IJPSR (2018), Volume 9, Issue 12 (Research Article)

INTERNATIONAL JOURNAL OF PHARMACEUTICAL SCIENCES AND RESEARCH

Received on 26 April, 2018; received in revised form, 02 July, 2018; accepted, 09 July, 2018; published 01 December, 2018

TREATING DIABETES AND RHEUMATOID ARTHRITIS USING PERSONALIZED MEDICATION

S. U. Dani and P. A. Kulkarni *

Department of Pharmaceutics, SVKM's NMIMS, School of Pharmacy & Technology Management, Shirpur, Dhule - 425405, Maharashtra, India.

Keywords:

Autoimmune Diseases, Biomarkers, Diabetes mellitus, Nutrigenomics, Pharmacogenetics, Personalized medicine

Correspondence to Author:
Dr. Parag. A. Kulkarni

Associate Professor, Department of Pharmaceutics, SVKM's NMIMS, School of Pharmacy & Technology Management, Shirpur, Dhule - 425405, Maharashtra, India.

E-mail: Parag.kulkarni@nmims.edu

ABSTRACT: As in the cases of non-communicable but chronic and autoimmune disorders like diabetes, cancer, anemia and rheumatoid arthritis are growing threat to humans. They depend on the genetic information and the genome sequence of the patients and other factors like environmental factors. In most of the cases, the pattern of the disease, *i.e.* pathogenesis is different for every individual, though phenotypic expression is same. Hence, there is a greater risk for the patient having the genes which are likely to cause above mentioned diseases and side effects or adverse drug reactions due to the therapy available. This occurs due to only one reason that every individual will not react to a drug in a same way. For minimizing these unwanted side effects or adverse reactions which are disease related and for giving patients a better and a bit healthy life, personalizing the medication is essential. This personalization involves the study of phenotypic and genotypic information of the patient and then planning the 3D's *i.e.* choice of drug, dose and diet is must. As the diabetic patients worldwide have risen steadily over the past three decades, one of the reasons behind such rise in autoimmune diseases may be the availability of very few specific drugs to treat them. Personalize medication is a hot topic nowadays. In this article we are discussing diabetes and rheumatoid arthritis management by studying the genotype and molecular profile of the patients.

INTRODUCTION: Personalized medication is the modification of the medical treatment to the individual characteristic of each patient and it shows emphasis specifically on the use of genetics and genomics [1]. It also includes any other biological information which predicts the risk of disease and what will be the reaction of the patient to the treatment.

QUICK RESPONSE CODE

DOI: 10.13040/IJPSR.0975-8232.9(12).5424-30

Article can be accessed online on: www.ijpsr.com

DOI link: http://dx.doi.org/10.13040/IJPSR.0975-8232.9(12).5424-30

Assessment is based on risk which comes along with sex, age. *e.g.* Menopausal status of women (sex based), risk of diabetes based on both age and sex, the risk of high blood pressure or high cholesterol level based on work type *i.e.* sedentary work style, it also includes specific biomakers like lipoprotein which are actually helpful in the assessment of risk of heart diseases or stroke in individuals [1].

The human genome has a unique variation in individual. It is a medical process which segregates the patients into different groups with medical treatment decision, practices, dosage course and risk of disease. Personalized medication can also be referred as a procedure which separates the patients

into various groups with different medical decisions, practice, dosage regimen, and that the products being altered for the individual patients on the basis of their predicted responses or risk of causing disease [1].

It is a rising concept for treating disease involving the determination of specific information regarding to a particular patient and then prescribing a treatment according to genetics and genomics that is specific for that patient [2, 3].

History: In ancient times, for the best course of treatment for each patient suffering from some genomics and genetics related diseases Hippocrates studied an assessment of the four humours- blood, phlegm, yellow bile and black bile. It was not until the middle of the 20th century, but when the world is getting the deeper molecular understanding of disease, and then at the turn of 21st century, with the modern techniques of sequencing the human genomes scientists were able to develop the tools to truly personalize diagnosis and treatment [1]. Now it is an emerging concept to provide the healthy life to the human being.

