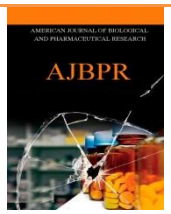
e-ISSN - 2348-2184
Print ISSN - 2348-2176

AMERICAN JOURNAL OF BIOLOGICAL AND PHARMACEUTICAL RESEARCH

Journal homepage: www.mcmed.us/journal/ajbpr



INFLUENCE OF DIABETIC BMI IN CAUSING MYOCARDIAL INFARCTION AS PER CART

S. Chandrasekaran[1], D. Sudarsanam[2], Divya Damodaran[3], Vimala Grace Rani[4]

[1]Assistant Professor. Dept of Mathematics, Pachaiyappa's College, Chennai, Tamilnadu, India.
[2]Professor, Department of Advanced Zoology and Biotechnology, Loyola College, Chennai, Tamilnadu, India.
[3]Research scholar, Department of advanced Zoology and Biotechnology, Loyola College, Chennai, Tamilnadu, India.
[4]Research scholar, Department of advanced Zoology and Biotechnology, Loyola College, Chennai, Tamilnadu, India.

Article Info

Received 29/07/2015
Revised 16/08/2015
Accepted 18/10/2015

Key words: Body Mass Index, Type 2 diabetes, Cardiovascular disease

ABSTRACT

Obesity may develop hypertension, diabetes and atherosclerosis, which in turn would manifest into high risk for cardiovascular disorders. Several case studies on the role of an increase in body weight in the manifestation of myocardial infarction in diabetics have been studied. Hence, the impact of Body Mass Index (BMI) was deciphered using the Classification and Regression Tree technique.

INTRODUCTION

An increase in body fat is generally associated with an increased risk for metabolic diseases. Body Mass Index (BMI) criteria is currently the primary focus of this study. Myocardial infarction in patients with diabetes increases the mortality. The impact of obesity on the risk of developing myocardial infarction in patients with diabetes is investigated.

MATERIALS AND METHODS

Collection of Data: Data on the following attributes (risk factors) were collected from 6073 diabetic subjects of MV Diabetics laboratory, Southern India, Chennai, laying emphasis on the 267 subjects of myocardial infarction. Risk factors such as body mass index (BMI), fasting blood sugar (FBS), haemoglobin (HBAIC), high density lipids (HDL), low density lipids (LDL), triglycerides (TGL), systolic blood pressure (BP-SYS), diastolic blood pressure (BP-DIA), nicotine usage, hypertension check (HT-CHECK), gender/sex, cholesterol (CHO) and diabetic-duration (DIA-DURATION) were considered for the statistical modeling.

Out of 6073 subjects, 267 were identified to have experienced myocardial infarction with due diagnostic features. Each of those myocardial infarction subjects was closely monitored in terms of the above thirteen risk factors found instrumental for the manifestation of myocardial infarction. Among the risk factors, gender/sex, nicotine usage and hypertension check are categorical factors and the body mass index is calculated using the conventional height and weight parameters, as given under.

$$BMI = \frac{Weight}{(Height)^2} \text{ where height is measured in meters and weight in kgs}$$

First 5000 data class were grouped by the Classification and Regression Tree technique and was used to build the model and the rest were used for validation. A Tree with 267 nodes was evolved and the information relating to BMI was discussed.

Corresponding Author

S. Chandrasekaran
Email:- chandra51071@yahoo.com

RESULTS

Classification and Regression Tree

File: 5000.xls
Target Variable: IHD_CHEC
Predictor Variables: HT_CHECC, SEX, NICOT, BMI, CHO, FBS, HBA1C, HDL, LDL, TGL, DIA_YEAR, BP_SYS, BP_DIA

Data Sample:

The above illustration (Figure 1-3 & Table 1) confirmed a relative cost profile and outlined the relationship between classification errors and tree size. Since scale was always between 0 and 1, it was called a relative error curve. While 0 meant no error or a perfect fit, 1 represented the performance of random guessing. In this model, the error started from 0.669 and ended at 0.963 indicating the model was of good fit.

