Excess Epicardial Fat [Cardio Obesity] Is Major Risk Marker for Cardiovascular Disease: Novel Data from our Center

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Cardiobesity is novel Risk factor
SARASOTA MEMORIAL HOSPITAL

Over 800 Beds
Over 800 Physicians on Staff
4000 Employees
20th Largest Public Hospital in US
27,000 Admissions per year
3 Time Magnet Nursing Status
Number One Ranking for MI Care

Disclosure of financial relationships:

M. El Shahawy, MD, MS, FESC, FACC, FAHA

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Faculty member CME: Expert exchange and NACE

Major stock shareholder: None
Epicardial fat belong to the following category of Fat tissue:

1. **White**: Subcutaneous Tissue
2. **Brown**: *Abdominal* Visceral tissue
3. **Inhomogeneous**: Perivascular [PVAT]:
Pre-Test Question 2

Perivascular/Epicardial adipose tissue volume is very commonly increased in Obese subjects:

A. True  
B. False

Pre-Test Question 3

Epicardial adipose tissue volume may be increased in none Obese subjects:

• A. True  
• B. false
Pre-Test Question 4

Atherogenic Adipokienes secreted by the Epicardial/Perivascular adipose tissue include all of the following except:
A. Leptin
B. Resistin
C. Visfatin
D. Omentin

Pre-Test Question 5

Cardio Obesity even without Visceral abdominal Obesity or other comorbidities is a disease

• A. True
• B. false
Pre-Test Question 6
PVAT has been recently recognized as novel factor in vascular biology with implications in the pathophysiology of CVD

• A. True
• B. false

Pre-Test Question 7
The most common early structural and functional cardiovascular abnormalities found in asymptomatic subjects with Cardio Obesity [even without abdominal Visceral obesity] is/are:

A. Abnormal rise in BP post mild exercise
B. Abnormal small vessel elasticity [C2]
C. Abnormal carotid intima media thickness
D. None of the above
E. All of the above
Pre-Test Question 8

- Cardio Obesity is best assessed by:
  - A. Echo
  - B. MRI
  - C. CT

Cardio obesity and CVD:
An Association or a Cause and Effect Relationship?
Objectives


2. Understand that excess amounts of perivascular/Epicardial Adipocytes are associated with an impairment of insulin sensitivity and other co-morbidities.

Continue Objectives

3. Understand that Epicardial Adiposity even without associated co-morbidities such as Visceral abdominal adiposity, hyperglycemia, hypertension, and hyperlipidemia, are responsible for cardiovascular disease risk.

4. Understand that Excess epicardial fat is a novel CV risk factor even in the absence of abdominal Visceral obesity and or other comorbidities.
The last statement is based on new findings and data from our Cardiovascular Diseases Assessment Center in Sarasota Florida, presented at the European Society of Cardiology in Paris, France in August 2011 and AHA/Stroke International meeting in New Orleans, February 2012 & ESC/Euro-prevent, Malaga, Spain April 8, 2017.

Planned Discussion on Cardio Obesity:

- Introductory facts/statistic about Perivascular adipose tissue [Epicardial Fat]
- Pathophysiologic link between excess Epicardial Fat and Atherosclerosis [Myocardial bridges free of atherosclerosis !!!!]
- Guidelines for Diagnosis
- Novel abnormal cardiovascular findings and data on Cardio obesity without comorbidities recently published from our CVDA Center in Sarasota [ESC/EAPC April 2017 Malaga Spain]
- Conclusions and Take home messages
Types of Fat and location

1. **White**: Subcutaneous Tissue
2. **Brown**: Visceral abdominal tissue
3. **Inhomogeneous Perivascular**:
   - Periaortic
   - Perirenal
   - Epicardial

Perivascular/Epicardial Adipose Tissue may be increased in visceral obesity

- Patient with visceral obesity
  - Hypertension
  - Diabetes
  - Dyslipidemia

- Patient with peripheral obesity
  - No metabolic complications

**Definition of PVAT [Epicardial fat]**

Virtually all blood vessels are surrounded by adipose tissue; the heart and kidney are also surrounded by fat. PVAT is inhomogeneous and differ from subcutaneous and visceral fat with regard to mRNA.

