The impact of psychotropic medications on the physical health of those with serious mental health conditions

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Overview

- Medications used in SMI
- Basic pharmacology of antipsychotic drugs
- Psychotropic medication side effects
- Combating weight gain and metabolic changes as a result of medication
- Implementing the new NICE recommendations with regard to medications and physical health
- Health promotion initiatives that work to reduce the impact on physical health
Medications used in SMI

- **Antipsychotics** – FGAs e.g. haloperidol, chlorpromazine; SGAs e.g. olanzapine, risperidone
- **Mood stabilisers** – lithium, sodium valproate, carbamazepine, lamotrigine, topiramate
- **Benzodiazepines** – diazepam, lorazepam, clonazepam
- **Antihistamines** - promethazine

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Side effect (Oxford English Dictionary)

• An effect (usually for the worse) of a drug other than that for which it has been administered.
• Can be avoided by using the most appropriate drug and dose
• As regards the dosage, the choice is based on previous history, physical condition, age and clinical evolution.

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Antipsychotic receptor binding and physical health

Cardiometabolic side effects, including weight gain, insulin resistance, and increased fasting triglycerides.

Increased Prolactin

Tardive Dyskinesia

EPS

From Stahl 2013

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Comparative Receptor Binding Profiles

- **Haloperidol**
  - A2, H1, D1
  - 5HT1A, 5HT2A

- **Clozapine**
  - M, D1, D2
  - H1, A2, 5HT1A

- **Seroquel**
  - D1, D2
  - 5HT2A, 5HT1A, A1

- **Olanzapine**
  - D1, D2
  - 5HT2A

- **Sertindole**
  - A1, D1
  - 5HT1A, A2, 5HT2A

- **Ziprasidone**
  - A1, D2
  - 5HT1A, 5HT2A

- **Risperidone**
  - A2, H1, D1, D2
  - 5HT1A, 5HT2A

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Reasons for variation in AP side effects

- Different receptor binding profiles
- Extent and site of D2 blockade
- Atypical APs – don’t cause EPS at clinically effective doses
- Pharmacodynamic and pharmacokinetic effects in individuals
- Idiosyncratic reactions in individuals
- Prescribing factors in doctors
- Dose-related side effects
Common physical conditions co-morbid with SMI

- **CVS disease** (Davidson 2002, Lambert 2003)*
- **Diabetes** (5x higher rates in SMI – Expert Consensus BJPsych 2003)*
- **Obesity** (36-75% obese, Cormac et al 2004)*
- **Hyperlipidaemia** (antipsychotics, Meyer 2002)*
- **Malignant neoplasms** (Halbreich 1996, Harris 1998)
- **HIV/AIDS** (4-23%, Davidson 2001)*
- **Hepatitis C**
- **Osteoporosis***
- **Hyperprolactinaemia***
- **Gastrointestinal disorders – IBS, H. Pylori** (19% IBS, 3x H. Pylori, Gupta 1997, De Hert 1997)

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Dopamine blockade -> raised Prolactin (hyperprolactinaemia)

- Anxiety
- Depression
- Growth
- Weight gain

Prolactin reduces:
- Testosterone levels
- Oestrogen levels
- Progesterone levels
- Infertility (reduced ovulation & sperm synthesis)
Effect of hyperprolactinaemia on sexual and reproductive function

- Reduced libido
- Hypogonadism
- Infertility
- Prolactin elevation
  - Erotic dysfunction
  - Ejaculatory dysfunction
  - Vaginal response abnormalities
  - Anorgasmia

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Long-term hypo-oestrogenic states in women associated with

- Reduced energy
- Skin changes
- Sexual dysfunction
- Osteoporosis
- Increased risk of cardiovascular disease

Antipsychotics ~ ?premature ageing in women

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Osteoporosis

- Progressive skeletal disease characterized by low bone density, with increase in bone fragility and susceptibility to fracture (WHO, 1994)
- 200,000 fractures per year in UK
- 40,000 hip fractures per year in UK
- ¼ women will die following hip fracture
- >2 million people in the UK
- >1/3 women will suffer from an osteoporotic fracture during lifetime
- 1/6 men affected
- Increases with age
Risk factors for Osteoporosis

- Smoking*
- Excess alcohol*
- Hypogonadism*
- Sedentary life style*
- Steroids
- Malabsorption
- Missing periods >6 months (excluding pregnancy) or hypogonadism in males should be investigated (Royal College of Physicians 1999)

- 25% patients with schizophrenia have had 1 or more atraumatic osteoporotic fracture (Abraham et al, 1996)
Figure 3.6  SEM of normal trabecular bone showing thick trabecular plates, which are all interconnected. Courtesy of Professor Alan Boyd.