The area under the curve (Figure 4) was highly significant and indicated that it was either a suitable or classical health model. Moreover, the area under the curve was determined to be 0.9564 which predicted a healthy model.

- From T43 and T 44, if BMI increased above 24.98 with HT_CHECC= 0, LDL <= 80.5, HBA1C > 9.55, BP_DIA <= 85, HDL > 46.5, the manifestation increased to 43% otherwise it was 0%.

- From T 56 and T 57, if the BMI range lay between 18.92 < BMI <= 19.215 with HT_CHECC = 0 HBA1C > 7.15 LDL > 80.5 BP_DIA <= 93 TGL > 71.5 BP_SYS > 102 BP_SYS <= 141 HDL <= 44.5 CHO <= 215.5 DIA_YEAR > 0.015 the manifestation increased of MI by 100% for BMI <18.92 it was 0%.

- From T68 and T69, the BMI range lay between 29.555 < BMI <= 30.01 HT_CHECC = 0 HBA1C > 7.15 LDL > 80.5 BP_DIA <= 93 TGL > 71.5 BP_SYS > 102 BP_SYS <= 141 HDL <= 44.5 CHO > 217.5 CHO <= 245.5. Thus, the manifestation of MI increased by 33% for BMI <29.555 it was 0%.

- From T110 and T 111 in the female subjects with HT_CHECC =1, HBA1C > 6.65, CHO <= 175.5, FBS <= 176.5, DIA_YEAR <= 5.95 the BMI ranged between 21.865< BMI <= 23.855 and the manifestation of MI increased by 66.67%. For BMI <21.865 it was 0%.

- From T153 and T 154 in the female subjects with HT_CHECC =1, HBA1C > 6.65, LDL > 107.5, CHO > 184.5, FBS > 121.5, BP_SYS <= 153, HDL <= 42.5, DIA_YEAR <= 5.05 if the BMI >34.78. Thus, the manifestation of MI increased by 33.33% for BMI <=34.78 it was 0%.

- From T 183 and T 184 in the male subjects HT_CHECC =1, HBA1C > 7.25, HBA1C <= 11.9,HDL > 25.5, HDL <= 54, LDL > 38,LDL <= 82.5,TGL > 117.5, TGL <= 322, BP_SYS <= 141, FBS > 113.5, FBS <= 253.5, DIA_YEAR > 0.69, DIA_YEAR <= 19.8, BMI <= 23.125; the manifestation of MI was found to be 33.33% and if the BMI lay between the range of 23.125< BMI <= 26.54. Thus, the manifestation of MI was found to be 91.67%, with an increase of 58.34%.

- From T197, T198 in the male subjects with HT_CHECC =1, DIA_YEAR > 0.69, LDL <= 82.5, BP_SYS <= 149, TGL > 56.5, TGL <= 177, HDL > 28, HBA1C > 5.85, HBA1C <= 7.55, the BMI range increased from 29.165< BMI <= 31.45 which increased the manifestation by 50% for the range of 28.315<BMI<=29.165 it was 0%.

- From T203 and T204 in the male subjects HT_CHECC =1, DIA_YEAR > 0.69, TGL <= 375, LDL <= 82.5, HDL > 25.5, HBA1C > 5.25, BP_SYS > 149If BMI range lied between 26.035< BMI <=31.45 and manifestation was found to be 22.22% for lesser values of BMI as it was null, i.e., for BMI <=26.035.

