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Abdominal Aortic Calcification and Its Determinants in People Living with Maintenance Haemodialysis in 2 Selected Public Hospitals from Myanmar

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ABSTRACT

Introduction: People living with maintenance haemodialysis have high mortality due to cardiovascular events which are related with vascular calcification. Vascular calcification is influenced by clinical and laboratory parameters. Some of them have controversial issues and are not studied in people living with maintenance haemodialysis in Myanmar. This study aimed to find out the prevalence of abdominal aortic calcification and its determinants in people living with maintenance haemodialysis attending in 2 selected public hospitals from Myanmar.

Methods: After getting informed consent, clinical assessment, hemoglobin, serum albumin, serum lipids, calcium, phosphate, CRP and serum fetuin-A level were done. And lateral lumber spine Xray was taken and abdominal aortic calcification score (AAC) was calculated.

Results: A total of 96 people living with maintenance haemodialysis were enrolled. The mean AAC score was 3.64 ± 4.86 and 61.5% (60/96) of paticipants had positive AAC score. A quarter 24.7% (24/96) had significant AAC score (score ≥ 5) and 37.5% (36/96) had AAC score 'Zero'. Mean age was 51 years and mean BMI was 21.6 ± 2.0 kg/m^2. Dialysis vintage ranged from 5 months to 8 years. All participants had hypertension whereas a quarter (24/96) had diabetes mellitus. All participants were clinically pale with mean hemoglobin 9.2 gm%. Mean serum albumin was 43.2 ± 9.9 g/L. Mean serum corrected calcium was low normal (8.8 ± 0.79 mg/dL) and mean serum phosphate was high normal (5.0 ± 1.6 mg/dL). Mean fasting total cholesterol was 195.13 ± 31.43 mg/dL. Mean HDL cholesterol was low (48.49 ± 15.62 mg/dL). Mean LDL cholesterol was high (130.0 ± 104.3 mg/dL). Mean serum triglyceride was high (152.44 ± 24.75 mg/dL). Mean CRP was 4.7 ± 1.9 mg/dL.

Mean Fetuin-A level was 412.3 µg/ml ± 170.5 µg/ml. And mean Fetuin-A level for those with AAC score 'Zero' was high (545.6 µg/ml ± 84.4 µg/ml) whereas it was low (181.2 µg/ml ± 90.0 µg/ml) in those with significant AAC score (AAC score ≥ 5). Those with non-significant AAC score (AAC score ≥ 5) had high Fetuin-A level (433.7 µg/ml ± 96.1 µg/ml).

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There was a statistically significant positive association between AAC score and age (*rho* = 0.296, p= 0.003); AAC score and fasting LDL cholesterol (*rho* = 0.226, p = 0.027); AAC score and fasting triglyceride (*rho* = 0.246, p = 0.016) and AAC score and CRP (*rho* = 0.404, p < 0.001). There was a statistically significant negative association between AAC score and fasting HDL cholesterol (*rho* = - 0.315, p < 0.001); and, AAC score and serum fetuin-A levels (*rho* = - 0.854, p < 0.001). AAC score was not associated with history of diabetes mellitus, hypertension, BMI, dialysis vintage, anemia, serum albumin, serum corrected calcium, serum phosphate.

Conclusion: In people living with maintenance haemodialysis, a significant positive association was found between abdominal aortic calcification and age, triglyceride, LDL cholesterol and CRP. Significant negative association was seen with HDL cholesterol and serum Fetuin-A level.

KEYWORDS: abdominal aortic calcification, maintenance haemodialysis, determinants

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INTRODUCTION

The global estimated prevalence of chronic kidney disease was 13.4% (Lv & Zhang, 2019); in population based studies, it increased with age (Q.-L. Zhang & Rothenbacher, 2008). Moreover, the prevalence of chronic kidney disease was increasing with time (Coresh et al., 2007). And, the etiology of chronic kidney disease was related mainly with non-communicable diseases like diabetes mellitus, hypertension and glomerular diseases related to autoimmune disorder (CHEGE & Yadla, 2023). For those with late stage of chronic kidney disease i.e., end stage renal disease (ESRD), hemodialysis is one of the treatment options. Peritoneal dialysis was not easily accessible in Myanmar. And, living donor renal transplant has been performing (Pyar KP et al., 2022).

The morbidity and mortality of cases with ESRD were mainly due to cerebrovascular accident, ischemic heart disease and sudden cardiac death (ALI, 2025); they were thought to be related with vascular calcification (Siracusa et al., 2024)(Makar & Pun, 2017). And, people living with maintenance hemodialysis were found to have significant vascular calcification (Wang et al., 2018) (Errihani et al., 2022). Therefore, vascular calcifications in people living with maintenance hemodialysis strongly influenced their prognosis. Kalra & Shanahan found that vascular calcification, regardless of its anatomical site, was an independent risk factor for cardiovascular mortality (Kalra & Shanahan, 2012). The presence of vascular calcification again caused progression of chronic kidney disease itself; moreover, it increased in cardiovascular mortality in people living with maintenance hemodialysis (Jia et al., n.d.). Therefore, it is important to detect factors provoking vascular calcifications in people living with maintenance hemodialysis. And, to improve the cardiovascular mortality, prevention and treatment of these factors which slow down the vascular calcification are essential.

Vascular calcification is the abnormal deposition of calcium, phosphorus, and other minerals in the vessel wall. It was commonly observed in diabetes, chronic kidney disease, uncommon genetic diseases and chronic inflammatory disease (Lanzer et al., 2014) and considered to be a passive process (Niu et al., 2020) (Goodman et al., 2004). However, it was found to be an active and regulated process, similar to bone mineralisation (Kraus et al., 2015); both vascular and valvular calcification were highly prevalent in people living with maintenance hemodialysis. It was highly regulated by multiple factors like bone-related proteins (Qin et al., 2021), genetic predispositions and molecular pathways (M. Mohamed et al., 2025) (Tatyana Archakova & Liudmila Nedosugova, 2018).

The vascular calcifications can be visualized by computed tomography (CT) scan, ultrasonogram and conventional X-ray. A computed tomography (CT) scan performed with and without contrast showed calcification of coronary arteries (Surana et al., 2008); and, peripheral vascular calcification was assessed easily using conventional Xray. Abdominal aortic calcification (AAC score) from lateral lumbar X-ray was reported as a reliable aortic calcification marker (Honkanen et al., 2008).

Abdominal aortic calcification (AAC score) from lateral lumbar X-ray was calculated as 'Zero' to '24'. 'Zero' means no calcification. Significant AAC score cut off value in people living with maintenance hemodialysis by Bai et al was an AAC score > 4.5 (Bai et al., 2023) and the value by Chen et al was greater than 5.5 (Chen et al., 2018). The atherosclerosis related vascular calcification in people living with maintenance hemodialysis was passive process and confined to tunica intima (Barreto et al., 2005) (Niu et al., 2020) (Goodman et al., 2004)(Honkanen et al., 2008) (Errihani et al., 2022) (Dhakshinamoorthy et al., 2017). It was part of aging process. Vascular calcification was found to be an independent predictor of cardiovascular morbidity and mortality(Qin et al., 2021) (Lanzer et al., 2014) (M. Mohamed et al., 2025). Moreover, vascular calcification in those with ESRD or people living with maintenance hemodialysis was found particularly in tunica media and it was not age specific. It was reported as an active and regulated process (Qin et al., 2021). It also pointed out the role of other factors contributing vascular calcifications in patients on maintenance hemodialysis; inflammatory pathways(Barreto et al., 2005)

(Siracusa et al., 2024a) (An & Son, 2013) (Dhakshinamoorthy et al., 2017) (Choi et al., 2019) (Dragoș et al., 2023), genetic predispositions and molecular pathways (Qin et al., 2021) (M. Mohamed et al., 2025) (Siracusa et al., 2024).