PVAT encroaches directly on the adventitia without visceral layer and thus humeral factors secreted by PVAT have easy access to the blood vessel wall and can function as paracrine organ transducing directly to the vascular cells. The mass of PVAT correlate negatively with Insulin sensitivity and increase production of Atherogenic factors.

**Function of perivascular adipose tissue [PVAT]**

- PVAT is composed mainly of adipocytes which release wide range of biologically active molecules, that may regulate vascular tone, smooth muscle contraction, proliferation and migration.
- PVAT also exerts anti-contractile effect in various vascular beds which seems to be mediated by perivascular relaxing factor [PVRF].
- Complex pathways seems to regulate PVAT function and more than one mechanism is responsible for PVAT actions in vascular biology.
PVAT in Obesity

PVAT is expanded in obesity and DM not only in the size of the adipocyte but also by accumulation of inflammatory cells and shift of production of Adipokines and cytokines. Thus providing an important role in the control of vascular dysfunctions in Obesity and diabetes.

Perivascular adipose tissue [PVAT]

PVAT has been recently recognized as novel factor in vascular biology with implications in the pathophysiology of CVD
Adiponectin (Insulin Sensitizer) Associated With Inflammatory Markers (C-reactive Protein) Free Fatty Acids (FFAs)

Excess PVAT/ Epicardial Adiposity = High-Risk Fat


Adiposity induces inflammatory changes in adipose tissue

Pathophysiologic link between Cardio Obesity and Atherosclerosis

Cardio Obesity and CVD:
Cardiobesity is novel Risk factor

Fat Cells & Mediators of Insulin Resistance:

FAT CELL
Adipokines: Metabolically active molecules link Cardio Obesity and Atherothrombosis

Atherogenic:
- Leptin
- Resistin
- Visfatin
- CRP
- IL-6
- PAI-1
- Angiotensinogen
- MCP-1

Antiatherogenic:
- Adiponectin
- Omentin
- Apellin


Excess Atherogenic Adipokines Cause Endothelial Dysfunction Resulting in Vascular Disease

IGF1 = Insulin-like Growth Factor 1; ANG-II = Angiotensin II; OxLDL-C = Oxidized Low-density Lipoprotein Cholesterol; HTN = Hypertension; NO = Nitric Oxide

Pathophysiology

From Epicardial Adiposity
To Co morbidities
&
Cardiometabolic Syndrome
And
Cardiovascular events

Epicardial fat in Health and Disease

Healthy Epicardial fat secretes protective adipokines i.e., adrenomedullin and omentin while alteration in epicardial fat as seen in obesity and Diabetes secret excessive harmful hormones leading to comorbidities i.e. Diabetes, Hypertension, Dyslipidemia which ultimately contribute to subclinical and clinical atherosclerosis.

Epicardial fat is expanded in Obesity and DM not only in size of the adipocyte but also by accumulation of inflammatory cells and shift of production of Adipokines and cytokines, thus providing an important role in the control of vascular dysfunction.
Epicardial Adipositopathy

Expansion of epicardial adipose tissue which commonly seen in obesity and Diabetes may create dysfunctional Adipocytes with over production of harmful Adipokines leading to subclinical atherosclerosis and ultimately cardiovascular complication even in the absence of any comorbidities i.e. Obesity, Diabetes, Hypertension or Dyslipidemia

Time to recognize excess epicardial fat as novel CV risk factor beyond visceral adiposity

Pathophysiologic link of excess Epicardial Fat to Atherosclerosis

Excess Epicardial Adipose tissue are the link for the development of comorbidities & Cardiovascular Disease Including Atherothrombosis

Reversible risk factors

Irreversible risk factors

Sub clinical atherosclerosis

Changes in Glucose, Hypertension and Lipid metabolism

Metabolic Syndrome

Disease and CV Events
- Diabetes
- Stroke
- MI

Death
Excess of Atherogenic Adipokines can lead to Endothelial dysfunction, sub-clinical and clinical Atherosclerosis

The location of excess adiposity is an important determinant of Cardiometabolic risk

Cardio adiposity is a major novel risk factor. Why? And what is the evidence?

