the person's life. Education and proper care have been shown to improve quality of life. [6] Some children with DS with many requiring a sheltered work environment.^[7] Support in financial and legal matters is often needed.[10] Life expectancy is around 50 to 60 years in the developed world with proper health care. [3][10] DS is the most common chromosome abnormality in humans, [3] occurring in

¹ Corresponding author: mustafahamzah2012@gmail.com

about one per 1000 babies born each year.[2] It is named after John Langdon Down, the British doctor who fully described the syndrome in 1866.[11] Some aspects of the condition were described earlier by Jean-Étienne Dominique Esquirol in 1838 and Édouard Séguin in 1844.[12] The genetic cause of Down syndrome—an extra copy of chromosome 21—was identified by French researchers in 1959.[11] DS (DS) is one of the most common causes of developmental disability with a prevalence of 1 of every 691 live births[13]Persons born with DS are at increased risk for various health conditions, including thyroid disease, leukemia, congenital heart defects, gastrointestinal tract abnormalities, obesity, and diabetes mellitus.[14] Despite this increased risk of chronic disease, life expectancy for individuals with DS has continued to improve with an estimated mean survival approaching 60 years of age [15] Increasing life expectancy along with an elevated risk of obesity and diabetes mellitus in individuals with DS, raise concerns health, in particular, atherosclerotic long-term cardiovascular disease. Obesity and insulin resistance, which are common among individuals with DS, are associated with unfavorable (more atherogenic) lipid profiles, characterized by high triglycerides (TGs) and low high-density lipoprotein (HDL) cholesterol. Previous studies comparing lipid and lipoprotein concentrations in individuals with DS with individuals without DS have produced conflicting results.[16-17] Additionally, it is unclear if individuals with DS have a particularly atherogenic lipid profile before developing obesity and diabetes. Recent data from 2 large epidemiological studies of individuals with DS suggest that they may actually have an increased risk of mortality from ischemic heart disease and cerebrovascular disease compared with the general population.[18-19] The aim of this study was to estimate the concentration of lipid profiles(total Cholestrol [TC], triglyceride [TG], low density lipoprotein [LDL], and high densitylipoprotein [HDL]) in down syndrome.

Materials and Methods

Study population:

This was –case control study conducted in period from January to May 2015-in Khartoum State, Sudan. A total of 35 samples were collected from children with DS as case, and 35 sample from healthy age matched individuals as control, the age of DS between 6 to 34 year and 15 female 20 male. The age for control between 6 to 34 year and 15 female 20 male. Body mass index (BMI) was calculated to exclude obesity. Procedures of BMI Demographic information was obtain via questionnaire from the teachers and families initial visit to the schools of DS. Were weighed without shoes, wearing a light-weight gown on a daily calibrated Scaletronix digital scale (use balance). Standing height was obtained via wall-mounted by meter. Weight and height were measured in triplicate by a trained anthropometrist with the use of research-standard method.[20] All participants were measured by 1 single high. BMI, defined as weight in kilograms divided by height in meters squared, was calculated. Because BMI varies with age and differs by gender, BMI z score was calculated by using age-and genderspecific BMI reference data.[21] After obtaining ethical clearance from an ethical review board and appropriate informed consent from the subjects as well as their parents and guardian.

Including criteria: DS in Khartoum state.

Exclusion criteria: history of congenital cardiac defect requiring open heart surgery- history of intestinal anomalies requiring bowel resection and/or ongoing medical intervention-history of hypothyroidism requiring medication or other chronic conditions known to affect energy balance or growth (including diabetes-and history of cancer and obesity.

Blood Sample:

Blood sample were collected from the all DS who fulfilled the inclusion criteria. After a 12-hour supervised inpatient overnight fast with 24-hour collect the sample by phlebotomy present, blood samples were drawn for total cholesterol, triglyceride, low density lipoprotein, and high density lipoproteinin DS and in healthy control. We were collected 5ml of venous blood was collected from DS and controls. As soon As the blood was collected from DS and controls, it was carried to Lab .the blood was allowed to clot and serum was separated by centrifugation at 5000 rpm for5 minutes. It was used to estimate the main parameters. Analyzed were by the Full Automated chemistry analyzer COBAS Model C311 method.