Why we are going to discuss only these two diseases?

The purpose of discussing these two diseases like Diabetes and Autoimmune disease (Rheumatoid Arthritis).

Diabetes: Diabetes is one of the most common and costly chronic condition worldwide. As we all know that diabetes is a non-communicable but rapidly growing disease [4]. Diabetes cases are raised due to many reasons such as changes in lifestyle, lots of sedentary work, stress, genetic makeup, patient's family history, race groups, health and environmental factors. Now a day's diabetes is a dangerous condition to human health and economic growth. Treatment of every disease is associated with some drawbacks as the diabetes treatment is also associated with potential side effect like hypoglycemia, weight gain, stomach upset, the risk of liver diseases, anemia risk, bloating and diarrhea, acute hypoglycemia, increased morbidity and increase in mortality rates [5]. Concurrent hypoglycemic episodes associated with increased risk of damage to the central nervous system and increased incidences of dementia [6].

Diabetes is a range of conditions associated with peripheral neuropathy, cardiovascular disease, blindness and even death characterized by chronic high blood glucose levels. Current treatments and strategies are not able to maintain the fluctuations in blood glucose levels, which lead to variations in severity of that disease [4].

Autoimmune Diseases: As we know there is no cure on autoimmune disorders, only symptoms can be managed with the help of drugs which are available in the common drug treatment of autoimmune disease like rheumatoid arthritis, asthma, pemphigus *etc*. The drugs used are monoclonal antibodies, TNFα, corticosteroids *etc*. Near about 80 autoimmune diseases, conditions that cannot be cured. Ideally to reduce many problems, finding of right drug to right patient is a prime requirement [7] to recognize the drug based on genetic and molecular profile of the patient. Simple drugs can lead to the development of serious new conditions which make previous disease more complicated. Because of heterogeneity of the autoimmune disease, there is no universal consensus associated with the treatment.

Reasons: Why Personalized Medication is an Emerging Concept?

1. Regulatory prescribes treatment to the patients by trial and error basis and one size fits all approaches, but not everyone responds in the same way.
2. A personalized medication helps us to predict the drug with the minimum side effects for individual patients.
3. It can improve the quality of treatment service and decrease the cost of the treatment at the same time.
4. Same symptoms: Same treatment- Patients diagnosed with same disease not necessarily react same to the same treatment. It is also considerable that same disease may undergo through different pathophysiology.
5. Recent major progress in biomarkers research.

Limitations of Personalized Medications: No medical school curriculum includes the training regarding to the use and implementation of the personalized medication by the healthcare workforce (including physicians).

There is a threat in public that their genetic data is not in a private place so they do not willingly take full participation in personalized medicine, research or clinical care, unless full genetic privacy is put in place.

Linking between patient information to genomic research and healthcare IT sections and maintaining the electronic medical records is also critical job.

Treatment:

Diabetes: Diabetes is not a curable disease; lifelong treatment along with regular exercise is the only option. The definition of personalized medication for diabetes (PMFD) is "the use of information about the genetic makeup of a person with diabetes to tailor strategies for preventing, detecting, treating, or monitoring their diabetes" [8]. Development of a strategic treatment model and formulation of a personalized medication treatment plan for the purpose of lowering and maintaining the BGC (Blood Glucose Concentration) within the ADA (American Diabetes Association) specified range (between 70 to 130 mg/dl).

Type 1 diabetes is caused due to an autoimmune attack on the insulin producing pancreatic β-cells. Type II diabetes is caused by decreased sensitivity to insulin, which causes difficulty for muscles and adipose tissue to utilize glucose [4].

Monitoring of the Blood Glucose Concentration (BGC): Disciplinary actions are needed to manage the diabetic disease condition. Motivational interviewing techniques help patients to gain his commitment to adhere to the decided therapy. A proper combination of diet, exercise, medication along with insulin therapy is recommended by the American diabetes association (ADA) to control and to lower the blood glucose level to a healthy level [9].