Recent Studies published from our center in Sarasota Florida:
Epicardial adiposity, regardless of visceral adiposity, is associated with significant cardiovascular abnormalities in untreated and asymptomatic subjects, as measured by the calcium score.

Background: It has been postulated that excess epicardial fat (ECF) is associated with cardiovascular disease (CVD) risk, yet a detailed association with calcium deposits has not been reported.

Objective: To identify the association between ECF and CVD risk, as measured by the calcium score, in both the obese and non-obese asymptomatic, and untreated subjects.

Methods: We screened 2490 asymptomatic subjects, 79% of which (1974 of 2490) were between the ages of 40 and 70, for CVD risk using ECVDRS. ECVDRS consists of 10 tests: large (C1) and small (C2) artery stiffness, blood pressure (BP) at rest and post mild exercise (PME), Carotid Intima Media Thickness (CIMT), abdominal aorta and left ventricle ultrasound (LVUS), retinal photography, microalbuminuria, ECG, and pro-BNP. 94 subjects underwent additional screening using CT scan to assess ECF in millimeters as well as calcium deposits.

Results: The mean calcium score (CS) was higher for subjects with epicardial fat, irrespective of their level of visceral adiposity. Non-obese subjects with excess ECF had an average CS of 265, whereas the non-obese with normal ECF had a mean CS of only 107. Similarly, the obese with excess ECF had a significantly higher mean CS than the obese without excess ECF. See Figure 1 for detailed results.

Conclusions: Abnormal levels of both micro and macrovascular stiffness were significantly higher in subjects with excess ECF than in those with normal ECF, irrespective of visceral obesity, indicating that excess ECF is linked with vascular dysfunction. The mean CS for non-obese subjects with excess ECF was more than two and a half times higher than that of the non-obese with normal ECF. This strongly suggests that excess ECF is associated with an elevated CS. Notably, the CS was higher in the non-obese with excess ECF than in the obese without excess ECF. This strongly suggests that epicardial adiposity—and not visceral obesity—may play a larger role in CVD structural and functional abnormalities than previously thought.
**Background**

- It has been postulated that excess epicardial fat volume (ECFV) is associated with cardiovascular disease (CVD) risk, yet a detailed association with coronary calcification [Calcium score] and other CV structural and functional abnormalities [C1, C2, CIMT and ARBP] in asymptomatic subjects [score] has not been reported.

**Objective:**

- To identify the association between excess ECFV and CVD risk, as measured by the calcium score and other parameters in both the obese and non-obese asymptomatic, and untreated subjects.
Method:

- We screened 2490 asymptomatic subjects, 79% of which (1974 of 2490) were between the ages of 40 and 70, for CVD risk using ECVDRS. ECVDRS consists of 10 tests: large (C1) and small (C2) artery stiffness, blood pressure (BP) at rest and post mild exercise (PME), Carotid Intima Media Thickness (CIMT), abdominal aorta and left ventricle ultrasound (LVUS), retinal photography, microalbuminuria, ECG, and pro-BNP. 94 subjects underwent additional screening using CT scan to assess ECFV in millimeters as well as calcium deposits.

Screening Tests for Early Detection And Determination of Cardiovascular Risk Category Include Tests for:

1. Vascular Evaluation
2. Cardiac Evaluation
3. Modifiable Disease Contributors
### Early CVD Risk score

#### Rasmussen Risk Score (RRS)

<table>
<thead>
<tr>
<th>Metric</th>
<th>Normal</th>
<th>Borderline</th>
<th>Abnormal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Resting Blood Pressure</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Exercise Blood Pressure</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Large Artery Elasticity ($C_1$)</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Small Artery Elasticity ($C_2$)</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Carotid IMT</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Retinal Vasculature</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Microalbuminuria</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Electrocardiogram</td>
<td>0</td>
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<td>2</td>
</tr>
<tr>
<td>LV Ultrasound</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>BNP</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>0-20</strong></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