Figure 3.7  SEM of osteoporotic trabecular bone showing marked thinning and disconnection of trabeculae. Courtesy of Professor Alan Boyd.
Antipsychotics, prolactin and BMD
(Meaney et al 2004)

- 57% males and 32% females osteopenic or osteoporotic in one or more vertebrae
- High prolactin associated with high doses of prolactin-raising medication
- Low BMD associated with high prolactin and low gonadal hormone levels.
Individual second-generation antipsychotics and prolactin

**Prolactin-raising**
- **Risperidone** - rises in prolactin similar to or greater than haloperidol (Claus et al 1992, Breier et al 1999)
- **Amisulpride** - large increases in prolactin

**Transiently prolactin-raising**
- **Olanzapine** - mild-mod increases (David et al 2000)
- **Ziprasidone** - transient rises in prolactin (Goff et al 1998, Miceli et al 2000)
- **Sertindole** - transient rises in prolactin

**Prolactin-sparing**
- **Quetiapine** - prolactin similar to placebo at all doses (Arvanitis et al 1997, Peuskens and Link 1997, Emsley et al 2000)
- **Clozapine** - no significant rises in prolactin (Breier et al 1999, Curtis et al 1995)
- **Aripiprazole** – no significant rises in prolactin. Can reduce hyperprolactin caused by other antipsychotics (Byerly et al 2009)
Other side effects

- Diabetes
- Lowered seizure threshold
- Myocarditis
- Neutropenia and other blood dyscrasias
- Agitation and anxiety
- Photosensitivity
- Neuroleptic malignant syndrome (NMS)
- Jaundice
- Cerebral oedema
Blood dyscrasias

- Can happen with any antipsychotic
- Agranulocytosis with clozapine can be fatal
- Low WCC in black pts cf white pts may be “Benign ethnic neutropenia”
- Lithium can help to increase white cell count
GI disorder

- Nausea common
- Constipation common
- Death from bowel obstruction – inc in clozapine, therefore treat constipation vigorously
Metabolic Syndrome (syndrome of chronic cardiovascular risk)

- Insulin resistance &/or impaired fasting glucose &/or impaired glucose tolerance AND 2 or more of:
  a. BMI > 30kg/m²
  b. Triglyceride level > 1.7mmol/l OR HDL <0.9 mmol/l in men and <1.0mmol/l in women
  c. BP > 140/90
  d. Microalbuminuria

- Core features
  - Obesity – central or upper body
  - Dyslipidaemia
  - Impaired glucose tolerance
  - Insulin resistance
  - Hypertension

(WHO criteria, from Alberti & Zimmet 1998)

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Cardiovascular risk factors – overview

BMI = body mass index; TC = total cholesterol; DM = diabetes mellitus; HTN = hypertension.


BMI >27  Smoking  TC >220  DM  HTN

Odds ratios

The Framingham Study

Single Risk Factors

Multiple Risk Factors

Smoking + BMI  Smoking + BMI + TC >220  Smoking + BMI + TC >220 + DM  Smoking + BMI + TC >220 + DM + HTN

0  2  3  4  5

5

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Metabolic syndrome increases CV morbidity and mortality

Metabolic syndrome present | Metabolic syndrome absent

<table>
<thead>
<tr>
<th>Condition</th>
<th>Prevalence (%)</th>
<th>Incidence (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>CHD</td>
<td>5.5</td>
<td>2.1</td>
</tr>
<tr>
<td>Previous MI</td>
<td>9*</td>
<td>4.8*</td>
</tr>
<tr>
<td>Previous stroke</td>
<td>2.1</td>
<td>1.4</td>
</tr>
</tbody>
</table>