Treatment to Control BGC:

The PMFD Involves Four Stages: Identification of genes and biomarkers which are responsible for diabetes and obesity.

As the genes and biomarkers are the causative agents, hence utilization of genetic tests and biomarkers are helpful in diagnosis and monitoring the course of diabetes.

Risk prone patients are identified after genetic testing for diabetes and directed for preventative measures such as lifestyle modification or medication for delaying or preventing disease. The efficiency of the drug treatment can enhanced if we are able to determine where to target the genetic and nutritionally determined drug in populations of patients causing diabetes. Setting aside and managing the available resources to prevent or diagnose the diabetes/ obesity phenotype in high risk individuals whose risk is based on their genotype.

Selection of Individual Therapies for Patients: Selection of therapies involves a decision on which drug of the same generation is to prescribe, what the dose of drug to use, according to the severity of disease and age, body weight related factors and which diet to prescribe can also be the part of the treatment. Also involves the decision of drug which causes least side effects or toxicity.

Measurement of circulating biomarkers of diabetes, for example, some novel plasma biomarkers like apoA4, CD5L, Hb1C, C1QB and IBP3 to monitor the response for prediction of rapid decline in renal function which recognizes the risk and help in prevention or in planning of strategic treatment [23].

Five Main Types of Research Initiatives:

1. Pharmacogenetics: It is a branch of science which explains the response of people for the same drug treatment in different ways. It involves testing of candidate genes for drug patient interactions [10].

2. Pharmacogenomics: [22] Pharmacogenomics can be a test not only for genes, but also for the expression of genes over time. Genomic information used to study individual responses to drugs. A person's DNA content (which comprises of genes) cannot change over time, but the expression of genetic content, *i.e.* RNA content (which reflects how much the gene is being utilized) can change over time [11].

3. Nutrigenomics: It is an approach which utilizes identification of action or response of genes towards food and then planning the diet accordingly so that advantages can be taken out of these responses.

4. Biomarkers: Biomarkers predict, diagnose or monitor the disease. Ex. Type 1 diabetes can be predicted by measuring Autoantibodies and type 2 can be predicted by measuring adipokinase [12, 13].

5. System Biology: System biology is involved in the measurement of the interaction between the biological components in the systems and how these interactions give rise to the functions and behaviors of that system. This system utilizes tools such as transcriptomics, proteomics, metabolomics and glycomics.

Persons with T1D and advanced T2D must frequently check their blood glucose level and inject insulin multiple times per day and due to this reason, recent developments in the area of creating devices which automate day to day control of diabetes. These devices are often called as artificial pancreas. These artificial pancreases can respond to the physical activity and change the blood glucose concentration by injecting fast acting insulin analogue and alerting patients when they must take their medications. These are also referred to as biomedical devices in modern electronic evolution.

Applications of Personalized Medication for Diabetes: [3]

TABLE 1: PREVENTION OF DIABETES MELLITUS WITH THE HELP OF PERSONALIZED MEDICATION

Prevention	
Observation	**Responses**
Genetic testing for the identification of the people who are at high risk for type I and II diabetes	Immune therapy for prevention of onset of type 1 diabetes mostly because of an autoimmune attack on pancreatic cells *E.g.* Vaccine to prevent type I
Genetic testing for the identification of the people who are at high risk for diabetic nephropathy	Lifestyle modification and drug therapy to prevent type II diabetes, but this is as a preventive measure
Genetic testing for the identification of people who are at high risk of obesity	Drug therapy to prevent nephropathy

TABLE 2: DIAGNOSIS OF DIABETES MELLITUS WITH THE HELP OF PERSONALIZED MEDICATION

Diagnosis	
Input	**Responses**
Neuropathy and NH2-terminal fragment of the brain Natriuretic peptide which acts as biomarker	Early detection of diabetic neuropathy with the help of genetic studies, tight control of DM with the help of drug therapy