It was related with hyperphosphatemia and hypercalcemia, and the loss of specific vascular calcification inhibitors including pyrophosphate, fetuin-A, osteoprotegerin, iPTH(M. Li et al., 2025) (Goodman et al., 2004), vitamin D, FGF 23, sclerostin and matrix GLA protein (Siracusa et al., 2024) (Petrović et al., 2024) (Barreto et al., 2005) (Niu et al., 2020). Serum calcium, phosphorus, and iPTH levels in people living with maintenance hemodialysis were related with vascular calcification and it's progression (H. Zhang et al., 2023) (Goodman et al., 2004) (M. Li et al., 2025) (Klein, 2024) (Errihani et al., 2022) (Spiegel et al., 2004). Those with longer duration of dialysis and with a history of cardiovascular and cerebrovascular events, their AAC score were significantly higher" (Peyro-Shabani et al., 2018) (Honkanen et al., 2008) (Errihani et al., 2022).

Diabetes mellitus was the most significant clinical risk factors for vascular calcifications (M. Li et al., 2025) (Taniwaki et al., 2005). Aging had add on effect on vascular calcification (Niu et al., 2020) (Goodman et al., 2004). Those with higher BMI prone to vascular calcification (Barreto et al., 2005). Having anemia in people living with maintenance hemodialysis promoted vascular calcification through oxidative stress and inflammation (Siracusa et al., 2024) (Tatyana Archakova & Liudmila Nedosugova, 2018) (Choi et al., 2019) (An & Son, 2013).

Lipids involves in atherosclerosis and AAC (Deng & Qin, n.d.) (MA, 2015). Dyslipidemia together with diabetes mellitus, and hypertension attributed vascular calcifications in people living with maintenance hemodialysis (Oprisiu et al., 2002) (An et al., 2009) (Siracusa et al., 2024). HDL cholesterol was reported as anti-calcification factor (Qunibi, 2005) (D.-Y. Li et al., 2024).

Non-calcium-containing phosphate binders, low-dose active vitamin D plus cinacalcet, modification of dialysate calcium concentration, and sodium thiosulfate were found to delay vascular calcification in dialysis patients (Ohtake & Kobayashi, 2017) (Huybrechts et al., 2005) (Niu et al., 2020).

METHODS

Study design and population

A cross-sectional descriptive study was conducted in January 2023 to December 2024. People living with maintenance hemodialysis at public hospital at Yangon and Nay Pyi Taw were selected. Non-probability sampling method, especially convenience sampling method was used in this study. Sampling procedure was accomplished after completing the minimum required sample size 96 cases.

Written informed consent was taken from all participants after thorough explaining about the purpose and the procedures of the study. History taking was obtained including age, sex, duration of dialysis and imaging records. This study was approved by the Hospital Research and Ethics Committee of No.(1) Defence Services General Hospital (1000-Bedded) Mingaladon, Yangon.

Data collection and procedure

Clinical characteristics (sex, age, height, weight, hypertension, diabetes mellitus) were collected using a standardized case report form. Body weight was measured at the end of hemodialysis and BMI was calculated.

One milliliter (1mL) of venous blood was withdrawn from the anterior cubital vein before dialysis. Blood for hemoglobin, CRP, calcium, phosphate, fasting lipid profile and fetuin-A estimation were done. For serum fetuin-A assay, serum level was measured by using ELISA (Enzyme- Linked Immunosorbent Assay) kit, according to the manufacture's recommendation with commercially available kit (BioTechne R &D System).

The abdominal aortic calcification was detected on a lateral lumbar X-ray. It was done either after hemodialysis or non-hemodialysis day depending on participant's choice. The grading was performed using kaupilla score in which the extent of calcific deposits is graded on a per segment basis using the lumbar vertebral segments L1-L4. Per segment a score between 0 and 3 was given for both the anterior and posterior wall of the Aorta. These eight scores resulted in a composite abdominal aortic calcification score (AAC score) ranging between 0 and 24 points. The severity is determined with Kaupilla score (0-24). Kaupilla score '1 and above' is taken as calcification present.

Both clinical, laboratory and AAC score parameters were recorded. The data were checked carefully. And, supervision, completeness, and consistency of collected data were performed. The results were informed to attending nephrologist for treatment and confidentiality was maintained.

Working Definition

Significant abdominal aortic calcification score (AAC score) was defined as AAC score ≥ 5 in this study. It was calculated on a lateral lumbar X-ray.

People living with maintenance hemodialysis was defined as patients with end stage renal disease having regular hemodialysis (at least twice a week) for more than 3 months. HD vintage (Hemodialysis vintage) was defined as the duration of maintenance hemodialysis which was expressed as months.

Body mass index (BMI) was a person's weight in kilograms divided by the square of height in meters, nan indicator of body fatness. BMI was categorized as underweight (< 18.5 kg/m^2), normal weight (18.5 to 24.9 kg/m^2), overweight (25.0 to 29.9 kg/m^2) and (≥ 30.0 kg/m^2) obese. Body weight was measured after completion of hemodialysis secession.

Diabetes mellitus was defined as known case of diabetes mellitus diagnosed by physician or new case of diabetes mellitus raised HbA1C more than 6.5% with raised fasting blood sugar more than 12 6 mg/dL.

Hypertension was defined as resting (rest for 30 minutes) sitting blood pressure more than 135/85 mmHg or known case of hypertension diagnosed by physician.

CRP, an acute-phase reactant reflecting the inflammatory activity, was defined as elevated when it was higher than 0.5 mg/dL (< 0.5 mg/dL).

Anemia was defined as clinical pallor plus hemoglobin less than 11gm%.

Low serum albumin was defined as serum albumin less than 34 g/liter (normal 35-55 g/liter).

Raised corrected calcium was defined as serum calcium corrected with serum albumin more than 10.5 mg/dl and low corrected calcium was defined as serum calcium corrected with serum albumin less than 8.5 mg/dL (normal range 8.5 to 10.5 mg/dL).

Raised phosphate was defined as raised serum phosphate more than 4.5 mg/dL irrespective of phosphate lowering drugs (normal range 3.4 to 4.5 mg/dL).

Fasting cholesterol was defined as raised cholesterol more than 200 mg/dL irrespective of lipid lowering drugs (normal <200 mg/dL).

Raised triglyceride was defined as raised triglyceride more than 150 mg/dL irrespective of lipid lowering drugs (normal <150 mg/dL).

Raised LDL cholesterol was defined as LDL cholesterol more than >100 mg/dL irrespective of lipid lowering drugs(normal <100 mg/dL).

High HDL cholesterol was defined as HDL cholesterol more than >130 mg/dL) irrespective of lipid lowering drugs (normal 60-130 mg/dL).

Statistical analysis

The data were analyzed in the statistical package for social science (SPSS), version 25. Category variables were described as number and percentage. Numerical variables were expressed as mean and standard deviation. The correlation was calculated by Spearman's correlation test and correlation coefficient was expressed as '*rho*'. P value < 0.05 was set as statistically significant.

RESULTS

A total number of 96 people living with haemodialysis at 1,000 bedded hospital in Yangon and Nay Pyi Taw were enrolled. Table (1) shows base line characteristic. Regarding sex distribution, males constituted 54.2% (n = 52) and females made up 45.8% (n = 44). Mean age was 51 years; the minimum was 24 years and the maximum was 73 years. Dialysis vintage varied from 5 months to 8 years. Diabetes mellitus was seen in 25% (24/96) cases. And nearly all participants (94/96) had hypertension. A quarter had IHD. Figure (1.a) demonstrates association between AAC score and age; AAC score became higher with increasing age. There was a statistically significant association between AAC score and age (p = 0.003).

Thirty-six cases had AAC score 'Zero'; their mean age was 47.7 years. In AAC score 'Zero' group, the youngest was 31 years and the oldest was 72 years. Eight cases were over 60 years; and, seven cases were 31 to 40 years. As shown in figure (1.b), the majority was in the younger age group (31-44 years) and the minority was over 58 years.