* p<0.001

Total mortality: 18* CV mortality: 12*
Weight gain significantly increases diabetes risk

Relative risk of type 2 diabetes according to BMI in women aged 30–55 years

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Weight gain and antipsychotics

- Weight gain is a well established side effect of antipsychotic therapy reported in up to 50% of patients.\(^1\)
- Weight gain liability varies significantly between antipsychotics
  - Atypical antipsychotics generally cause more weight gain than conventional agents
- Weight gain causes 416 extra deaths per 100,000 clozapine patient years, compared to 492 saved by suicide prevention.\(^2\)
ADA consensus on antipsychotic drugs and obesity

<table>
<thead>
<tr>
<th>Drug</th>
<th>Weight Gain</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clozapine</td>
<td>+ + +</td>
</tr>
<tr>
<td>Olanzapine</td>
<td>+ + +</td>
</tr>
<tr>
<td>Risperidone</td>
<td>+ +</td>
</tr>
<tr>
<td>Quetiapine</td>
<td>+ +</td>
</tr>
<tr>
<td>Aripiprazole*</td>
<td>+/-</td>
</tr>
<tr>
<td>Ziprasidone*</td>
<td>+/-</td>
</tr>
</tbody>
</table>

+ = increased effect; - = no effect

*Newer drugs with limited long-term data. Precise risk estimates are not available.

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Short-term mean weight change with antipsychotics

Estimated weight change at 10 weeks on “standard” dose

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One-year weight gain: mean change from baseline weight
## ADA consensus on antipsychotic drugs and diabetes

<table>
<thead>
<tr>
<th>Drug</th>
<th>Diabetes Risk</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clozapine</td>
<td>+</td>
</tr>
<tr>
<td>Olanzapine</td>
<td>+</td>
</tr>
<tr>
<td>Risperidone</td>
<td>+</td>
</tr>
<tr>
<td>Quetiapine</td>
<td>+</td>
</tr>
<tr>
<td>Aripiprazole*</td>
<td>-</td>
</tr>
<tr>
<td>Ziprasidone*</td>
<td>-</td>
</tr>
</tbody>
</table>

+ = increased effect; - = no effect; D = discrepant results.

*Newer drugs with limited long-term data. Precise risk estimates are not available.

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Mechanisms for antipsychotic-associated diabetes

- Weight gain (clozapine & olanzapine)$^1$
- $\uparrow$ Insulin resistance (clozapine & olanzapine)$^2$
- ? Direct metabolic effect (DKA)
- Receptor antagonism
  - central dopamine D$_2$ receptors
  - 5-HT receptors
- Chemical damage to the pancreas (pancreatitis)$^3$
- $\uparrow$ Leptin

$^1$Czobar et al, 2002; $^2$Newcomer et al, 2002, $^3$Koller et al 2003

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Obesity, dyslipidaemia and SMI

• Weight gain is usually associated with increase in lipids
• Prevalence in SMI is poorly characterized
• Insulin resistance is a key factor in dyslipidaemia
Dyslipidaemia and SMI

• Good fat = High Density Lipoprotein (HDL)
• Bad fat = triglyceride and Low Density Lipoprotein (LDL)
• Inc TG – independent risk factor for MetSyn
• Low HDL – independent risk factor for MetSyn
• 10% reduction in cholesterol ~ 20-30% reduction in CVS risk
• 10% increase in cholesterol ~ 20-30% increase in CVS risk

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Antipsychotics and dyslipidaemia

- Prevalence of dyslipidaemia poorly characterised in schizophrenia
- Several reports note elevated cholesterol and triglycerides with clozapine, olanzapine, quetiapine and risperidone
- Weight gain is usually associated with increase in lipids
- Antipsychotics may cause hyperlipidemia independent of changes in weight

Obesity, hypertension and SMI

• High rates of hypertension in SMI
• Hypertension occurs despite blood pressure lowering effect of many antipsychotics
• 1kg weight loss ~ 1-2mmHg decrease in BP
CATIE Study, n=1493

- 27% hypertension
- 14% diabetes
- 50% triglyceride abnormalities
- 68% cigarette smokers
- 10 yr CHD risk significantly higher than in matched controls
- **Women have more metabolic risk factors than men!** (Enger et al 2004)