### Results:

#### Comparison of Non-diabetes Subjects and Obese Subjects

<table>
<thead>
<tr>
<th>Metric</th>
<th>Non-diabetes subjects</th>
<th>Obese subjects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean Age</td>
<td>56</td>
<td>56</td>
</tr>
<tr>
<td>Mean BMI</td>
<td>26.1</td>
<td>25.8</td>
</tr>
<tr>
<td>Mean Rasmussen Risk Score (RRS)</td>
<td>4</td>
<td>7</td>
</tr>
<tr>
<td>Mean Carotid IMT</td>
<td>107</td>
<td>205</td>
</tr>
<tr>
<td>Mean LV Ultrasound</td>
<td>30</td>
<td>20</td>
</tr>
</tbody>
</table>

#### High-Fat Percentages

- **Epicardial Fat (ECF)**
  - Normal: 4%
  - Extra: 10%
- **Macro-arterial Stiffness ($C_1$)**
  - Normal: 10%
  - Abnormal: 30%
- **Micro-arterial Stiffness ($C_2$)**
  - Normal: 12%
  - Abnormal: 35%
- **Carotid Intima-Media Thickness (CIMT)**
  - Normal: 47%
  - Abnormal: 50%
Results:

• 1) The mean calcium score (CS) was higher for subjects with excess epicardial fat, irrespective of their level of abdominal visceral adiposity.
• Non-obese subjects with excess ECFV had an average CS of 265, whereas the non-obese with normal ECFV had a mean CS of only 107.

Continue Results:

• 2) The mean CS for non-obese subjects with excess ECFV was more than two and a half times higher than that of the non-obese with normal ECFV. This strongly suggests that excess ECFV is associated with an elevated CS.
Conclusion
Epicardial adiposity, in untreated asymptomatic subjects regardless of abdominal visceral adiposity, is associated with significant cardiovascular structural and functional abnormalities including abnormal calcium score.

Results: Subjects with normal epicardial fat, obese and non-obese

<table>
<thead>
<tr>
<th></th>
<th>Normal Epicardial Fat (&lt; 110mm)</th>
<th>30 people</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Level of Obesity</strong></td>
<td>Non-obese (BMI &lt; 30)</td>
<td>Obese (BMI &gt;/= 30)</td>
</tr>
<tr>
<td></td>
<td>24 subjects</td>
<td>6 subjects</td>
</tr>
<tr>
<td>Mean Age</td>
<td>58</td>
<td>57</td>
</tr>
<tr>
<td>Mean BMI</td>
<td>26.1</td>
<td>31.5</td>
</tr>
<tr>
<td>Mean Rasmussen Risk Score (RRS)</td>
<td>4</td>
<td>6</td>
</tr>
<tr>
<td>Abnormal Rise in Blood Pressure (BP Rise &gt; 30 mmHg)</td>
<td>42% abnormal</td>
<td>67% abnormal</td>
</tr>
<tr>
<td>Macro-arterial Stiffness (C1)</td>
<td>17% abnormal</td>
<td>0% abnormal</td>
</tr>
<tr>
<td>Micro-arterial Stiffness (C2)</td>
<td>12% abnormal</td>
<td>17% abnormal</td>
</tr>
<tr>
<td>Carotid Intima-Media Thickness (CIMT)</td>
<td>33% abnormal</td>
<td>50% abnormal</td>
</tr>
<tr>
<td>Mean Calcium Score</td>
<td>107</td>
<td>20</td>
</tr>
</tbody>
</table>
Non-obese subjects with normal and excess levels of epicardial fat (ECF)