- From T 220 , T221 and T222, in the male subjects with HT_CHECC =1,DIA_YEAR > 0.69, BP_SYS <= 171, TGL > 60,TGL <= 375,FBS > 104.5, LDL > 82.5,LDL <= 173.5 if the BMI <=17.46 MI the manifestation was found to be 100% , when the BMI increased the range from 17.46< BMI <=19.32 and MI manifestation was found to decline to 16.67 and if BMI >19.32 MI manifestation was found 0%.
- From T 254, T255 and T256 in the male subjects HT_CHECC =1, BP_SYS <= 171, LDL > 82.5, LDL <= 173.5,CHO > 176.5,BP_DIA > 79,HDL > 39.5,DIA_YEAR > 0.69,DIA_YEAR<= 9.3,FBS > 108.5,HBA1C <= 9.3,TGL > 83,TGL<= 267.5 with BMI ranged between 25.2< BMI <= 26.09 and 28.29< BMI <= 28.485 and MI manifestation was found to be 100% and ranged between 26.09 <= BMI <28.29 as the MI manifestation found was 0%.

Thus, from the CART analysis of T153 and T154, it may be inferred that, an increase of BMI with same cholesterol, fasting blood sugar, HBA1c, HDL, LDL, Dia-Year, BP_SYS with hypertension, increased the probability by 33.3% in the case of female subjects. For male subjects, the values of T 254 and T 255 confirmed that even a slight increase in BMI could lead to MI with same CHO, FBS, HBA1c, HDL, LDL, TGL, Dia_Year, BP-SYS, BP_DIA even though hypertension decreased by 100%.

