Childhood Obesity: Current Perspectives

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UAMS
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Discussion Points

• History of Obesity
• Trends
• Type 2 Diabetes a comorbid condition
Beginning the Journey
Why Worry About Childhood Obesity?

- Epidemiology
  - Worldwide over a billion people are obese or overweight (data from the International Obesity Task Force)
  - 17 – 20% of US children are obese
  - Over 2/3 of obese children (10 yrs and older) will become obese adults
- Medical complications
- Detrimental social stigmata
Bias and Discrimination

- Society as a whole does not tolerate obesity or those affected by it
- 28% of teachers sampled in one survey stated that becoming obese was the ‘worst thing that could happen to a person’
- 24% of nurses questioned said they were ‘repulsed’ by obese people
- Parents provide less college support for their overweight children than their lean offspring
- Other areas of bias include in the workforce, adoption proceedings, jury selection, and housing

History of Obesity

• Weight gain, obesity and the knowledge of associated comorbidities have been documented for thousands of years.
• History shows how we have accepted and then rejected obesity.
  – Some societies view weight gain as a indicator of wealth.
Ancient Egypt’s View of Obesity
2000 BC

• Even though they believed obesity to be a sign of prosperity many statues and depictions of prominent leaders did not show them as such.
  • Queen Henut-Tawy

• This wall relief from Karnac (along the Nile close to the Valley of Kings) shows an obese man with gynecomastia and prominent pannus

• Evidence suggests the Egyptians recognized obesity to be a cause of certain medical problems
Chinese Medicine
300 AD

• Acupuncture as a treatment strategy for obesity
  – Sharp object in the pinna of the ear to reduce appetite
Greco-Roman Medicine
360 BC

• Dionysius – tyrant of Heracleia of Pontius
  – First historical figure with “obesity and somnolence”
  – Servant inserted “long needles” to wake him up when he fell asleep

• Magas – King of Cyrene (258 BC)
  – “weighted down with monstrous masses of flesh” died of asphyxiation.
1700

- 1150 – 1450 – Medieval medicine teachings from Aristotle on the 4 humors (temperaments)
- Bonetus publishes some of the first anatomical descriptions of obese individuals – 1700 AD
William Banting

• A carpenter and undertaker
• By 1862, at the age of 65, he weighed 202 lbs (14st 6 lbs) and was 5 ft 5 ins tall.
  – "I could not stoop to tie my shoes, so to speak, nor to attend to the little offices humanity requires without considerable pain and difficulty which only the corpulent can understand, I have been compelled to go downstairs slowly backward to save the jar of increased weight on the knee and ankle joints and have been obliged to puff and blow over every slight exertion, particularly that of going upstairs."

• Popularized weight loss diets and wrote a very popular pamphlet
• Letter on Corpulence (1864).
  – sold 63,000 copies in UK alone
  – also translated and sold heavily in France, Germany and USA.
  – at least 1800 readers wrote testimonials supporting his assertions and praising his diet
Banting Diet

- **Banting's Diet** (devised for him by Dr. Harvey)
  - Animal food, 13-16 oz.; bread, 2 oz.; **fruit and vegetables**, 6-12 oz.; total **fluid**, 35 oz.
    - 9 a.m.....A large cup, 9 oz., of **tea** without milk or sugar.
    - 1 oz. of toast; 4-6 oz. of beef, mutton, **kidneys** or broiled fish.
    - 2 p.m.....Two or three glasses, 10 oz., of claret, sherry or madeira. 1 oz. of dry toast; 5-6 oz. of lean meat, poultry, game or fish; any vegetable **except potato, parsnip, carrot, turnip or beetroot**; unsweetened cooked **fruit** out of a pudding.
    - 6 p.m.....A large cup of plain tea, 9 oz.; 2 or 3 oz. of cooked fruit, and toast.
    - 9 p.m.....A glass or two, 7 oz., of claret or sherry and **water**;
    - 3 or 4 oz. of meat or fish as at dinner.
    - A glass of grog (weak beer), without sugar, or a glass or two of claret or sherry was allowed as a nightcap.

- **Not Allowed**
  - Pork or veal; eels, salmon and herrings; champagne, port and beer; certain vegetables above
1920s – 1940s

• Public view of obesity fluctuates

• French designer Paul Poiret regarded overweight as “unfashionable.”