TABLE 3: TREATMENT OF DIABETES MELLITUS WITH THE HELP OF PERSONALIZED MEDICATION

Treatment	
Input	**Responses**
Genetic testing, demonstrating subpopulation within type 1Abnormal insulin molecule Abnormal release (LADA)	Type 1: Treatment such as intensive insulin therapy, immune therapy, or gene therapy, which relates to the causative agent can be ensured by early detection of disease to avoid complications
Classification into subpopulation on the basis of new-onset and ketosis-prone patients	
Classification into subpopulation on the basis of autoimmune diabetes in late-onset type and type 1 diabetes	Type 2: For proper islet function early suggestion of thiazolidinediones or insulin
Genetic testing, demonstrating subpopulations within type 2	LADA: Early initiation of insulin treatment to maintain euglycemia in patients with this diagnosis
Mechanisms of the cause of type 2 must be understood and presented	
Response of drugs for diabetes by gene, *e.g.*, metformin and sulfonylureas	MODY-sulfonylurea therapy and focus on specific vascular risks
Differentiation of MODY from type 2 DM	Neonatal diabetes: switch from insulin to sulfonylureas
Genetic testing of neonatal diabetes	Diet for type 2 diabetes
Nutrigenomics studies of food impact	Exercise therapy for type 2 diabetes

TABLE 4: MONITORING OF DIABETES MELLITUS WITH THE HELP OF PERSONALIZED MEDICATION

Monitoring	
Input	**Response**
Type 1: Autoantibodies and antigens, cytokine levels, and inflammatory serum markers which are biomarkers for autoimmunity	Type 1: Immune intervention. Technology: continuous glucose monitoring, insulin pump therapy, and artificial pancreas
Type 2: Lipids, C-reactive protein: cytokines, retinol-binding protein, and other inflammatory markers, New markers of glycemia in addition to glucose and hemoglobin A1c	Type 2: Anti-inflammatory therapy with thiazoldinediones or statins
Glycemic variability, Advanced glycated end products, 1,5-Anhydroglucito, Glycated Albumin Exhaled methyl nitrate, Physiologic hypoglycemia detection, Smart Shirt and Smart Glove for detecting diabetic autonomic neuropathy	
Abbreviations used: MODY: maturity onset diabetes of the young; LADA: latent autoimmune diabetes; DM: diabetes mellitus;	

Autoimmune Diseases: According to the survey, there are about 80 autoimmune conditions that cannot be cured [7]. As a treatment for a majority of autoimmune diseases, there are only a few medications like immunosuppressant, TNF-α *etc*. It is not necessary that a single drug is effective in every patient and there is great variability in toxicity and price.

Rheumatoid Arthritis: RA is the most common autoimmune provocative *i.e.* inflammatory disease [7]. Multiple Disease Modifying Anti rheumatic Drugs (DMARD'S) and eight biological agents are approved by USFDA for RA treatment. Rheumatoid arthritis is still poorly characterized in the era of the 21st century. Ideally, for any particular disease the planning of the treatment or the selection of the treatment is based on certain criteria which are individual characteristics of the patient including age, sex, body mass index, diet, and molecular profile of the patient, environment and genetic profile.

Personalized medication includes the study of metabolomics or the systematic study of the unique chemical fingerprints these fingerprints are specific which are left behind by the cellular processes are studied and assessed which helps in the identification of right phenotype and also the study of genes like BACH2 and RAD51B [19] which are responsible for the expression of rheumatoid arthritis. Screening process for change in protein/ biomarkers is useful for monitoring of disease progression. Currently there are many examples which use genetic information to predict the chances of getting a better response to certain drugs. Ex. Genetic testing of S-methyl transferase (TPMT). The thiopurine drugs like 6-mercaptopurines and azathiopurines are being catalyzed by the S-methylation by the enzyme TPMT can be prescribed to the patients. The polymorphism controlsthe activity of TPMT in RBC is which is being studied in relation to AZA toxicity. In the subpopulations of the patients with wild type of TPMT alleles and mutant alleles the wild type of TMPT alleles possessing patients tolerate therapy longer than patient with the mutant alleles who are likely to develop low leukocyte count. Testing of patients for drugs is done before prescribing the drugs, hence due to this, patients who are not getting the benefit from the particular drug can be identified and the expenses, possible side effects and toxicity can be minimized.