In this study, AAC score (abdominal aortic calcification score) was calculated from lateral lumbar spine Xray. It ranged from 'Zero' to '24'. AAC score ≥ 5 was defined as significant AAC score.

Table (2) reveals frequency distribution of various AAC score. Mean AAC score in this study was 3.64 ± 4.86. The lowest was 'Zero' and the highest was '20'. Over sixty percent 61.9% (60/96) had positive AAC score (1 - 24); and (38.1%, 36/96) did not have AAC i.e., AAC score 'Zero'.

Because AAC score ≥ 5 was defined as significant AAC score in this study, 24.7% (24/96) had significant AAC score.

AAC score 18 and above was found in 4 cases and their ages were 34 years, 48 years, 58 years and 64 years. Maximum AAC score '20' was seen in 65 years old man with diabetes mellitus with HD vintage of 26 months.

Diabetes mellitus was seen in 25% (24/96) cases. Mean AAC score in this study was 3.64 ± 4.86. Mean AAC score in participants with diabetes mellitus was 4.8; it was higher than participants without diabetes mellitus where AAC score was 3.2.

Mean body mass index (BMI) was 21.6 ± 2.0 kg/m^2. The lowest and the highest BMI were 18.4 kg/m^2 and 26.4 kg/m^2 respectively. Figure (2) reveals the association between AAC score and BMI. There was no association between AAC score and BMI.

Mean CRP was 4.7 ± 1.9 mg/dL; the range was 1.5 to 7.0 mg/dL. Normal CRP was less than 1.0 mg/dL. The level of CRP became higher with increasing AAC score. Figure (10) demonstrates the association between AAC score and CRP; it was statistically significant (p < 0.001).

Mean serum albumin was 43.2 ± 9.9 g/liter. Figure (3) illustrates the association between AAC score and serum albumin (p = 0.205). Ninety percent of participants in this study were anemic; mean hemoglobin was 9.2 gm%.

Mean serum corrected calcium was 8.8 ± 0.79 mg/dL (normal range 8.5 to 10.5 mg/dL). Mean serum phosphate was 5.0 ± 1.6 mg/dL (normal range 3.4 to 4.5 mg/dL). Figure (4) demonstrates the association between AAC score and corrected calcium (p = 0.111). Figure (5) shows the association between AAC score and serum phosphate (p = 0.341). Neither serum corrected calcium nor serum phosphate was related with AAC score.

Mean fasting total cholesterol was 195.13 ± 31.43 mg/dL. The association between AAC score and fasting total

cholesterol was not significant (p = 0.181); it was highlighted in figure (6).

Mean HDL cholesterol was low (48.49 ± 15.62 mg/dL). Figure (8) reveals the association between AAC score and fasting HDL cholesterol. It was statistically significant (p < 0.001).

Mean LDL cholesterol was high (130.0 ± 104.3 mg/dL). Figure (9) demonstrates the association between AAC score and fasting LDL cholesterol; it was statistically significant (p = 0.027).

Mean serum triglyceride was high (152.44 ± 24.75 mg/dL). The association between AAC score and fasting triglyceride is revealed in figure (7). It was statistically significant (p = 0.016).

Mean serum Fetuin-A level for all paticipants (people living with maintenance haemodialysis) was 412.3 μg/ml ± 170.5 μg/ml. And mean Fetuin-A level for those with AAC score 'Zero' was high (545.6 μg/ml ± 84.4 μg/ml) whereas it was low (181.2 μg/ml ± 90.0 μg/ml) in those with significant AAC score (AAC score ≥ 5). Those with non-significant AAC score (AAC score ≥ 5) had high Fetuin-A level (433.7 μg/ml ± 96.1 μg/ml). Figure (11) demonstrated the association between AAC score and serum Fetuin-A level. A significant association was found between AAC score and serum fetuin-A levels (p < 0.001).

DISCUSSION

Vascular calcification has now been recognized as a major problem in people living with maintenance hemodialysis because of its strong influence on the prognosis. Delaying or improving the vascular calcification was thought to be very important to improve the cardiovascular mortality in them (Wang et al., 2018). To prolong the quality of life of people living with maintenance hemodialysis, detection and surveillance of vascular calcification as well as its prevention and treatments are extremely important.

Abdominal aortic calcification (AAC score) from lateral lumbar X-ray was calculated as 'Zero' to '24'. 'Zero' means no calcification. Bai et al reported that people living with maintenance hemodialysis with an AAC score > 4.5 had significantly elevated all-cause and cardiovascular mortality compared with those with an AAC score ≤ 4.5 (Bai et al., 2023). On the other hand, AAC score greater than 5.5 in people living with maintenance hemodialysis was reported as a reliable abdominal aortic calcification marker (Chen et al., 2018). Because the best cut-off value of AAC score was 5.5 by Chen et al and > 4.5 by Bai et al, AAC score ≥ 5 was defined as significant AAC score in this study. One quarter of participents in this study 24.7% (24/96) had significant AAC score; the range of age was 24 - 73 years.

The atherosclerosis related vascular calcification was mainly confined to tunica intima. Several reports on vascular calcification in those with ESRD or people living with maintenance hemodialysis was found particularly in tunica media and it was not age specific. In this study, mean age was 51 years; not young. Their AAC score became higher with increasing age and it was statistically significant. Having statistically significant relation between AAC score and age in this study strongly supported the known fact that vascular calcification was a degenerative disease which occurred in older adults. Therefore, this study highlighted the atherosclerosis related vascular calcification in people living with maintenance hemodialysis (Barreto et al., 2005) (Niu et al., 2020) (Goodman et al., 2004)(Honkanen et al., 2008) (Errihani et al., 2022) (Dhakshinamoorthy et al., 2017). People living with maintenance hemodialysis might probably have accelerated atherosclerosis which led to premature vascular calcification.

The AAC score 'Zero' was recorded in 36 participents in this study; no calcification in aorta. In AAC score 'Zero group', the youngest was 31 years and the oldest was 72 years. Nonetheless, the proportion of younger age group (31- 44 years) having AAC score 'Zero' was highest; and, older age group (58 – 72 years) had the lowest proportion. It was compatible with former report 'vascular calcification was an aging process' (Barreto et al., 2005) (Niu et al., 2020) (Goodman et al., 2004) (Errihani et al., 2022).

In this study, mean AAC score (abdominal aortic calcification score) was 3.64 ± 4.86. The lowest was 'Zero' and the highest was '19'. Higher AAC score (18 and above) was found in 4 cases and their respective ages were 34 years, 48 years, 58 years and 64 years. Having higher AAC score in relatively young age (34 years and 48 years) could be explained by possible accelerated nature of vascular calcification, not passive process like atherosclerosis. It confirmed the fact that vascular calcification in people living with maintenance hemodialysis was an active and regulated process (Qin et al., 2021). Furthermore, it also pointed out the role of other factors contributing vascular calcifications in people living with maintenance hemodialysis; inflammatory pathways, genetic predispositions and molecular pathways (Qin et al., 2021) (M. Mohamed et al., 2025) (Siracusa et al., 2024).

History of ischemic heart disease was seen in '24' cases; and their mean AAC score was '10'. None of them had cerebrovascular accident. Therefore, it supported former findings "AAC score was significantly higher in those with longer duration of dialysis and patients with a history of ischemic heart disease" (Peyro-Shabani et al., 2018) (Honkanen et al., 2008) (Errihani et al., 2022).

Diabetes mellitus was reported as the most significant clinical risk factor for coronary artery calcification (M. Li et al., 2025) as well as vascular calcifications. Combination of old age and diabetes mellitus was found to cause rapid progression of vascular calcification (Niu et al., 2020) (Goodman et al., 2004). In this study, a quarter of participants (24/96) were diabetics and their mean AAC score was '4.8'; it was higher than that of non-diabetics (3.2). And, maximum AAC score

(AAC score '20') was seen in male participant, 65 years old with diabetes mellitus with HD vintage of 26 months. This finding supported the former report that AAC score in people living with maintenance hemodialysis with diabetes mellitus was found to be higher than that of non-diabetic participants even with short dialysis vintage (Taniwaki et al., 2005) (Peyro-Shabani et al., 2018) (Honkanen et al., 2008) (Errihani et al., 2022).