Table 1. Sample data details

Class	Learn	%	Total
0	4764	95.28	4764
1	236	4.72	236
Total	5000	100.00	5000

Figure 1. CART Tree showing the node informations

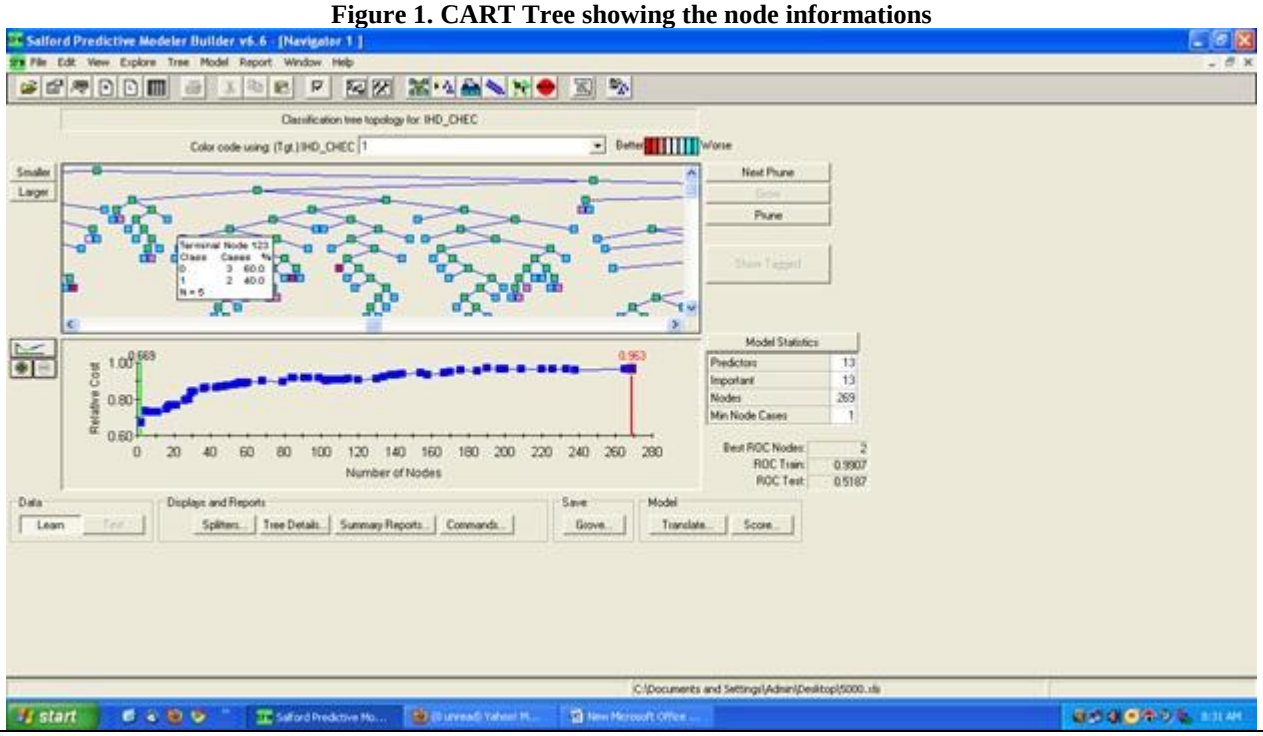


Figure 2. Classificatory tree topology based on 5000 data sets being used for building the following model for IHD_CHEC