The Current Approaches for the Treatment of Rheumatoid Arthritis: Early and correct diagnosis starts with the treatment of DMARDs [21] in which MTX (Methotrexate) is the standard one in the treatment of RA. Although MTX and biologic agents are the best ever drugs which gives better outcomes for arthritic patients, up to 40 - 60% of RA patients fail to achieve a satisfactory response, and about 15 - 30% of the patients develop adverse drug events [14]. Failure in the treatment is due to toxicity produced by MTX and lack of effectiveness, a new therapeutic option is searched which is most advantageous. This selection will reduce the cost and potential adverse effects. The traditional markers like Auto-antibodies, acute phase reactants, bone and cartilage markers and the variety of cytokines are not sufficient for the study of personalized medicine and hence it is very important that identification of novel and better biomarkers [20] is used for the understanding of the molecular pathways involved in pathogenesis.

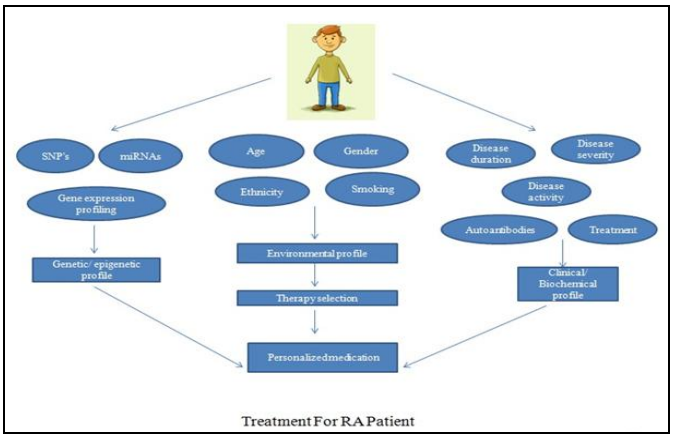

FIG. 1: PROCEDURE FOR THERAPY OPTIMIZATION IN RA

1. SNPs- Single-nucleotide polymorphism it is nothing but a variation in a single nucleotide which only occurs at a specific position in the genome. It is investigated that the genetic contribution of cytokine gene cluster in chromosome 5q31 is responsible for the disease Rheumatoid arthritis.

2. GWAS- Genome Wide Association Studies are very useful and successful in the detection and identification of the susceptibility of RA. In

this method RA susceptibility loci are discovered with the help of study of gene and linkage of the candidate. There are two loci which are at high risk for RA which are Human leukocyte antigen (HLA-DRB1) and protein Tyrosine phosphate (PTPN22). The combination of both loci *i.e.* HLA-DRB1 and PTPN22 can predict near about 40% of the genetic risk for RA [15, 7].

3. miRNA-Microarray profiling: Pharmaco-genomics- The miRNA plays a crucial regulator's role in the regulation of immune response both phenotypically and geno-typically. It was considered that miRNA act as an intracellular modulator of gene expression. It is investigated that miRNAs are stably present in body fluids such as plasma or serum in the cell free form. The presence and expression level of miRNA can be easily measured in tissues and body fluids with the help of quantitative Polymerase chain reaction (qPCR) and microarrays. These are the new biomarkers which help for the purpose of diagnosis and prognosis in various diseases including RA. It is also investigated that miRNA has a role in metabolomics as a modulator of cellular metabolism. Several miRNA increase a cellular response to a molecular stimulus in both plasma or serum fluids and inflamed joints. They may be helpful in monitoring severity of RA and understanding its pathogenesis, as the miRNAs can be apparently expressed even at different stages of disease progression.

4. Patient characteristics such as age, sex, habits like smoking habits, drinking habits etc and ethnic group. These parameters superficially say phenotypically predict the risk of the RA in Susceptible patients.