BMI was measured in dry weight situation; after completion of hemodialysis section. Normal range of BMI was 18.5 to 24.9 kg/m^2 in normal healthy person. Mean BMI was 21.6 ± 2.0 kg/m^2 in this study. The lowest and the highest BMI were 18.4 kg/m^2 and 26.4 kg/m^2 respectively. There was no association between AAC score and BMI in this study. Mean AAC score of those having BMI 25 kg/m^2 and above (18/3) was '6'. Barreto et al reported that coronary artery calcification was highly prevalent in people living with maintenance hemodialysis; and it was associated with older age, higher BMI, inflammation and reduced trabecular bone volume (Barreto et al., 2005). Therefore, this study was not comparable with the findings of Barreto et al; it was probably due to relatively low sample size.

Normal CRP was less than 1.0 mg/dL. In this study, mean CRP of people living with maintenance hemodialysis was high (4.7 ± 1.9 mg/dL); their range was 1.5 to 7.0 mg/dL. The level of CRP became higher with increasing AAC score; it was statistically significant. It strongly suggested that vascular calcification found in people living with maintenance hemodialysis was an inflammatory process; and it was in line with previously reports (Barreto et al., 2005) (Siracusa et al., 2024) (An & Son, 2013) (Dhakshinamoorthy et al., 2017) (Choi et al., 2019) (Dragoș et al., 2023).

In this study, mean serum albumin was not low (43.2 ± 9.9 g/liter). There was no association between AAC score and serum albumin; the possible reason was that people living with maintenance hemodialysis in this study did not have hypoalbuminemia. Malnutrition was suggested as one possible cause of AAC and its progression in people living with maintenance hemodialysis patients (Choi et al., 2019).

In this study, almost all participants had anemia; and, mean hemoglobin was 9.0 gm%. Having early onset of vascular calcification and accelerated pattern of its course in people living with maintenance hemodialysis was associated with new or non-traditional risk factors such as oxidative stress, anemia, and inflammation (Siracusa et al., 2024) (Tatyana Archakova & Liudmila Nedosugova, 2018). Choi et al reported that conditions which increase hemoglobin level would retard progression of vascular calcification in people living with maintenance hemodialysis (Choi et al., 2019) (An & Son, 2013).

Neither serum corrected calcium nor serum phosphate was related with AAC score in this study. It was suggested that having significant vascular calcification in people living with maintenance hemodialysis was due to an accumulation of calcium and phosphate deposits within the walls of blood vessels; and, it caused the loss of elasticity of the arterial walls. Several reports mentioned association between serum corrected calcium and serum phosphate levels and vascular calcifications in people living with maintenance hemodialysis. Siracusa et al reported that hyperphosphatemia and hypercalcemia attributed to vascular calcification in them (Siracusa et al., 2024). The study from Japan found that an increase in serum phosphate level was associated with an increased vascular calcification burden in people living without maintenance hemodialysis (early cases of ESRD) undergoing cardiovascular surgery (Kinugasa et al., 2016). Taniwaki et al found that hyperphosphatemia was an independent risk factor for increasing AAC score in people living with maintenance hemodialysis without diabetes mellitus (Taniwaki et al., 2005). In this study mean serum corrected calcium level was lower limit of normal range; and serum phosphate was slightly higher than normal value. Both serum corrected calcium and serum phosphate were not significantly related with AAC score in this study; it did not support former reports.

Nonetheless, Zhang et al suggested that maintenance of serum calcium, phosphorus, and iPTH target levels in people living with maintenance hemodialysis patients was essential in order to lower the risk of progression of vascular calcification; and it was supported by several studies (H. Zhang et al., 2023) (Goodman et al., 2004) (M. Li et al., 2025) (Klein, 2024) (Errihani et al., 2022) (Spiegel et al., 2004). Moreover, non-calcium-containing phosphate binders, low-dose active vitamin D plus cinacalcet, modification of dialysate calcium concentration, and sodium thiosulfate showed beneficial effect on delaying vascular calcification in people living with maintenance hemodialysis (Ohtake & Kobayashi, 2017) (Huybrechts et al., 2005) (Niu et al., 2020). Relation between AAC and iPTH serum levels was positive in earlier finding (Kimura et al., 1999); however, no significant correlation was found in later reports (Peyro-Shabani et al., 2018). In this study, iPTH level was not measured as we were in limited setting.

Depending on relation between AAC and bone related factors, serum calcium, phosphorus, and iPTH, there were controversial interventions in managing vascular calcifications like calcium channel blockers, renin–angiotensin system inhibitions, statins, bisphosphonates, denosumab, vitamins, and ion conditioning agents (Mizuiri et al., 2021) (Pan et al., 2023) (Raggi, 2002) (Spiegel et al., 2004) (Mccullough & Soman, 2004). In this study nearly 90% of participants were taking calcium channel blockers for control of hypertension and nearly 80% of them had statins for hypercholesterolemia. We need long term study whether they have real impact on vascular calcification or not.

Lipids involves in atherosclerosis and vascular calcification, AAC. There was no association between AAC score and fasting total cholesterol in this study; it confirmed earlier

research where there was no correlation between lipid level (LDL cholesterol, HDL cholesterol and triglyceride) and AAC (Kimura et al., 1999). A significant inverse U-shaped correlation between lipoprotein (LAP) and the prevalence of AAC was noted by Deng & Qin et al (Deng & Qin, n.d.); LAP could be a potential biomarker for evaluating AAC risk. Another finding in 2015 was that 'dyslipidemia including hypertriglyceridemia and hypo-HDL cholesterol were common in people living with maintenance hemodialysis' (MA, 2015); and and they were potentially atherogenic.

On the other hand, AAC score was inversely associated with fasting HDL cholesterol in this study. As HDL cholesterol is a good-lipids, it has protective effect on vascular calcification. HDL cholesterol was reported as anti-calcification factor (Qunibi, 2005). And, elevated serum HDL3-C levels in people living with maintenance hemodialysis was an independent risk factor for cardiovascular calcification (D.-Y. Li et al., 2024). In this study, both serum LDL cholesterol and triglyceride were directly related with AAC score. Traditional risk factors like age, sex, dyslipidemia, diabetes mellitus, and hypertension determined vascular calcifications in people living with maintenance hemodialysis (Oprisiu et al., 2002) (An et al., 2009) (Siracusa et al., 2024a). Therefore, having direct relationship between AAC score and bad lipids, LDL cholesterol and triglyceride in this study, strongly supported former reports.

Fetuin-A, a circulating inhibitor of calcification, is a marker of inflammatory-nutritional state. Therefore, serum Fetuin-A reliably predicted vascular calcification and carotid intima media thickness in some reports. Serum Fetuin-A and vascular calcification emerged as significant risk factors for all-cause and cardiovascular mortality even in patients with non-dialysis chronic kidney disease (early stage ESRD). Decreased levels of fetuin A was thought to be involved in the pathogenesis of coronary artery disease in people living with maintenance hemodialysis. In this study, nearly 62% had positive AAC score (1-24). Those with AAC score 'Zero' had the highest serum fetuin-A level and those with significant AAC score (AAC score ≥ 5) had the lowest level.

Having low serum Fetuin-A was related with high AAC score in this study supported former findings; lower serum fetuin-A was found to be associated with severe abdominal aortic calcification (AAC) in early 2000 (Schoppet et al., 2015). Serum fetuin-A levels had inhibitory effect on vascular calcification (Turkmen et al., 2011) (Hendig et al., 2006) (Siracusa et al., 2024a) (O. N. Mohamed et al., 2024) (Gluba-Brzózka et al., 2014).