Statistical Analysis:

Spss for windows version-16(2007) was employed for statistical analysis. The Independent-'t'test procedure was used to compare the mean of the cases and controls. The result presented as mean± standard deviation a P value of .05 was accepted as statistically significant.

Results:

Mean + SD of study population

Table (1)

parameter	DS N=35	Control N=35	P.Value
TC	126.54 ± 35.6	97.7± 11.4	.000
TG	104.83± 35.3	88± 10.8	.010
HDL	37.7± 13,5	62.14± 7.6	.000
LDL	76.8± 33.05	21± 10.9	.000

Discussion

Results from this study suggest that the lipid profile of DS is less favorable than that of their siblings (control), with higher concentrations of TC, LDL, TG and low HDL, as shown in table

(1) there was a significant increase in the mean of total cholesterol in DS when compared to controls group (P<0.05). There was a significant increase in the mean of low density lipoprotien (LDL) in DS when compared to controls group (P<0.05). There was a significant increase in the mean of triglycerides in DS when compared to controls (P<0.05). While that there was a significant decrease in the mean of HDL in DS when compared to controls group (P<0.05). The levels are unlikely to explain the difference in lipid profile in these the DS compared with their siblings, and the question of whether over expression of chromosome 21 directly influences lipid profile can be raised. In a study screening for additional familial combined hyperlipidemia genes, a locus conferring susceptibility to elevated apoB levels was identified on chromosome 21.[22] concentrations of HDL after adjustment for important confounding factors. Our findings of increased TC, LDL, TG and decreased HDL in individuals with DS are similar to 1 previous report by Zamorano et al in Chile in 1991.[23]

Other studies of lipid and lipoprotein concentrations in individuals with DS have produced varying results ranging from no significant difference in cholesterol, triglycerides, and lipoprotein levels between DS and non-DS groups to reports of increased serum cholesterol, triglycerides and oxidatively modified LDL. Our study is the first to use exclusively sibling controls to show all measured lipid parameters to be less favorable in the DS group after adjustment for important confounding factors. laboratory assessment protocols for the general population should also be applied to DS.

Conclusions

This study concludes that there is significantly increased TC, TG, LDL, while significant decrease HDL in DS. It will be important to conduct long-term surveillance of DS to

determine whether these differences in lipid profile translate into increased morbidity and mortality from CVD.

REFERENCES

- 1. Patterson, D. "Molecular genetic analysis of Down syndrome.". *Human Genetics* **126** (1): 195–214. doi:10.1007/s00439-009-0696-8. PMID 19526251.(Jul 2009).
- 2. Weijerman, ME; de Winter, JP. "Clinical practice. The care of children with Down syndrome.". *European journal of pediatrics* **169** (12): 1445–52. doi:10.1007/s00431-010-1253-0. PMID 20632187.(Dec 2010).
- 3. Malt, EA; Dahl, RC; Haugsand, TM; Ulvestad, IH; Emilsen, NM; Hansen, B; Cardenas, YE; Skøld, RO; Thorsen, AT; DaviDS en, EM. "Health and disease in adults with Down syndrome." *TiDS skrift for den Norske laegeforening: tiDS skrift for praktisk medicin, ny raekke* **133** (3): 290–4. doi:10.4045/tiDS skr.12.0390. PMID 23381164(Feb 5,2013).
- Natoli, JL; Ackerman, DL; McDermott, S; EdwarDS, JG (Feb 2012). "Prenatal diagnosis of Down syndrome: a systematic review of termination rates (1995–2011).". *Prenatal diagnosis* **32** (2): 142–53. doi:10.1002/pd.2910. PMID 22418958.
- 4. Mansfield, C; Hopfer, S; Marteau, TM (Sep 1999). "Termination rates after prenatal diagnosis of Down syndrome, spina bifida, anencephaly, and Turner and Klinefelter syndromes: a systematic literature review. European Concerted Action: DADA (Decision-making After the Diagnosis of a fetal Abnormality).". *Prenatal diagnosis* 19 (9): 808–12. doi:10.1002/(sici)1097-0223(199909)19:9<808::aid-pd637>3.0.co;2-b. PMID10521836.
- 5. Roizen, NJ; Patterson, D . "Down's syndrome". *Lancet* (Review) **361** (9365): 1281–89. doi:10.1016/S0140-6736(03)12987-X. PMID 12699967.(April 2003).

