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Antidiabetic efficacy of *Habbul aas* (*Myrtus communis*): Case report

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Abstract
Type 2 diabetes mellitus (DM) is a chronic metabolic disorder in which prevalence has been increasing steadily all over the world. As a result of this trend, it is fast becoming an epidemic in some countries of the world with the number of people affected expected to double in the next decade due to increase in ageing population, thereby adding to the already existing burden for healthcare providers, especially in poorly developed countries. Worldwide, people are less physically active and this has become a leading risk factor for ill health as millions of lives are lost per year due to physical inactivity which leads to a host of disorders and DM is one of the four major NCDs (Cancer, Diabetes, Chronic respiratory and Cardiovascular diseases) which account for most of the disease burden and premature mortality in the European Region. The WHO has projected that diabetes will be the 7th cause of death by 2030. A patient 34years-old Muslim woman was screened as diabetis mellitus. Patient attended National institute of Unani medicine with complain of Polyuria, polyphagia and burning sensation in a feet. A single drug was used in *powdered* form for 60 days.
The results was assessed after taking unani drug suggested reduction in serum FBS and PPBS level in 60 days. In this case report, we suggested a significant result of *Safoofe Habbul Aas* for Type 2 diabetes mellitus (DM). By this study, this single unani drug can be used as boon choice therapy for Type 2 diabetes mellitus (DM).

Keywords: Type 2 diabetes mellitus; diagnosis; management; *Habbul Aas*

Introduction
Diabetes mellitus (DM) is probably one of the oldest diseases known to man. It was first reported in Egyptian manuscript about 3000 years ago [1]. In 1936, the distinction between type 1 and type 2 DM was clearly made [2]. Type 2 DM was first described as a component of metabolic syndrome in 1988 [3]. Type 2 DM (formerly known as non-insulin dependent DM) is the most common form of DM characterized by hyperglycemia, insulin resistance, and relative insulin deficiency [4]. Type 2 DM results from interaction between genetic, environmental and behavioral risk factors [5, 6]. People living with type 2 DM are more vulnerable to various forms of both short- and long-term complications, which often lead to their premature death. This tendency of increased morbidity and mortality is seen in patients with type 2 DM because of the commonness of this type of DM, its insidious onset and late recognition, especially in resource-poor developing countries like Africa [7].

Lifestyle, genetics, and medical conditions
Type 2 DM is due primarily to lifestyle factors and genetics [8]. A number of lifestyle factors are known to be important to the development of type 2 DM. These are physical inactivity, sedentary lifestyle, cigarette smoking and generous consumption of alcohol [9]. Obesity has been found to contribute to approximately 55% of cases of type 2 DM [10]. The increased rate of childhood obesity between the 1960s and 2000s is believed to have led to the increase in type 2 DM in children and adolescents [11]. Environmental toxins may contribute to the recent increases in the rate of type 2 DM. A weak positive correlation has been found between the concentration in the urine of bisphenol A, a constituent of some plastics, and the incidence of type 2 DM [12]. Obesity (which is an independent risk factor for type 2 DM) is strongly inherited [13]. Monogenic forms like Maturity-onset diabetes of the young (MODY), constitutes up to 5% of cases [14]. There are many medical conditions which can potentially give rise to, or exacerbate type 2 DM. These include obesity, hypertension, elevated cholesterol (combined hyperlipidemia), and with the condition often termed metabolic syndrome (it is also known as Syndrome X, Reaven's syndrome) [15].

Other causes include acromegaly, Cushing's syndrome, thyrotoxicosis, pheochromocytoma, chronic pancreatitis, cancer, and drugs [16]. Additional factors found to increase the risk of type 2 DM include aging [17], high-fat diets, and a less active lifestyle [18].