In this study, paticipants with low serum fetuin-A level (below normal range) had a higher mean AAC score compare to those with normal serum fetuin-A levels. This data suggested that people living with maintenance hemodialysis with lower serum fetuin-A levels had higher mean AAC score (Lyu et al., 2018) (Petrović et al., 2024) (Siracusa et al., 2024) (Schoppet et al., 2015) (Turkmen et al., 2011) (Hendig et al., 2006). Gheorghe et al found that fetuin-A levels were inversely associated with coronary artery calcification quantified by multidetector computed tomography (CT) in patients undergoing hemodialysis (Gheorghe et al., 2024); it could predict future coronary events.

CONCLUSION

AAC was common among people living with maintenance hemodialysis. Older age related positively with AAC score. Those with high BMI, history of diabetes mellitus and history of ischemic heart disease had high AAC score. Dyslipidemia (high triglyceride and LDL cholesterol) increased the AAC score whereas high HDL cholesterol had reverse effect. Inflammatory markers (CRP) positively related with AAC score. Serum Fetuin-A negatively related with AAC score.

RECOMMENDATION

Several risk factors for vascular calcification, circulating soluble thrombomodulin, vitamin K deficiency, pyrophosphate, osteoprotegerin, and matrix GLA protein, iPTH, vitamin D, FGF 23, sclerostin, osteopontin and bone morphogenetic protein-7 (BMP-7), should be analyzed. We need to explore vascular calcification in association with vascular bed differences, sex differences, and ethnic differences. Therapeutic trials like vitamin K therapy, statin therapy, ACEI, calcium free phosphate binder, non-calcium-containing phosphate binders, low-dose active vitamin D plus cinacalcet, modification of dialysate calcium concentration, and sodium thiosulfate should be done.

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ETHICAL CONSIDERATION

The data collection using standardized case report forms was approved by Institutional Research and Ethic Committee from Defence Services Medical Academy, Myanmar. Informed consent was also taken from each participent. Privacy and confidentiality of information were maintained throughout the study process.

DECLARATION OF CONFLICT OF INTEREST

The authors declared no potential conflicts of interests with respect to authorship and publication of this article.

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Table (1). Baseline characteristics of people living with maintenance haemodialysis (n=96)

SR	Parameters	Mean ± SD
1	Age (years)	51.0 ± 12.0
2	BMI (kg/m^2)	21.6 ± 2.0
3	CRP (mg/dL)	4.7 ± 1.9
4	AAC score (abdominal aortic calcification score)	3.64 ± 4.86
5	Hemoglobin (gm%)	9.2
6	Serum albumin (g/liter) (30-50)	43.2 ± 9.9
7	Serum corrected calcium (mg/dL) (8.5 to 10.5)	8.8 ± 0.79
8	Serum phosphate (mg/dL) (3.4 to 4.5)	5.0 ± 1.6
10	Serum Fasting total cholesterol (mg/dL) (<200)	195.13 ± 31.43
11	Serum HDL cholesterol (mg/dL) (>50)	48.49 ± 15.62
12	Serum triglyceride (mg/dL)(<150)	152.44 ± 24.75
13	Serum Fetuin-A level (µg/mL) (303-671)	412.32 ± 170.52

Table (2). AAC score groups and Serum Fetuin-A level (n=96)

AAC score group	**Number of people living with maintenance hemodialysis (%)**	**Serum Fetuin-A level (303-671 µg/mL) (Mean ± SD)**	**Remark**
AAC score (0-24)	96 (100)	412.3 ± 170.5	
AAC score 'Zero'(0)	36 (38.1%,36/96)	545.6 ± 84.4	
AAC score (1-24)	60 (61.9%,60/96)		
AAC score (≥ 5)	24 (24.7%,24/96)	181.2 ± 90.0	AAC cut off value 5.5 by Chen et al > 4.5 by Bai et al
AAC score (1-4)	36 (37.1%,36/96)	433.7 ± 96.1	

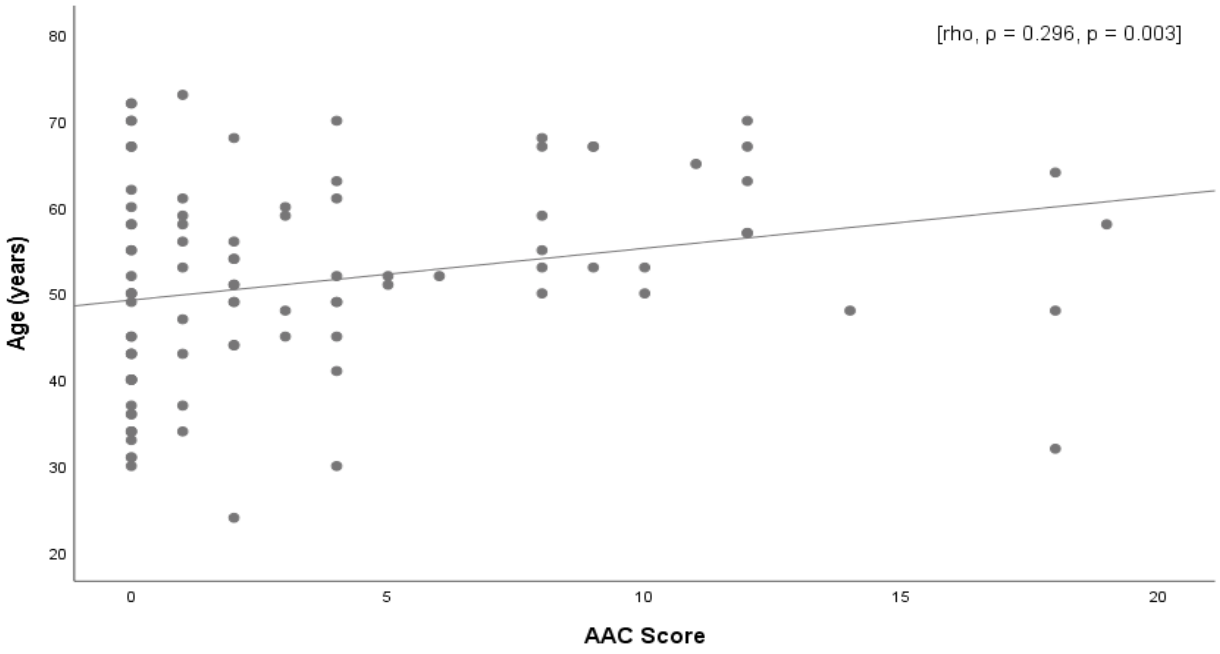

Figure (1.a). Association between AAC score and age (n=96)

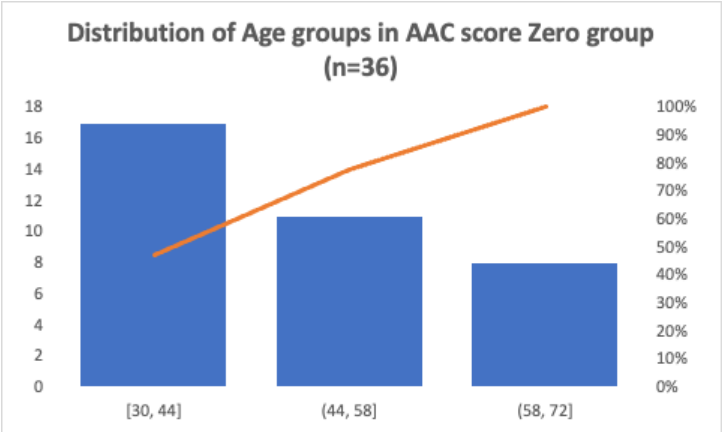

Figure (1.b). Distribution of 'Age groups' in AAC score 'Zero' groups (n = 36)