5. Disease pathogenesis, disease severity and disease duration are also helpful in identifying and planning the treatment strategy. With the help of this data, we can predict the generation of drug and dose of the drug. For example: for the disease duration longer than a month a combination of high dose drug of the newer generation can suggest as a part of the treatment strategy.

These all genetic information gives the best idea about the risk of disease and is helpful in planning the treatment.

Personalized Medicine in Future for RA: "Cut-to-cut" studies which are related to different biological therapies that indicate the correct and accurate selection of therapy are increasingly taken into consideration. For example, it is studied that the drug tocilizumab is very much appropriate as the first choice in biological therapy for the patients who cannot tolerate Methotrexate. However, these studies must be interpreted in the circumstances of the precisely selected clinical trial population [16-18].

CONCLUSION: On the basis of the information collected, the personalized medication is the most effective as it includes the detailed study about the genes, genotype, phenotype, various receptors and the loci for genes on chromosome. This all study when put into a context, the trial on the patient is carried out to check the response and the drug which gives the best response is selected for the treatment plan along with the diet and exercise. These two diseases which are discussed above can be treated rather managed for the symptoms on the basis of the risk for the patient to cause disease which can be diagnosed either phenotypically or genotypically. Early detection of these two diseases and preventive and curative measures for these diseases can give a better life experience to the patient. Hence, the planning strategy of the medication for the patient is the best way to serve the medical treatment. miR 146 is a family found in mammals and human which is of microRNA family. miR 146 specifically miR 146a has been highly upregulated in osteoarthritis cartilage. In addition it was found to be absent from the exosomes of prion infected cells suggesting it could be used as a biomarker for prion infection.

ACKNOWLEDGEMENT: Authors acknowledge the support and motivation from Management of SVKM's NMIMS, our Director Pharma Institutions Dr. R. S. Gaud sir and Associate Dean Dr. A. S. Deshpande madam.

CONFLICT OF INTEREST: Author declares no conflict of interest.

FUNDING INFORMATION: NA

REFERENCES:

1. Rate AR and Date AR: What is personalized medicine?.
2. Wilson BJ, Nicholls SG. The Human Genome Project, and recent advances in personalized genomics. Risk management and healthcare policy 2015; 8: 9.
3. Klonoff DC: Personalized medicine for diabetes. J Diabetes SciTechnol [Internet]. Diabetes Technology Society; 2008; 2(3): 335-41. Available from: http://www.ncbi.nlm.nih.gov/pubmed/19885196
4. Kissler SM, Cichowitz C, Sankaranarayanan S and Bortz DM: Determination of personalized diabetes treatment plans using a two-delay model. J TheorBiol [Internet]. Elsevier; 2014; 359: 101-11. Available from: http://dx.doi.org/10.1016/j.jtbi.2014.06.005
5. Phillips RA and Vögele C: Personalized Medicine. Int Encycl Soc Behav Sci [Internet]. 2015; 17: 925-31. Available from: http://linkinghub.elsevier.com/retrieve/pii/B9780080970868141492
6. Klonoff DC: Personalized Medicine for Diabetes. J Diabetes Sci Technol [Internet]. 2008; 2(3): 335-41. Available from: http://dst.sagepub.com/lookup/doi/10.1177/193229680800200301
7. Burgos PI, Danila MI, Kelley JM, Hughes LB and Bridges SL: Understanding personalized medicine in rheumatoid arthritis: A clinician's guide to the future. Ther Adv Musculoskelet Dis. 2009; 1(2): 97-105.
8. Klonoff DC: Personalized Medicine for Diabetes. J Diabetes SciTechnol [Internet]. SAGE Publications; 2008; 2(3): 335-41. Available from: http://journals.sagepub.com/doi/10.1177/193229680800200301
9. Ceriello A, Barkai L, Christiansen JS, Czupryniak L, Gomis R and Harno K: Diabetes as a case study of chronic disease management with a personalized approach: The role of a structured feedback loop. Diabetes Res Clin Pract. 2012; 98(1): 5-10.
10. Reddy SSK: Evolving to Personalized Medicine for Type 2 Diabetes. Endocrinol Metab Clin North Am [Internet]. 2016; 45(4): 1011-20. Available from: http://www.ncbi.nlm.nih.gov/pubmed/27823602
11. Karter AJ, Ackerson LM, Darbinian JA, D'Agostino RB, Ferrara A and Liu J: Self-monitoring of blood glucose levels and glycemic control: the Northern California Kaiser Permanente Diabetes registry. Am J Med [Internet]. 2001; 111(1): 1-9. Available from: http://www.ncbi.nlm.nih.gov/pubmed/11448654
12. Jain KK: Role of Biomarkers in Personalized Medicine. In: Textbook of Personalized Medicine [Internet]. New York, NY: Springer New York 2015; 91-7. Available from: http://link.springer.com/10.1007/978-1-4939-2553-7_3
13. Goran MI, Lane C, Toledo-Corral C and Weigensberg MJ: Persistence of Pre-Diabetes in Overweight and Obese Hispanic Children: Association With Progressive Insulin Resistance, Poor -Cell Function, and Increasing Visceral Fat. Diabetes [Internet]. 2008; 57(11): 3007-12. Available from: http://www.ncbi.nlm.nih.gov/pubmed/18678615
14. Goulielmos GN, Zervou MI, Myrthianou E, Burska A, Niewold TB and Ponchel F: Genetic data: The new challenge of personalized medicine, insights for rheumatoid arthritis patients. Gene [Internet]. Elsevier B.V. 2016; 583(2): 90-101. Available from: http://dx.doi.org/10.1016/j.gene.2016.02.004
15. Butts C, Kamel–Reid S, Batist G, Chia S, Blanke C, Moore M, Sawyer MB, Desjardins C, Dubois A, Pun J, Bonter K. Benefits, issues, and recommendations for personalized medicine in oncology in Canada. Current Oncology 2013; 20(5): e475.
16. Sonkoly E, Ståhle M and Pivarcsi A: MicroRNAs and immunity: novel players in the regulation of normal immune function and inflammation. Semin Cancer Biol [Internet]. 2008; 18(2): 131-40. Available from: http://www.ncbi.nlm.nih.gov/pubmed/18291670
17. Gregory RI, Chendrimada TP, Cooch N and Shiekhattar R: Human RISC couples microRNA biogenesis and posttranscriptional gene silencing. Cell [Internet] 2005; 123(4): 631-40. Available from: http://www.ncbi.nlm.nih.gov/pubmed/16271387
18. Ambros V: microRNAs: tiny regulators with great potential. Cell [Internet] 2001; 107(7): 823-6. Available from: http://www.ncbi.nlm.nih.gov/pubmed/11779458
19. McAllister K, Yarwood A, Bowes J, Orozco G, Viatte S, Diogo D, Hocking LJ, Steer S, Wordsworth P, Wilson AG and Morgan AW: Brief Report: Identification of BACH2 and RAD51B as Rheumatoid Arthritis Susceptibility Loci in a Meta-Analysis of Genome-Wide Data. Arthritis & Rheumatism 2013; 65(12): 3058-62.
20. Mahajan TD and Mikuls TR: Recent advances in the treatment of rheumatoid arthritis. Current opinion in rheumatology 2018; 30(3): 231-7.
21. Cutolo M: Chronobiology and the treatment of rheumatoid arthritis. Current opinion in rheumatology 2012; 24(3): 312-8.
22. Florez JC: Precision medicine in diabetes: is it time? Diabetes Care 2016; 39(7): 1085-8.
23. Pearson ER: Personalized medicine in diabetes: the role of 'OMICS' and biomarkers. Diabetic Medicine 2016; 33(6): 712-7.

How to cite this article:
Dani SU and Kulkarni PA: Treating diabetes and rheumatoid arthritis using personalized medication. Int J Pharm Sci & Res 2018; 9(12): 5424-30. doi: 10.13040/IJPSR.0975-8232.9(12).5424-30.