<table>
<thead>
<tr>
<th>Epicardial Fat (ECF)</th>
<th>Non-obese subjects (BMI &gt;/= 30) 47 people</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Normal ECF ECF &lt; 110 mm 6 subjects</td>
</tr>
<tr>
<td></td>
<td>Excess ECF ECF &gt;/= 110mm 41 subjects</td>
</tr>
<tr>
<td>Mean Age</td>
<td>58</td>
</tr>
<tr>
<td>Mean BMI</td>
<td>26.1</td>
</tr>
<tr>
<td></td>
<td>60</td>
</tr>
<tr>
<td></td>
<td>25.8</td>
</tr>
<tr>
<td>Mean Rasmussen Risk Score (RRS)</td>
<td>4 7</td>
</tr>
<tr>
<td>Abnormal Rise in Blood Pressure (BP Rise &gt; 30 mmHg)</td>
<td>42% abnormal 78% abnormal</td>
</tr>
<tr>
<td>Macro-arterial Stiffness (C1)</td>
<td>17% abnormal 30% abnormal</td>
</tr>
<tr>
<td>Micro-arterial Stiffness (C2)</td>
<td>12% abnormal 35% abnormal</td>
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<tr>
<td>Carotid Intima-Media Thickness (CIMT)</td>
<td>33% abnormal 43% abnormal</td>
</tr>
<tr>
<td>Mean Calcium Score</td>
<td>107</td>
</tr>
<tr>
<td></td>
<td>265</td>
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</tbody>
</table>

Subjects with excess epicardial fat, obese and non-obese

<table>
<thead>
<tr>
<th>Level of Obesity</th>
<th>Excess Epicardial Fat (&gt;/= 110mm) 64 people</th>
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<tbody>
<tr>
<td></td>
<td>Non-obese (BMI &lt; 30) 23 subjects</td>
</tr>
<tr>
<td></td>
<td>Obese (BMI &gt;/= 30) 41 subjects</td>
</tr>
<tr>
<td>Mean Age</td>
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<td>Mean BMI</td>
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<tr>
<td>Abnormal Rise in Blood Pressure (BP Rise &gt; 30 mmHg)</td>
<td>78% abnormal 71% abnormal</td>
</tr>
<tr>
<td>Macro-arterial Stiffness (C1)</td>
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<td>43% abnormal 54% abnormal</td>
</tr>
<tr>
<td>Mean Calcium Score</td>
<td>265</td>
</tr>
<tr>
<td></td>
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</table>
### Abnormal rise in BP Non-obese subjects with normal and excess levels of epicardial fat (ECF)

<table>
<thead>
<tr>
<th>Epicardial Fat (ECF)</th>
<th>Non-obese subjects (BMI &lt; 30)</th>
</tr>
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<tbody>
<tr>
<td></td>
<td>47 people</td>
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<tr>
<td></td>
<td>265</td>
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</tbody>
</table>

**Award for scientific research on Epicardial fat from European Society of Cardiology April 2017**

![Award Certificate Image](image-url)
Studieds on obesity published from our center:

1. Is Epicardial Fat Volume Gender Specific? Is Its Excess a Villain for Cardiovascular Disease Risk in Metabolic Syndrome subjects?, presented at the 6th Annual Scientific Meeting of the Society of Cardiovascular Computed Tomography [SCCT], July 2011

2. Epicardial adiposity, regardless of visceral adiposity, is associated with significant cardiovascular abnormalities in untreated and asymptomatic subjects, as measured by the calcium score - April 8, 2017, Malaga/Spain

Annual Meeting of the European Congress on Preventive Cardiology, EuroPrevent in Malaga, Spain, April 6th-8th, 2017

Other recent studies on obesity without comorbidities published* from our Cardiovascular Health Assessment Center in Sarasota, Florida

- Decades of obesity even without comorbidities in asymptomatic subjects is associated with significant cardiovascular structural and functional abnormalities - April 6, 2017, Malaga/Spain
**Background:** Does the decade of obesity presence affect the extent of CV structural and functional abnormalities in asymptomatic subjects without comorbidities?

**Objective:** To determine whether obesity of longer duration in asymptomatic subjects without comorbidities is associated with greater CV structural and functional abnormalities than in the younger cohort, as assessed by the Early CVD Risk Score (ECVDRS) system, also known as the Rasmussen Risk Score (RRS).