McEvoy et al 2005
Implementing NICE recommendations

1. Monitor everybody (and do something if you find a problem)
2. Identify and target high risk patients
3. Prevent physical health problems by preventing exposure to known risk factors
4. Provide Health Promotion Interventions as early as possible into treatment
5. Develop an effective mechanism to ensure that patients get appropriate physical health input

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1. Monitoring: The National Audit of Schizophrenia (NAS) launches a new clinical resource (Lester adaption UK)

- Developed by the NAS led by the Royal College of Psychiatrists’ Centre for Quality Improvement
- Funded by the Healthcare Quality Improvement Partnership (HQIP)
- Involving a close collaboration with the Royal College of General Practitioners and the Royal College of Nursing.

Available to Download on:
www.rcpsych.ac.uk/quality/NAS/resources
2. Prevention Strategies

Health promotion from the start of treatment
Make diet and exercise part of your prescription
as with medication or CBT (regardless of
medication being taken).
Prescribe medications with fewer metabolic
side effects
Effects of antipsychotic medications on CVD risk
(adapted from Smith, J Psychopharm 2007)

<table>
<thead>
<tr>
<th>Antipsychotic medication</th>
<th>Weight gain</th>
<th>Dyslipidaemia</th>
<th>Diabetes risk</th>
<th>Metabolic syndrome risk*</th>
<th>CVD risk</th>
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<tbody>
<tr>
<td>Haloperidol</td>
<td>+/-0</td>
<td>+/-0 (MD)</td>
<td>+/-0 (MD)</td>
<td>+/-0 (MD)</td>
<td>+</td>
</tr>
<tr>
<td>Clozapine</td>
<td>++</td>
<td>+</td>
<td>++</td>
<td>++</td>
<td>++</td>
</tr>
<tr>
<td>Risperidone</td>
<td>+</td>
<td>+/-0</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Olanzapine</td>
<td>++</td>
<td>+</td>
<td>++</td>
<td>++</td>
<td>++</td>
</tr>
<tr>
<td>Quetiapine</td>
<td>+</td>
<td>+/-0 (MD)</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Ziprasidone†</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Aripiprazole</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Zotepine†</td>
<td>++/+ (MD)</td>
<td>(MD)</td>
<td>(MD)</td>
<td>+ (MD)</td>
<td>+ (MD)</td>
</tr>
<tr>
<td>Amisulpride</td>
<td>+/-0 (MD)</td>
<td>(MD)</td>
<td>0 (MD)</td>
<td>+/-0 (MD)</td>
<td>+/-0 (MD)</td>
</tr>
</tbody>
</table>

*NCEP ATP III definition
†Not approved in the UK;
++ highly-increased effect; + medium-/low-increased effect; 0 minimal effect; MD minimal controlled data
3. Identify High Risk Patients

- Females have greater cardiovascular risk than males
- Ethnicity – black African origin, South Asian
- FH of diabetes and CVS disease
- Drug-naïve patients
- High potency antipsychotics
- High dose antipsychotics
- Polypharmacy
- Substance misuse, especially cocaine/crack/stimulants
4. Health Promotion Interventions

• Behavioural studies
  – Well-being programmes (combine diet with exercise and mental well-being)
  – CBT based approaches to weight management
  Diet and nutrition
  MI focused

Pharmacological studies (designed to attenuate AP-induced weight gain)
  – Metformin > d-fenfluramine > sibutramine > topiramate > reboxetine > amantadine > nizatidine > orlistat > metformin+sibutramine > famotidine > dextroamphetamine > fluoxetine > rosiglitazone. (Maayan et al., 2010)

• Mixed behavioural/pharmacological interventions
## Physical Health Interventions