• Flappers rebelled against idea of the “Victorian Woman” and began to diet

• During WWII, women wanted to appear stronger
  – Rosie the Riveter
Then Came Insurance,…

• In the 1940s, Metropolitan Life Insurance developed a chart depicting ideal body weight for various heights.
  – Noted continued weight gain throughout life not acceptable and associated with health risks
  – Reproduced in the 1950s and 1980s
  – Government and medical community direct programs against obesity

• After WWII, people begin to lose the desire to remain thin
  – Immigrants from Europe
  – Hollywood (Marilyn Monroe)
the Federal Government,…

- NHES (1959 – 1970) was designed to secure statistics on the health status of the U.S. population including heights and weights in children aged 6 – 17 years of age
- NHANES (1970s - ) follows health and nutrition status of U.S.
- Government and medical societies begin to take notice of increase in weight gain for population with data charted around 1985
- BMI chart published in 1996
and the Media.

• In 2004, Super Size Me released
Figure 2. Trends in overweight, obesity and extreme obesity, ages 20-74 years

Note: Age-adjusted by the direct method to the year 2000 US Bureau of the Census using age groups 20-39, 40-59 and 60-74 years. Pregnant females excluded. Overweight defined as 25<=BMI<30; obesity defines as BMI>=30; Extreme obesity defines as BMI>=40.
Arkansas 2016

- 35.7% of adult population is obese
- 28.6% of young adults (18 – 25 y/o)
- 20% 10 – 17 y/o, 14.4% 2 – 4 y/o
- Ranked 3rd of 51 states or territories for obesity
- Ranked 4th in United States for Diabetes
- DM
  - 265,417 cases in 2010
  - 381,937 cases projected by 2030
Type 2 Diabetes as an Example of Comorbidity
## Current Definition of Diabetes Mellitus (ADA)

<table>
<thead>
<tr>
<th>IFG</th>
<th>IGT</th>
<th>Diabetes</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>FPG (mg/dL)</strong></td>
<td>100 - 125</td>
<td>NA</td>
</tr>
<tr>
<td><strong>PCPG (mg/dL)</strong></td>
<td>NA</td>
<td>140-199</td>
</tr>
</tbody>
</table>
Risk Factors

• Ethnicity - 2/3 patients are AA or MA
• Family History - 60 - 80% have at least one 1st degree relative
• BMI
• Sedentary lifestyle
• Low fiber, high fat diet
• Gestational diabetes
• IUGR - IR and β-cell dysfunction
• Puberty – noted 30% reduction in insulin sensitivity in healthy children
• Acanthosis Nigricans
Acanthosis Nigricans
Age-adjusted Percentage of U.S. Adults Who Were Obese or Who Had Diagnosed Diabetes

**Obesity (BMI ≥30 kg/m²)**

- **1994**
- **2000**
- **2008**

**Diabetes**

- **1994**
- **2000**
- **2008**

Pathophysiology of Type 2

- A heterogeneous disorder produced from both peripheral insulin resistance and β-cell dysfunction (relative insulin deficiency)

- The temporal development of type 2 diabetes varies between genders, ethnic groups, stage of puberty, and BMI

- The preceding event seems to be the evolution of insulin resistance and then worsening glucose tolerance, which may predate DM by several years

JCEM 94(7): 2215 - 2220, 2009
Clinical Features

- Most present with evidence of hyperglycemia
- 33% may be picked up on routine lab work
  - Rare in Type 1 Diabetes
- Most are obese
  - 33% BMI > 40 kg/m²
  - 17% BMI > 45 kg/m²
- Mean age 13 - 14 yrs (rare case < 10 yrs)

Obesity Management: 170 - 173, 2007
# Clinical Features

<table>
<thead>
<tr>
<th></th>
<th>T1DM</th>
<th>T2DM</th>
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<tbody>
<tr>
<td>Ketonuria/DKA</td>
<td>Yes</td>
<td>* Up to 42%</td>
</tr>
<tr>
<td>Autoimmune Markers</td>
<td>Yes</td>
<td>10 - 15% (12% ICA or GAD, 4% both)</td>
</tr>
<tr>
<td>Accidental Dx</td>
<td>Rare</td>
<td>33%</td>
</tr>
<tr>
<td>Age at Dx</td>
<td>Peaks 7 &amp; 14 yrs, but any age</td>
<td>Usually during puberty</td>
</tr>
<tr>
<td>Wt</td>
<td>Wide range</td>
<td>Most obese</td>
</tr>
<tr>
<td>AN</td>
<td>Rare</td>
<td>60 - 90%</td>
</tr>
<tr>
<td>HTN</td>
<td>Rare</td>
<td>55% preHTN, 49% HTN</td>
</tr>
<tr>
<td>Microalb</td>
<td>Uncommon</td>
<td>22% in one series</td>
</tr>
<tr>
<td>Dyslipidemia</td>
<td>Uncommon</td>
<td>18 - 27%</td>
</tr>
<tr>
<td>Steatohepatitis</td>
<td>Uncommon</td>
<td>8 - 48%</td>
</tr>
</tbody>
</table>