Pathophysiology

Type 2 DM is characterized by insulin insensitivity as a result of insulin resistance, declining insulin production, and eventual pancreatic beta-cell failure [19, 20]. This leads to a decrease in glucose transport into the liver, muscle cells, and fat cells. There is an increase in the breakdown of fat with hyperglycemia. The involvement of impaired alpha-cell function has recently been recognized in the pathophysiology of type 2 DM [21]. As a result of this dysfunction, glucagon and hepatic glucose levels that rise during fasting are not suppressed with a meal. Given inadequate levels of insulin and increased insulin resistance, hyperglycemia results. The incretins are important gut mediators of insulin release, and in the case of GLP-1, of glucagon suppression. Although GIP activity is impaired in those with type 2 DM, GLP1 insulinotropic effects are preserved, and thus GLP-1 represents a potentially beneficial therapeutic option [21].

Diagnostic Criteria

World Health Organization recommendations-2006 for the diagnostic criteria of DM and intermediate hyperglycaemia:
DM should be diagnosed if one or more of the following criteria are met:

- Fasting plasma glucose ≥ 7.0 mmol/L (126 mg/ dl)
- Two-hour plasma glucose ≥ 11.1 mmol/L (200 mg/dl) following a 75g oral glucose load.
- Impaired Glucose Tolerance (IGT) should be diagnosed if both of the following criteria are met:
- Fasting plasma glucose < 7.0 mmol/L (126 mg/ dl)
- Two-hour plasma glucose 7.8-11.1 mmol/L (140 -200 mg/dl) following a 75g oral glucose load.
  Impaired Fasting Glucose (IFG) should be diagnosed if both of the following criteria are met:
- Fasting plasma glucose 6.1-6.9 mmol/L (110-125 mg/ dl)
- Two-hour plasma glucose < 7.8 mmol/L (140 mg/dl) following a 75g oral glucose load [22].

Diagnostic approach of Unani Physicians

The ancient Atibbas were of the belief that if the patient complains of intense thirst, the back and region of the loin have a feeling of warmth and the other symptoms of Su-e-Mizaj Haar are dominant but there is no change of colour or any burning sensation in the Qarura (urine) then this due to Haraarat-e-Gurda. On the other hand if the Qarura exhibits altered colour and the warmth or heat is felt in the right side of the abdomen then it is due to the Hararat-e-Jigar.
Inspite of the thirst that has been exhibited by the patients, if the symptoms of Su-e-Mizaj Barid is dominant, the body starts to become more and more weak and lean, the strength of the body and desire for food declines and the patient gets relieved by consumption of warm water for a while and at the end the disease reaches its final stage in the form of Istesqa (Ascites) then it was concluded that the disease was due to Su-e-Mizaj Barid [23].

Concept of Diabetes in Unani medicine

It is characterized by excessive thirst, excessive urination, presence of sugar in urine, increased appetite and gradual loss of body weight.10 which has been explained by the physicians of today in the form of lassitude, progressive weakness and ready fatigue [23]. According to the Unani concept the virtual cause of Ziabetus is the fire like heat in the kidneys where Rutubat in the form of water is absorbed in large amounts and this cannot be filtered as the kidneys absorb water beyond their capacity and they are not able to retain the volume as a result of which the kidneys just get rid of the water by pushing it towards the bladder. The Quwate Masika weakens because of the intense heat of the kidneys [24] the patient is thirsty helplessly consuming water which is passed out through the kidney immediately. It is similar to Zalqul Meda wal Ama [25], in which the current recommendation is to start the Metformin therapy as soon as the patient is diagnosed with DM [26]. Metformin is used as a first line therapy irrespective of body weight [27]. It is not prudent to watch out for achieving glycemic targets through weight management and exercise, all the other co-morbid conditions and a number of factors seen in a patient should be considered. Dosage: Low dose of 500 mg twice daily and a maintainance dose of 1 gm twice daily. Contraindications: Impaired hepatic function, alcoholics and in lactic acidosis.