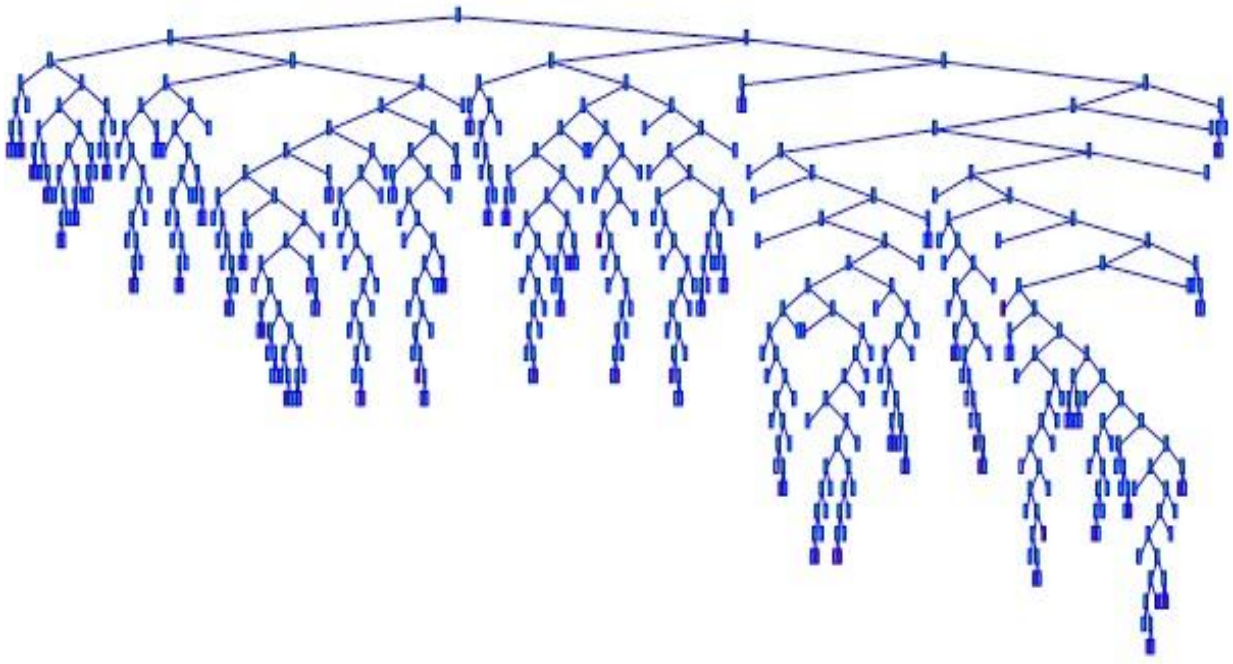


Figure 3. Error Curve

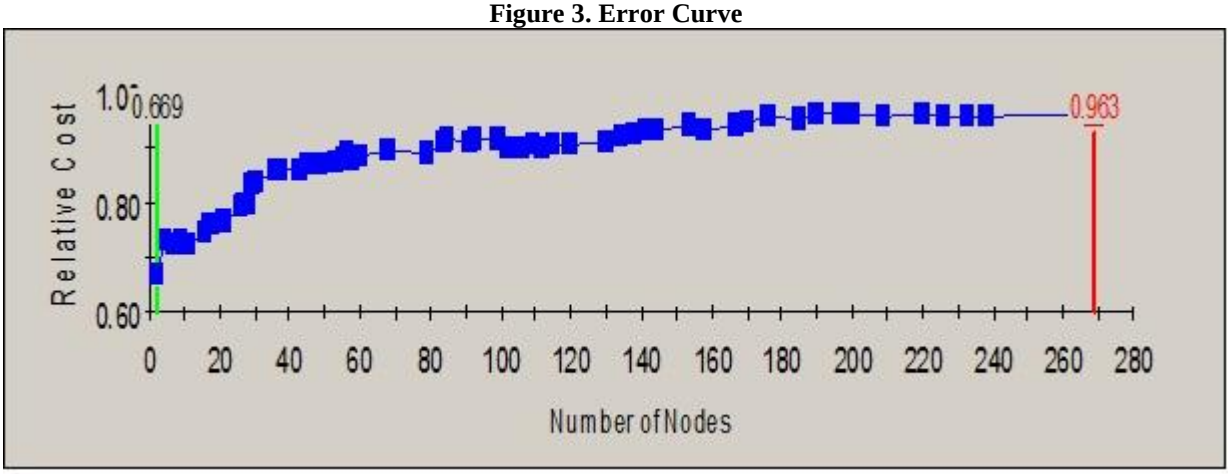
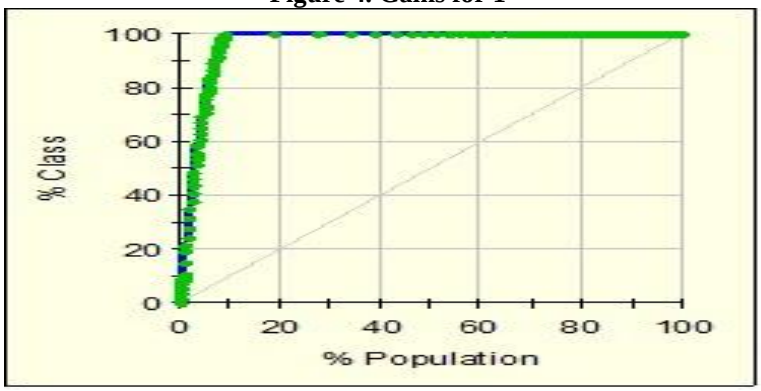


Figure 4. Gains for 1



DISCUSSION

Obesity is one of the major health problems in our society and its prevalence is rising worldwide [1-2]. In adults, being overweight is defined as a Body Mass Index (BMI) of 25 to 29.9 kg/m^2 and obesity as BMI-30 kg/m^2. Recent evidences indicate that obesity is associated with an increased morbidity and that it will soon overtake cigarette abuse as the leading cause of preventable death.

Obesity increases the risk of many physical and mental conditions. These co-morbidities are most commonly shown in a metabolic syndrome, a combination of medical disorders, which includes diabetes mellitus of type 2, high blood pressure, high blood cholesterol and high triglyceride levels. Excess body weight increases the risk of hypertension, coronary artery disease and stroke [3]. Several studies have described the association between Body Mass Index and mortality as indicated by a U-shaped curve, demonstrating increased mortality in the lowest and highest BMI distribution [4]. Obesity was also strongly associated with an increased risk of diabetes.

Complications are either directly caused by obesity or indirectly related through mechanisms sharing a common cause, such as a poor diet or a sedentary lifestyle. The strength of the link between obesity and specific conditions vary widely. One of the strongest are the link with type 2 diabetes and excess body fat underlies 64% of cases of diabetes in men and 77% of cases in women [5]. Hyperglycemia has been shown to increase the release of pro-inflammatory mediators such as IL-6, IL-8, and TNF, which are important in inflammation [6]. In addition, high glucose levels have been shown to have deleterious effects on optimal macrophage and neutrophil function [7]. These negative effects of hyperglycemia are responsible for the increased risk of infection leading to cardiovascular manifestation [8].