**Methods**
We screened 2480 asymptomatic subjects, 79% of which (1974 of 2490) were between the ages of 40 and 70, for CVD risk using ECVDRS. ECVDRS consists of 10 tests: large (C1) and small (C2) artery stiffness, blood pressure (BP) at rest and post mild exercise (PME), Carotid Intima Media Thickness (CIMT), abdominal aorta and left ventricle ultrasound (LVUS), retinal photography, microalbuminuria, ECG, and pro-BNP. Comorbidities also measured, but not factored into the risk score, include abnormal cholesterol, abnormal blood pressure, and obesity, defined by BMI.

**Results**
Within obesity classes, subjects who were older exhibited higher levels of structural and functional CV abnormalities. Notably, among subjects who were overweight, mild or moderately obese, those in their 70’s had risk scores two times as high as those in their 40’s. See table for full results.

**Conclusions**
Among subjects of the same BMI class, older subjects consistently exhibit more structural and functional CV abnormalities. This is significant, as the ECVDRS metrics are corrected for age.

1) This evidence contradicts one of the main claims of the “obesity paradox” that mild to moderate obesity is protective in older populations. In fact, it appears to increase the likelihood of having structural and functional CV abnormalities.

2) As obesity is a disease that begins in childhood, we can conclude that while being obese is a major risk factor for CVD, the length of time that one is obese is an even bigger risk factor. Thus, we believe it is crucial to initiate obesity reduction early in life for optimal risk mitigation.

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**Table 1: Average Early CVD Risk Score in Obese Subjects without Comorbidities.**

<table>
<thead>
<tr>
<th>BMI Class</th>
<th>Average Risk Score</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
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</table>

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**References**
- The study was supported by the Cardiovascular Center of Sarasota Foundation for Research and Education.

**Presented at EuropePrevent 2017: European Association of Preventive Cardiology, April 6-8, 2017, Malaga, Spain.**

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**Conclusions and CLINICAL Perspective on Epicardial fat**

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Take Home Messages

Take Home Messages
TAKE HOME MESSAGES

#1

Excessive production of the Atherogenic Adipokines and Angiotensinogen are the pathophysiological link between Cardio Obesity, Metabolic syndrome and Atherosclerosis

#2

Early identification and treatment of cardiovascular abnormalities associated with Cardio Obesity, can now be achieved with cutting edge tools and dedicated and specialized team, in this rapidly evolving area of cardiovascular prevention.
TAKE HOME MESSAGE

# 3
Cardio Obesity with and without Visceral obesity even without other co-morbidities is associated with structural and functional CV/Stroke risk markers far and beyond those seen obese subject without cardio obesity
[cardio Obesity is a significant cardio and cerebrovascular disease risk marker]

TAKE HOME MESSAGE

# 4
Medical management must shift its emphasis from treatment of advanced disease to early prevention of disease progression.

One ounce of Early CVD/Stroke prevention is better than pounds of late cure
Time to Focus on early CVD prevention
Evidence for early CVD/Sub clinical Atherosclerosis in Asymptomatic Obese or non obese subjects Still:

• Under Diagnosed
• Under Treated
• Under Controlled

Time for a change
The new practice pattern for CV risk reduction in the 21st century:

All health care providers must focus on Early detection of subclinical atherosclerosis keeping in mind that one ounce of early detection of CVD is better than pounds of late Cure
Epicardial Adiposity

Time to recognize excess epicardial fat as novel CV risk factor beyond visceral adiposity

Cardiobesity is novel Risk factor
Final departing Recommendation

Donot ever forget that:

One ounce of Early cardiovascular prevention is better than

pounds of cure

Today we must practice
Evidence-based Medicine
We are ALL

Preventive Health Care Providers

Children Should Know Their Grandparents and Become Great Grandparents Themselves
2004
Grand Parents should live long enough
to enjoy their grand children and grand children should grow
up to know their grand parents
[ December 2008]
Thank You

Improvement Never Ends . .

This material was prepared by Florida Medical Quality Assurance, Inc., under contract with the Centers for Medicare & Medicaid Services (CMS), an agency of the U.S. Department of Health and Human Services. The contents presented do not necessarily reflect CMS policy.