Material and methods

For this study, a 32-year-old woman was selected who visited to the OPD at National Institutes of Unani Medicine on December 12, 2016, with the complain of neck pain. The patient had no history of Hypertension, diabetes, tuberculosis, Thyroid disorder and ischemic heart disease. Whereas Fatigue, Weight gain and neck pain present since last 6 months and also complaining of polyuria since 3 months but not taking medicine for all this complains.
On Examination, the patient was conscious, oriented to time, place and person, with no evidence of any focal neurological deficit. She was afebrile; pulse rate 92/min, regular; blood pressure 130/80 mmHg; respiratory rate 18/min, weight – 91kg. All other systemic examination were normal. No history of swelling and stiffness in the neck.
Our patient was not a known case of Type 2 DM, and she did not give any history of taking anti-diabetic drug instead she was admitted and diagnosed as cervical spondolysis and for that treatment is given. But as per the hospital protocol after doing all routine investigation, she was accidentally diagnosed with type 2 DM. A confirmatory test with HbA1c report was done to start the treatment of the newly diagnosed patient.
Based on all investigations, Diagnosis of DM was made.
BSL –F= 128; PP= 171
HbA1C = 7.16

Treatment given

Safoofe Habbul Aas [28] = 3gm BD daily before meals for 60days from 20 Dec 2016 with twice a month follow up. Patient responded within 2 weeks with decreased in the frequency of micturition. This follow-up is done twice a month for compliance of medicine and to see for symptomatic relief.
Reviewed After 60 days, the patient showed decreased in Blood sugar levels after taking Unani medicine (*Safoofe Habbul Aas* = 3gm BD daily before meals) along with decreased in the frequency of micturition.

Discussion

As it is known that there is no complete cure for Diabetis, the pharmacological treatment options are very much limited, and having some side effects of modern medicine, hence now days, the Conventional treatment like Unani medicine etc. becomes most effective. In USM, there is wide-ranging dosage formulations, which cure Diabetis. In classical text, it is mentioned that, ancient physician treats Zaibetus. In Unani System of Medicine (USM) the word "Diabetes" is derived from Greek language of "Ziabetus", which means, "to run through" or "Siphon", is characterized by hyperglycemia, polyuria, polyphagia, polydipsia and gradual loss of body weight. Therefore, the present study has been designed to validate the effect of the *Safoofe Habbul Aas* [28] in DM. According to Unani literature, this drug have astringent property and most of the scientific reports possess that *Mytrus communis* have anti diabetic activity along with anti oxidative properties also. It has been suggested that oxidative stress constitutes the key role in the pathogenesis of different diabetic complication [29]. Blood sugar profile was performed after 2 months. It is a single patient case report.

The result of this study suggest that *Safoofe Habbul Aas* is beneficial in DM, however the finding cannot be generalized, so further studies are recommended on large number of patients to established the effect of *Safoofe Habbul Aas* and also the long term follow up should be done to know the long term effect of *Safoofe Habbul Aas* in DM.

Conclusion

The observations indicated the anti diabetic activity of *Safoofe Habbul Aas* in Unani literature. These results are encouraging and on this basis, further study may be carried out to elaborate clinical trial.