Health consequences fall into two broad categories, which are those attributed to the effects of increased fat mass, which includes osteoarthritis, obstructive sleep apnea, social stigmatization and those connected with increased number of fat cells, such as diabetes, cancer, cardiovascular disease and non-alcoholic fatty liver disease. Increase in body fat alters the body's response to insulin, potentially leading to insulin resistance. Increased fat also creates a pro-inflammatory state and a pro-thrombotic state. In individuals, without a known cardiovascular disease, an elevated Body Mass Index (BMI) (weight/height^2) was associated with an increased risk of mortality. However, in patients with certain specific chronic diseases, including heart failure,

low BMI has been associated with increased mortality. The association between BMI and mortality was proved to be unaltered by age and smoking status [9].

Studies have shown that BMI was associated with higher rates of heart failure among men with every one-unit of increase in BMI being associated with a 4% higher heart failure rate, irrespective of the man's waist size.

In women too BMI was associated with increased heart failure rates among the subjects with the largest waists. The association between BMI and heart failure events declined with age, suggesting that younger the age, greater is the impact of weight and in turn heart-related ailments.

CONCLUSION

Prevention of heart failure should begin early and must include regular, periodic BP checks and the screening for obesity shall begin at the early adulthood, resulting in behavioral, life style and diet modifications to reduce the elevated BMI. Higher BP and BMI along with other risk factors exert adverse effects on physiology and increase the risk of instant heart failure.

ACKNOWLEDGEMENT: None

CONFLICT OF INTEREST

The authors declare that they have no conflict of interest.

REFERENCES

1. Hedley AA, Ogden CL, Johnson CL, Carroll MD, Curtin LR, Flegal KM. (2004). Prevalence of overweight and obesity among US children, adolescents, and adults, 1999–2002. *JAMA*, 29, 2847-2850.
2. Daviglus ML, Liu K, Yan LL, Pirzada A, Manheim L, et al. (2004). Relation of body mass index in young adulthood and middle age to Medicare expenditures in older age. *JAMA*, 292, 2743-2749.
3. Mancia G, Seravalle G, Grassi G. (2002). Systolic blood pressure: an underestimated cardiovascular risk factor. *J Hypertens*, 20(5), 21–7.
4. Franklin SS, Gustin W, Wong ND, Larson MG, Weber MA, Kannel WB, Levy D. (1997). Hemodynamic patterns of age-related changes in blood pressure. *Circulation*, 96, 308-315.
5. Prescott B, Beth R. (2009). Waist size predictor of heart failure in men and women in Israel Deaconess Medical Centre. *J of Sc. R*, 112, 345-60.
6. Petrelli JM, Calle EE, Rodriguez C, Thun MJ. (2002). Body mass index, height, and postmenopausal breast cancer mortality in a prospective cohort of US women. *Cancer, Causes, Control*, 13, 325-332.
7. Landi F, Onder G, Gambassi G, Pedone C, Carbonin P, Bernabei R. (2000). Body mass index and mortality among hospitalized patients. *Arch Intern Med*, 160, 2641-644.
8. Singh PN, Lindsted KD, Fraser GE. (1999). Body weight and mortality among adults who never smoked. *Am J Epidemiol*, 150, 1152-1164.
9. Kenchaiah S, Pocock SJ, Wang D, Finn PV, Zornoff L, Skali H, et al., (2007). Body Mass Index and Prognosis in Patients With Chronic Heart Failure Insights From the Candesartan in Heart failure: Assessment of Reduction in Mortality and morbidity (CHARM) Program. *Circulation*, 116, 627-636.