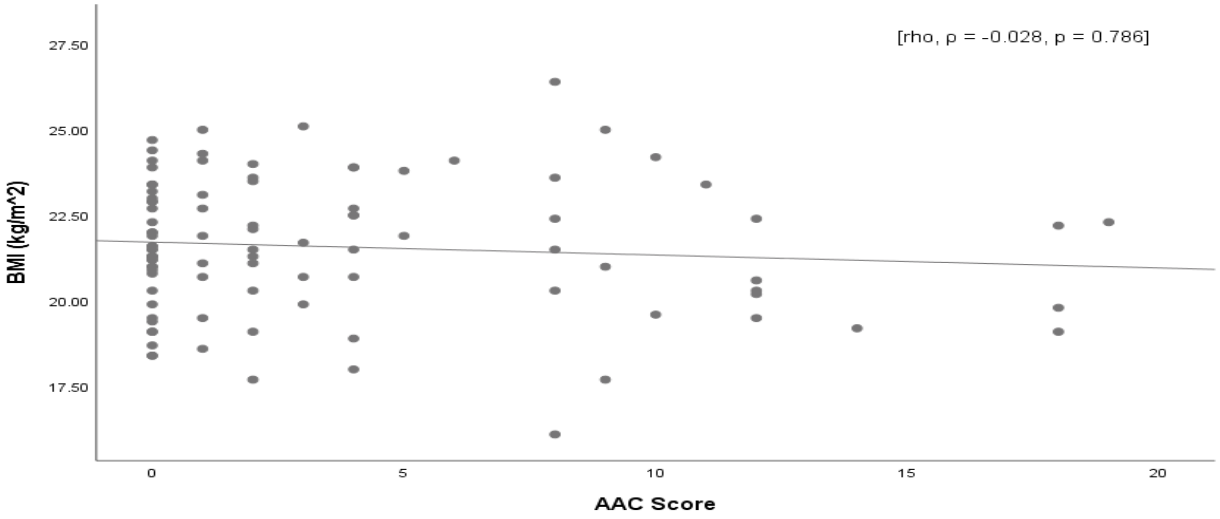

Figure (2). Association between AAC score and BMI

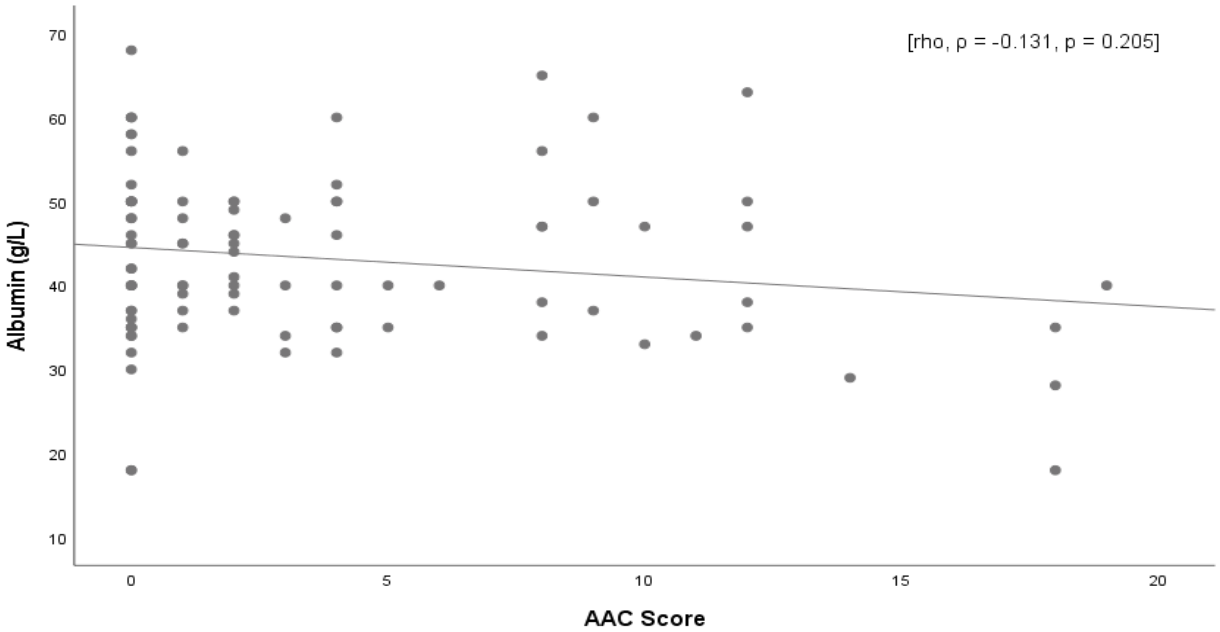

Figure (3). Association between AAC score and serum albumin

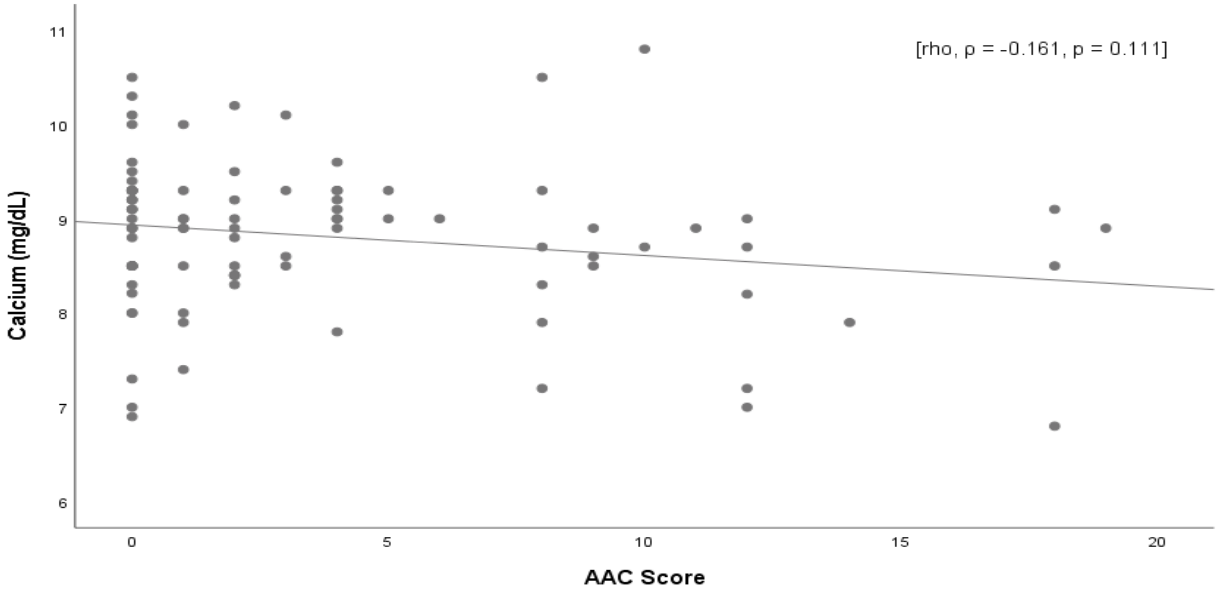

Figure (4). Association between AAC score and corrected calcium

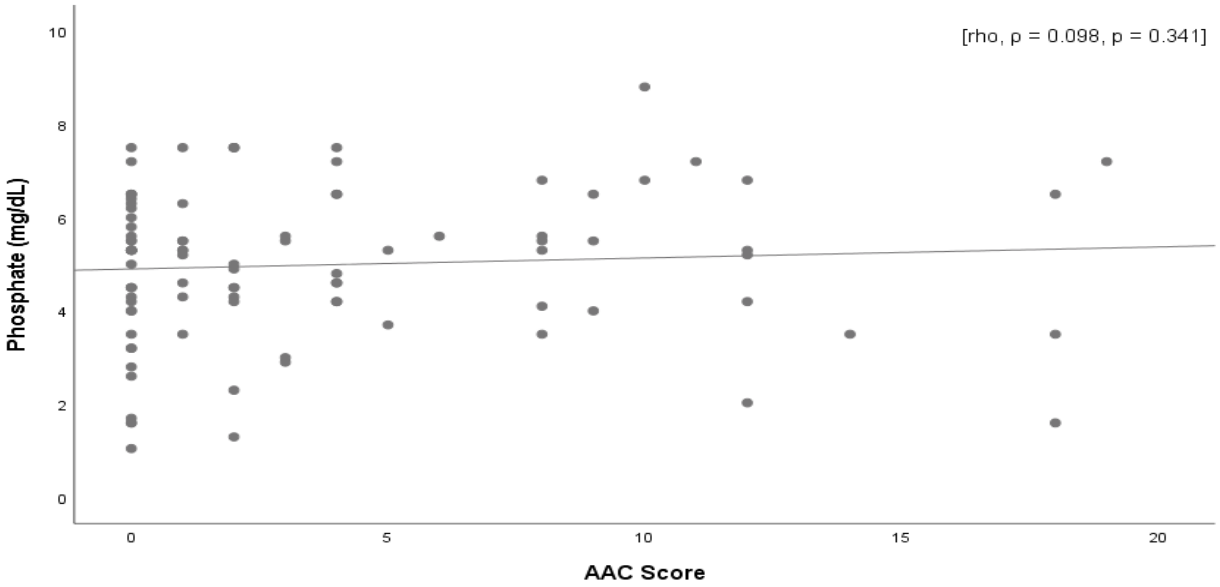