<table>
<thead>
<tr>
<th>Author</th>
<th>Design</th>
<th>No. of pts</th>
<th>Control group</th>
<th>Duration</th>
<th>Place</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Menza et al 2004</td>
<td>Naturalistic multimodal design</td>
<td>31 (67%)</td>
<td>20 TAU</td>
<td>52 weeks</td>
<td>US</td>
<td>Sig ↓BMI, ↓wgt, ↓BP, ↓HbA1c cf TAU</td>
</tr>
<tr>
<td>Kwon et al 2006</td>
<td>RCT</td>
<td>48 (75%)</td>
<td>15 TAU</td>
<td>12 weeks</td>
<td>Korea</td>
<td>↓wgt noticed by 8/52</td>
</tr>
<tr>
<td>Pendlebury et al 2007</td>
<td>Naturalistic wgt mx group</td>
<td>93 (77%)</td>
<td>No</td>
<td>4 years</td>
<td>UK</td>
<td>Mean ↓wgt 6.2kg (sessions attended p&lt;0.0001)</td>
</tr>
<tr>
<td>Poulin et al 2007</td>
<td>Controlled study</td>
<td>59 (85%)</td>
<td>51 TAU</td>
<td>18 months</td>
<td>Canada</td>
<td>Sig improvements cf baseline cf TAU (BMI, lipids, WC, Fglc)</td>
</tr>
<tr>
<td>Smith et al 2007</td>
<td>Naturalistic</td>
<td>957 (80%)</td>
<td>No</td>
<td>2 years</td>
<td>UK</td>
<td>42% ↓wgt, sig improve in diet, activity, smoking and alcohol intake</td>
</tr>
<tr>
<td>Lee et al 2008</td>
<td>Naturalistic (33 centres)</td>
<td>232</td>
<td>No</td>
<td>12 weeks</td>
<td>Korea</td>
<td>BMI ↓ 0.98 p&lt;0.001</td>
</tr>
<tr>
<td>Chen et al 2009</td>
<td>Naturalistic</td>
<td>33</td>
<td>No</td>
<td>10 weeks</td>
<td>Taiwan</td>
<td>BMI ↓ 0.18, 1.5, 1.1 (wks 10, 24, 48)</td>
</tr>
<tr>
<td>Lindenmayer et al 2009</td>
<td>Naturalistic Modular manualised</td>
<td>275 inpts</td>
<td>No</td>
<td>36 weeks</td>
<td>US</td>
<td>↓BMI, ↓wgt (4.8lbs), ↓MSyn (25.5% to 19.6%)</td>
</tr>
</tbody>
</table>
Well-being Support Program
(WSP, Smith et al 2007)

WSP
966 patients enrolled
48.4% female, 51.6% male
7 sites across UK
General adult population, 18-65
Chronic mental illness, ie >2 years
Only 31% had physical health check in the previous year

Baseline Physical risk factors
75% overweight/obese (22% severely obese)
35% hypertensive
44% smokers
63% teetotal, 10% drank >21 units alc/week
Glucose (2% diagnosed in programme)
10% raised cholesterol
24% hyperprolactinaemic
13% abnormal LFTs
WSP findings after 2 years

- *p<0.001, **p<0.0001, NS – not significant

<table>
<thead>
<tr>
<th>Indicator</th>
<th>Baseline</th>
<th>2 years</th>
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<tbody>
<tr>
<td>BMI</td>
<td></td>
<td></td>
</tr>
<tr>
<td>systolic BP</td>
<td></td>
<td></td>
</tr>
<tr>
<td>cigarettes</td>
<td></td>
<td></td>
</tr>
<tr>
<td>activity (mins)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>diet (quality)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>self esteem</td>
<td></td>
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</tr>
</tbody>
</table>

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CVS risk factors, preliminary baseline findings – IMPaCT (2013)

- Hypertension
- Obesity (BMI>30)
- MetS
- smoking

UK General Population vs People with SMI
Other health promotion interventions that work

- John Pendlebury et al 2007 - Weight management group
- Ball et al 2001 – Weight watchers group
- McKibbin et al 2006 – DART (Diabetes Awareness and Rehabilitation Training)
- See Rethink Physical Health resources page http://archive.rethink.org/how_we_can_help/physical_health/physical_health_resources

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Initiatives that work continued...