- 6."Facts About Down Syndrome". National Association for Down Syndrome. Retrieved 20 March 2012.
- 7. Steinbock, Bonnie (2011). *Life before birth the moral and legal status of embryos and fetuses* (2nd ed.). Oxford: Oxford University Press. p. 222. ISBN 978-0-19-971207-6.
- 8. Szabo, Liz (May 9, 2013). "Life with DS is full of possibilities". *USA Today*. Retrieved 7 February 2014.
- 9. Kliegma, Robert M. "DS and Other Abnormalities of Chromosome Number". *Nelson textbook of pediatrics*. (19th ed.). Philadelphia: Saunders. pp. Chapter 76.2. ISBN 1-4377-0755-6. (2011).
- 10. Hickey, F; Hickey, E; Summar, KL (2012). "Medical update for children with DS for the pediatrician and family practitioner.". *Advances in pediatrics* **59** (1): 137–57. doi:10.1016/j.yapd.2012.04.006. PMID 22789577.
- 11. Evans-Martin, *Down syndrome*. New York: Chelsea House. p. 12. ISBN 978-1-4381-1950- \Box f.Fay(2009).
- 13. Parker SE, Mai CT, Canfield MA, et al. National Birth Defects Prevention Network Updated National Birth Prevalence estimates for selected birth defects in the United States, 2004-2006. Birth Defects Res A Clin Mol Teratol. 2010;88(12):1008–1016 [PubMed]
- 14. Roizen NJ, Patterson D. Down's syndrome. Lancet. 2003;361(9365):1281–1289 [PubMed]
- 15. Glasson EJ, Sullivan SG, Hussain R, Petterson BA, Montgomery PD, Bittles AH. The changing survival profile of people with Down's syndrome: implications for genetic counselling. Clin Genet. 2002;62(5):390–393 [PubMed]
- 16. Murdoch JC, Rodger JC, Rao SS, Fletcher CD, Dunnigan MG. Down's syndrome: an atheroma-free model? BMJ. 1977;2(6081):226–228 [PMC free article] [PubMed]
- 17. Nishida Y, Akaoka I, Nishizawa T, Maruki M, Maruki K. Hyperlipidaemia in patients with Down's syndrome. Atherosclerosis. 1977;26(3):369–372 [PubMed]

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- 18. Hill DA, Gridley G, Cnattingius S, et al. Mortality and cancer incidence among individuals with Down syndrome. Arch Intern Med. 2003;163(6):705–711 [PubMed]
- 19. Day SM, Strauss DJ, Shavelle RM, ReynolDS RJ. Mortality and causes of death in persons with DS in California. Dev Med Child Neurol. 2005;47(3):171–176 [PubMed]
- 20. Cameron N. The methoDS of auxologic anthropometry. In: Falkner F, Tanner JM, eDS. Human Growth. 2nd ed. New York, NY: Plenu Press; 1986:3–43
- 21. Rosner B, Prineas R, Loggie J, Daniels SR. Percentiles for body mass index in U.S. children 5 to 17 years of age. J Pediatr. 1998;132(2):211–222
- 22. Pajukanta P, Terwilliger JD, Perola M, et al. Genomewide scan for familial combined hyperlipidemia genes in Finnish families, suggesting multiple susceptibility loci triglyceride, cholesterol, and apolipoprotein B levels. Am J Hum Genet. 1999:64(5):1453–1463
- 23. Zamorano A, Guzmán M, Aspillaga M, Aventine A, Gatica M. [Concentrations of serum lipiDS in children with Down's syndrome]. Arch Biol Med Exp (Santiago). 1991; 24(1):49–55