Figure (5). Association between AAC score and serum phosphate

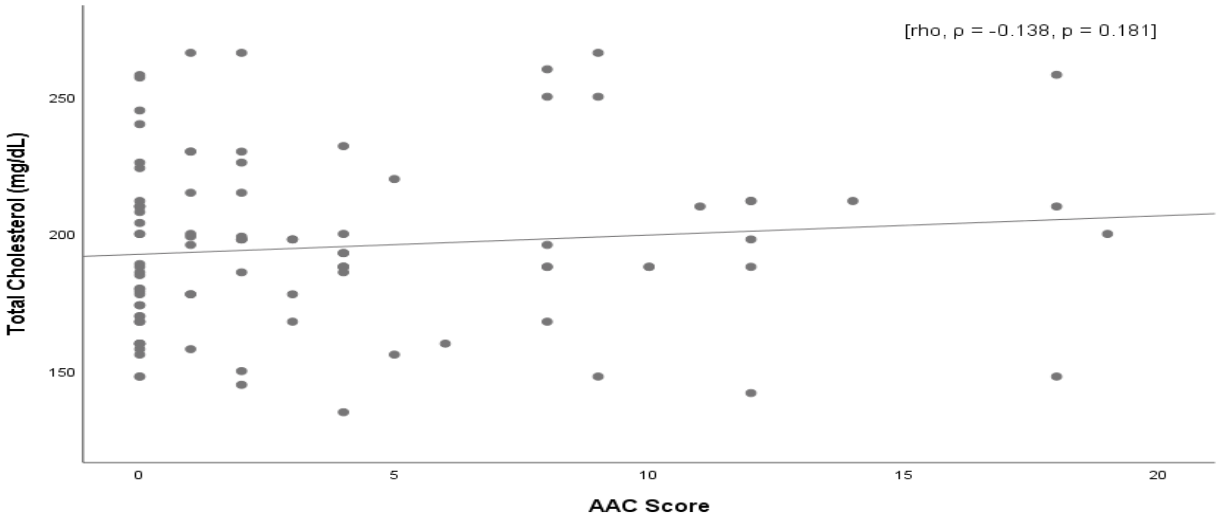

Figure (6). Association between AAC score and fasting total cholesterol

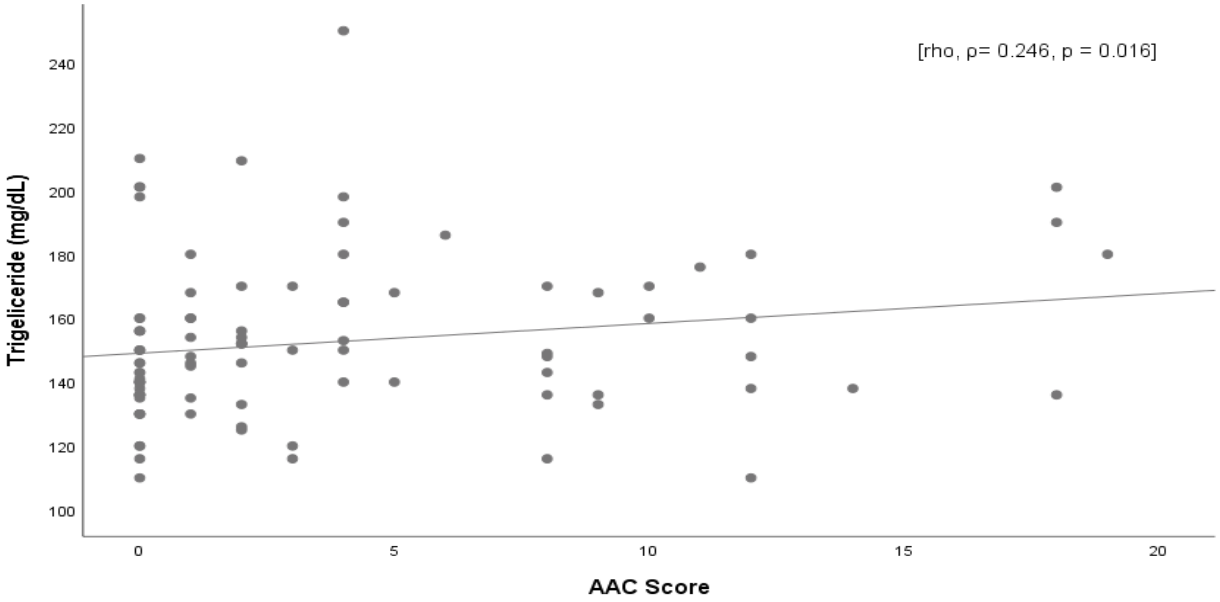

Figure (7). Association between AAC score and fasting triglyceride

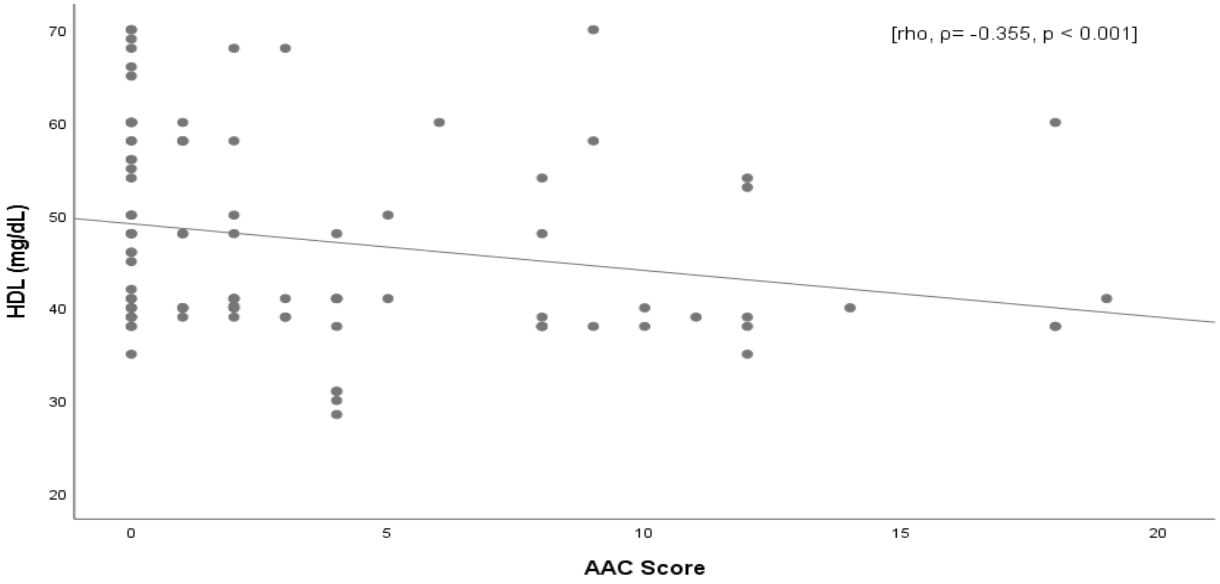

Figure (8). Association between AAC score and fasting HDL cholesterol