Pediatric Clin N Am 52: 1579 - 1609, 2005  * More common in AA and Hispanic population
Life-expectancy in Type 2 Diabetes

- *Subjects in NHANES I (1971 - 75) followed for 22 years
  - 3.6 fold increase in mortality in patients aged 25 - 44 years
- **1996 - 1997 HRQOL measured in Ontario Diabetes Database
  - Death rate 2-times higher in patients with diabetes
  - Estimated life lost due to diabetes was 2.8 years for men and 2.6 years for females
  - Effects on HALE more impressive (58.3 years vs 70.2 years in males with and without diabetes respectively)

*Diabetes Care 21: 1138 - 1145, 1998
**Diabetes Care 27: 407 - 414, 2004
Choosing the Appropriate Therapy for Type 2

• Symptomatic or Ketotic?
• Efficacy of therapy?
• Hemoglobin A1c – stage of Diabetes
• Diet, exercise, medication?
Diet and exercise

- Restrict concentrated sweets (sodas, candy, juices, etc), reduce snacks and limit fast food.
- Portion sizes discussed
- Carbohydrates monitored, but not as strict as constant carb diets for Type 1 patients
- Patients with dyslipidemia counseled on lowering fats and fried foods
- Exercise (per DPP) to be at least 30 - 60 minutes per day.
  - My 3 rules are
    - Sweat
    - Increased HR
    - Safety
Oral Hypoglycemics

• If, hemoglobin A1c reasonable (< 9%) begin oral monotherapy immediately
  – Metformin
  – SUR

• Which one?
# Efficacy of Oral

<table>
<thead>
<tr>
<th>Type</th>
<th>Action</th>
<th>HgA1c Reduction</th>
</tr>
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<tbody>
<tr>
<td>Metformin</td>
<td>Lowers Hepatic Glucose Production</td>
<td>0.8 - 3.0</td>
</tr>
<tr>
<td>SUR</td>
<td>Augments Insulin Secretion</td>
<td>0.9 - 2.5</td>
</tr>
<tr>
<td>TZD</td>
<td>Lowers Glucose Production and uptake by unclear mechanism</td>
<td>1.1 - 1.6</td>
</tr>
<tr>
<td>AGI</td>
<td>Inhibits CHO absorption (no direct action on BG)</td>
<td>0.4 - 1.3</td>
</tr>
<tr>
<td>Insulin</td>
<td>Lowers BG Directly</td>
<td>Compliance?</td>
</tr>
</tbody>
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Insulin

- If HgA1c persistently > 7% and not responding to oral tx or initial HgA1c above 10%
  - Chosen as first line therapy if child presents with ketosis or ketoacidosis (possible if β-cell loss and glucotoxicity prevalent)
- Well tolerated, but higher risk of hypoglycemia and more technically challenging
- Beginning dose depends on age, weight, stage of Diabetes, and pubertal status (also insulin infusion rate on drip), but can range between 0.5-1.2 units/kg/day split into 3 - 4 SQ injections
- Rapid acting (Novolog or Humalog) plus Lantus or Levemir
Follow up

• For children with any form of DM
  – Routine Follow Up is every 3 months
  – Dieticians see them yearly or if issues arise
  – Education classes offered

• Some require closer supervision
# Attrition at CMC

<table>
<thead>
<tr>
<th>Group</th>
<th>Lost to Follow Up</th>
<th>Age at Last Visit</th>
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<tbody>
<tr>
<td>Oral</td>
<td>81%</td>
<td>15.0</td>
</tr>
<tr>
<td>Oral to insulin</td>
<td>31%</td>
<td>15.3</td>
</tr>
<tr>
<td>Insulin</td>
<td>54%</td>
<td>16.4</td>
</tr>
<tr>
<td>Insulin to oral</td>
<td>30%</td>
<td>16.7</td>
</tr>
</tbody>
</table>
And so...