• Broadmoor (Alan Cohen’s work)
  – Targeted intervention – used QoFs to evaluate care; identified high risk patients; needed up to date IT system; someone to co-ordinate and organise the MDT
  – CVS risk – used QRISK2 and traffic light system to determine interventions
    • Exercise
    • Diet
    • Medications – statins, metformin, antihypertensives
  – Smoking stopped at Broadmoor in 2008
  – 75% of patients smoked
  – Careful planning
    • Effects on pharmacokinetics
    • Aggression

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Broadmoor QRISK2 over the years (courtesy of Alan Cohen)

Proportion of people with QRISK2 score >20%

- In the community: 2%
- With SMI in the community: 4%
- In high secure hospitals: 6%

% of patients at Broadmoor who have a QRISK2 score that is

<table>
<thead>
<tr>
<th></th>
<th>2010</th>
<th>2011</th>
<th>2012</th>
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<tbody>
<tr>
<td>High risk</td>
<td>6.99%</td>
<td>2.96%</td>
<td>2.11%</td>
</tr>
<tr>
<td>Medium risk</td>
<td>6.45%</td>
<td>1.48%</td>
<td>2.82%</td>
</tr>
<tr>
<td>Low risk</td>
<td>86.5%</td>
<td>95.5%</td>
<td>95.1%</td>
</tr>
</tbody>
</table>
5. Develop an Effective Mechanism to deliver physical health care

• Local mental health staff trained to understand importance of ongoing monitoring and when to refer e.g. MEWS chart
• All patients registered with a GP
• Agreement with GP about speedy response to referrals and clear actions and responsibilities.
• In-house primary care clinics
• Agreements with relevant 2ndry care services, e.g. Diabetes services, Cardiologists (ECG reading arrangements)
• NICE – 2ndry care should monitor patients’ physical health for 12 months or until condition stabilized (whichever is longer)

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Management of side-effects I

- Be aware of individual drug pharmacology and likely side effects
- Inform patients about side effects including potentially sensitive issues such as sexual function.
- Use rating scales e.g. LUNSERS
- Regular wgt checks, glucose measurement
Management of side-effects II

- Sedation – usually transient, change dosing schedule, reduce dose
- Hypotension – nursing advice (not getting up too quickly, not getting out of hot baths too quickly etc). Less likely with high potency drug
- Anticholinergic effects – constipation (water, exercise, roughage), dry mouth (water, sugar-free sweets or chewing gum), sialorrhoea (sleep with towel on pillow, hyoscine or pirenzepine
Management of side-effects III

- Weight gain – pretreatment planning, healthy eating advice, exercise advise, baseline and regular weight checks, glucose checks, education re; obesity, refer to dietician or weight management clinic
- Hyperprolactinaemia – check prl, reduce dose, change to prl-sparing med
- Sexual SEs- reduce dose, change med
Interventions to reduce risk of cardiometabolic and hormonal ill-health

- Is your patient in a high-risk group?
- Take a family history. Patients with type 2 diabetes or hypertension in their family have a higher genetic vulnerability.
- Monitor, monitor and monitor again! Basic physical health checks will pick up many of the disorders associated with the metabolic syndrome. Picking these up early will allow for easier intervention (prevention is better than cure).
- Checks at baseline should include: height, weight (for body mass index), waist circumference, BP, electrocardiogram and smoking (traditional cardiovascular risk factors remain very important).
- Routine blood tests at baseline should include: fasting sample (remind patient to come in straight after getting up having had no breakfast) for glucose and lipids, plus prolactin in addition to usual standard baseline investigations. Organise a breakfast for them after the test if necessary.
- Six weeks after starting a new medication/change of medication, repeat the physical health check.
• Refer to primary care specialist (ensure that patient is registered with a general practitioner) for health promotion or physical health advice. If the problem is clearly a drug-related side effect then act according to side-effect guidelines (e.g., Maudsley prescribing guidelines).
• Prescribe exercise: it reduces the risk of a cardiac event whether the patient is obese, diabetic, etc.
• Encourage healthy diet – suggest vitamin supplements if necessary
• Use a statin. These are commonly used in patients who have the metabolic syndrome who do not have mental health problems – our patients do not have the same access to these potentially life-saving drugs.
• Consider bisphosphonates for those with osteopaenia
• Use an antipsychotic medication that is less likely to result in cardiovascular or hormonal side effects (Table).
THANKYOU!