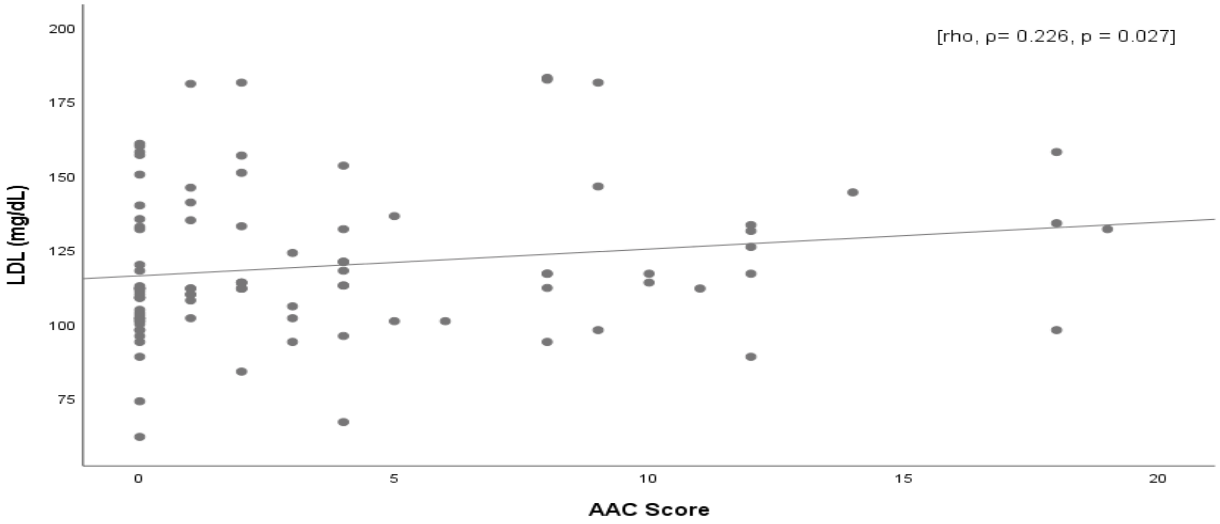

Figure (9). Association between AAC score and fasting LDL cholesterol

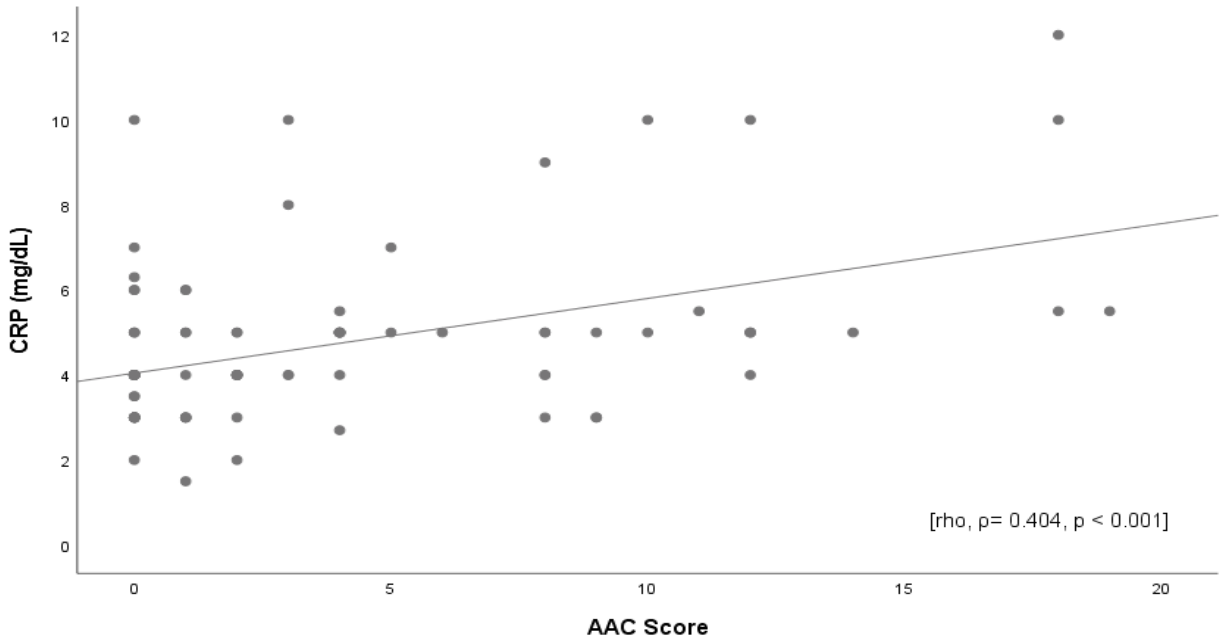

Figure (10). Association between AAC score and CRP

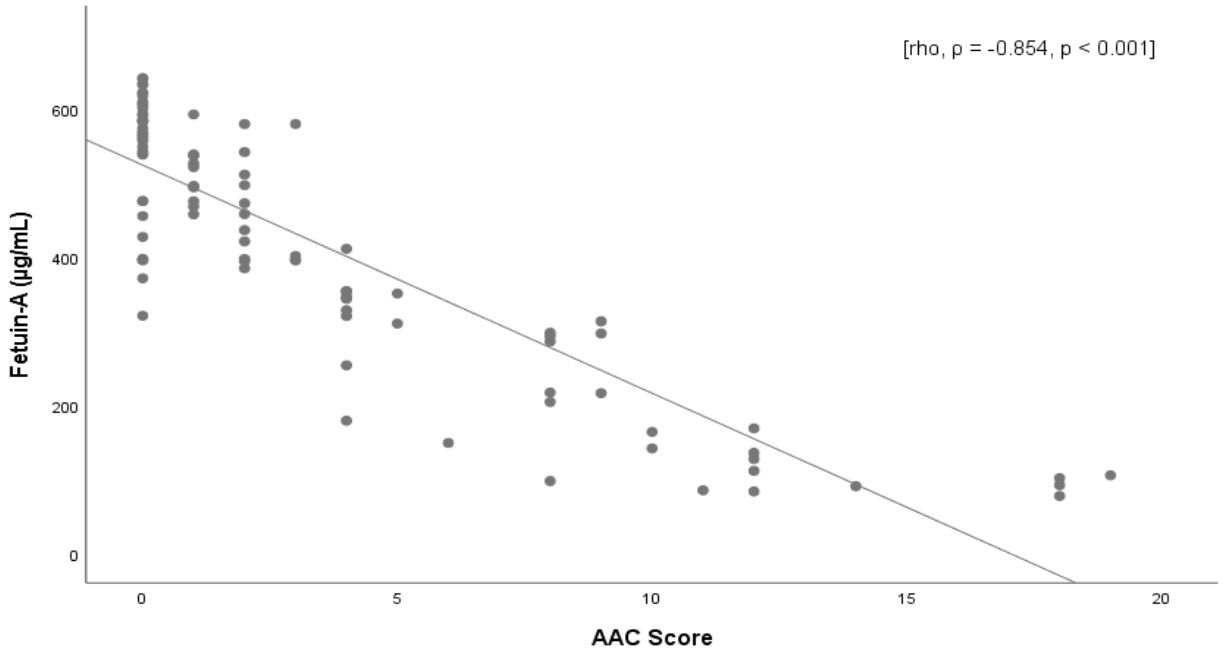

Figure (11) Association between AAC score and serum Fetuin-A

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