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Conflict of interest: None

References

1. Ahmed AM. History of diabetes mellitus. Saudi Med J. 2002; 23(4):373-78.
2. Diabetes mellitus history- from ancient to modern times. Available at http:// science.jrank.org/pages/2044/Diabetes-Mellitus.html. (Accessed on 22nd September, 2017)
3. Patlak M. New weapons to combat an ancient disease: treating diabetes. FASEB J. 2002; 16(14):1853.
4. Maitra A, Abbas AK. Endocrine system. In: Kumar V, Fausto N, Abbas AK (eds). Robbins and Cotran Pathologic basis of disease (7th ed). Philadelphia, Saunders, 2005, 1156-1226.
5. Chen L, Magliano DJ, Zimmet PZ. The worldwide epidemiology of type 2 diabetes mellitus: present and future perspectives. Nature reviews endocrinology. Available at: www.nature.com/uidfinder. (Accessed 22nd December 2011)
6. Genetic basis of type 1 and type 2 diabetes, obesity, and their complications. Advances and emerging opportunities in diabetes research: a Strategic Planning report of the DMICC. www2.niddk.nih.gov/NR. (Accessed 22nd December 2011).
7. Azevedo M, Alla S. Diabetes in sub-saharan Africa: kenya, mali, mozambique, Nigeria, South Africa and zambia. Int J Diabetes Dev Ctries. 2008; 28(4):101-108.
8. Ripsin CM, Kang H, Urban RJ. Management of blood glucose in type 2 diabetes mellitus. Am Fam Physician. 2009; 79(1):29-36.
9. Hu FB, Manson JE, Stampfer MJ, Colditz G, Liu S, Solomon CG *et al*. Diet, lifestyle, and the risk of type 2 diabetes mellitus in women. N Engl J Med. 2001; 345(11):790-797.
10. Prevalence of overweight and obesity among adults with diagnosed Diabetes United States, 1988-1994 and 1999-2000"Centers for Disease Control and Prevention (CDC) (November 2004) MMWR. Morbidity and Mortality Weekly Report. 53(45):1066-68.
11. Barlow SE. The Expert committee. Expert committee recommendations regarding the prevention, assessment and treatment of childhood and adolescent overweight and obesity: Summary report. Paediatrics. 2007; 120:S164-S192.
12. Lang IA, Galloway TS, Scarlett A, Henley WE, Depledge M, Wallace RB *et al*. Association of urinary bisphenol A concentration with medical disorders and laboratory abnormalities in adults. JAMA. 2008; 300(11):1303-1310.
13. Walley AJ, Blakemore AI, Froguel P. Genetics of obesity and the prediction of risk for health. Hum Mol Genet. 2006; 15(2):124-130.
14. Camastra S, Bonora E, Del Prato S, Rett K, Weck M, Ferrannini E. EGIR (European Group for the Study of Insulin Resistance). Effect of obesity and insulin resistance on resting and glucose-induced thermogenesis in man. Int J Obes Relat Metab Disord. 1999; 23(12):1307-13.
15. Alberti KG, Zimmet P, Shaw J. IDF Epidemiology Task Force Consensus Group. The metabolic syndrome–a new worldwide definition. Lancet. 2005; 366(9491):1059-1062.
16. Powers AC. Diabetes mellitus. In: Fauci AS, Braunwauld E, Kasper DL, Hauser SL, Longo DL, Jameson JL, Loscalzo J (eds). Harrison's Principles of Internal Medicine.17th ed, New York, McGraw-Hill, 2008, 2275-2304.
17. Jack Jr L, Boseman L, Vinicor F. Aging Americans and diabetes. A public health and clinical response. Geriatrics. 2004; 59(4):14-17.
18. Lovejoy JC. The influence of dietary fat on insulin resistance. Curr Diab Rep. 2002; 2(5):435-440.
19. Kahn CR. Banting Lecture. Insulin action, diabetogenes, and the cause of type II diabetes. Diabetes. 1994; 43(8):1066-1084.
20. Robertson RP. Antagonist: diabetes and insulin resistance–philosophy, science, and the multiplier hypothesis. J Lab Clin Med. 1995; 125(5):560-564. discussion 565.
21. Fujioka K. Pathophysiology of type 2 diabetes and the role of incretin hormones and beta-cell dysfunction. JAAPA, 2007, 3-8.
22. Anonymous. World Health Organization recommendations, 2006.
23. Khan MA. Akseer-e-Azam (Al-Aksir) (Urdu translation by M. Kabiruddin,) New Delhi: Idarae Kitab-al-Shifa, 2011, 705-706.
24. Razi Al-Hawi Fit Tib Z. 10 CCRUM. New Delhi.

Ministry of Health &Family Welfare, Govt of India, 2010, 181-87.
25. Sina Al-Qanoon Fit-Tib I. (Urdu translation by Kanturi G.H). New Delhi: Idara Kitab-al-Shifa. 2010; 2(3):248-49.
26. Diabetes Mellitus & Hypoglycemia. Chapter 27. CMDT, 2015.
27. Pearson ER, Mc Crimmon RJ. Diabetes mellitus. 22nd edition. Davidsons Principles and Practice of Medicine. Churchill Livingstone Elsevier. Printed in China, 2014.
28. Ghani Khazainul Advia N. New Delhi: Idara Kitabul Shifa, 2010, 642.
29. Alipour G, Dashti S, Hosseinzadeh H. Review of pharmacological effects of *Myrtus communis* L. and its Active Constituents. Phytotherapy research. 2014; (28):1125-36.