OBSESSIVE COMPULSIVE DISORDER
PSYCHIATRIC CONSIDERATIONS
This trouble is described since ancient time

Before the eighties these symptoms belong to the psychoanalytic concept of *obsessional neurosis*

First definition appears with Diagnostic Statistical Manuel *DSM III R* in 1984 belonging to anxious disorder group

With the new DSM V (ed 2013) OCD is the base of a new group labelled *obsessive compulsive disorder and related disorders* (trichotillomania, hoarding disorder, body dysmorphic disorder...)
Obsessions (DSM V)

Are defined by

- recurrent and persistent, \textit{thoughts, urges or images} that are experienced at some time during the disturbance, as \textit{intrusive and unwanted}, and that in most individuals cause marked anxiety or distress

- the individual attempts to \textit{ignore or suppress} such thoughts, urges or images or to neutralize them with some other thought or action (i.e. by performing compulsion)
Obsessions (DSM V)

Obsessions have different themes including:

- Contamination (viruses, radiation, chemicals) or dirt fears
- Pathological doubt: fear of saying exactly the right things
- Obsession of symmetry or order
- Somatic obsessions
- Scrupulosity obsession about religion, sexuality: fear to be attracted by children
- Aggressive obsessions: fear of pushing somebody under a train or a bus or harming him/herself or somebody
compulsions (DSM V)

- Repetitive behaviors or mental acts that the individual feels driven to perform in response to an obsession or according to rules that must be applied rigidly.

- The behaviors or mental acts are aimed at preventing or reducing anxiety or distress or preventing some dreaded event or situation; however, these behaviors or mental acts are not connected in a realistic way with what they are designed to neutralize or prevent, or are clearly excessive.

- Washing checking, ordering repeating behaviors

- Mental ritual: Counting, praying, mental checking, repeating mentally
Since the eighties the interest for OCD grew promoted by:

- Epidemiological studies that rate a higher incidence among the population around 2-3% (Weissman, 1994) instead of 0.50% evaluated in 1950

- Discovery of the efficiency of serotonin treatment and Cognitive behavioral Therapy (CBT)

- Input of neuroimagery observations and experiences
4th range of psychiatric disorders

Behind phobia, depression, substance abuses

Onset: before age 25 years
Consequences of OCD

- Time consuming
- Negative effects on family and relationships
- On professional activities

Co-morbidities:
- Risks of depression, substance abuses and suicide
Etiology

Development of OCD depends on:

- Neurobiological factors
- Genetic factors
- Psychological factors
- Environmental factors
Treatments

Psychotherapy: Cognitive and behavioral therapy

Pharmacological treatment
Pharmacotherapy of OCD

Serotonin antidepressant

- First antidepressant: Clomipramine
- Selective serotonin reuptake inhibitors (SSRIs)
  - Fluoxetine
  - Paroxetine
  - Citalopram, escitalopram
  - Fluvoxamine
  - Sertraline
- Serotonin norepinephrine reuptake inhibitors (SNRIs) venlafaxine duloxetine
Pharmacotherapy of OCD

- Dosage: 3 times more / depression
- Efficiency after 2 months
- Problems of side effects and drop out
New Antipsychotic treatment

- Quetiapine
- Risperidone
- Aripiprazole
Assess severity of OCD

Mild to moderate

Option

CBT with exposure and response prevention

Satisfactory improvement

Complete initial treatment course
Consider periodic “booster” sessions of CBT with exposure and response prevention

Unsatisfactory improvement

SSRI with or without CBT with exposure and response prevention

Satisfactory improvement

Continue medication for one to two years before attempting to taper
Consider periodic “booster” sessions of CBT with exposure and response prevention

Unsatisfactory improvement

Add CBT to SSRI monotherapy
Switch to new SSRI

Unsatisfactory improvement

Switch to clomipramine (Anafranil), venlafaxine (Effexor), or mirtazapine (Remeron)
Augment with atypical antipsychotic

Severe

Initiate psychiatric referral
Cognitive and behavioural therapy of OCD

Emotion: anxiety, disgust, distress

Beliefs about responsibility, perfectionism

Situation

Behaviour: Compulsions, avoidance

Cognitions: obsessions, images, thought, urges
Cognitive dysfunctions

- Overestimation of probability of danger
- Overestimation of the consequences of negative events
- Imagination = action or reality

If I think or imagine doing something that becomes the reality: «maybe I’ve touched this bin, I’m sure I’ve touched it»
Cognitive dysfunctions

- Misinterpretation about the presence of the intrusive thoughts
- Misinterpretation about the presence of the Anxiety. Anxiety confirms the reality of the danger

- OCD patients try to suppress unwanted thoughts instead of being able to accept and let them dismiss by themselves
Behavioral dysfunction

- Physical or mental compulsions or avoidance of fearful situations will not allow the patient to experience that nothing happens if he confronts the situation without ritualizing.

- As patient feels a relief from anxiety or distress when he completes his rituals he is convinced about:
  - the efficiency of his rituals on the danger
  - the reality of this danger
Cognitive therapy includes

- Discussing the real efficiency of rituals, Re-evaluating responsibility and consequences of not performing rituals
- Today the therapy works less on the content of the thoughts and more on the acceptance of their presence (influenced by techniques coming from mindfulness therapy)
Behavioural therapy

**Exposure with response prevention (ERP)**

- **Exposure** to the thoughts, images, objects and situations that trigger anxiety, distress, obsessions and need to realize compulsions
- **Response prevention** not performing, or postponing or reducing the compulsive behaviour after the anxiety or obsessions have been "triggered."
- this is done under the guidance of a therapist

The goal is:
- to confront and to cope with anxiety
- To not achieve compulsions and to notice that with time the anxiety will decrease, the need to realize rituals decreases and any dreaded consequences happen
**OCD Hierarchy**

Make a list of situations that trigger your OCD obsessions and compulsions. Put them in order of least anxiety-provoking at the bottom, to most anxiety-provoking at the top. Give each one a rating (0-100) for how distressing it would be.

<table>
<thead>
<tr>
<th>Situations</th>
<th>Distress 0-100</th>
</tr>
</thead>
<tbody>
<tr>
<td>Picking up dog dropping with plastic bag without gloves</td>
<td>100</td>
</tr>
<tr>
<td>Picking up dog dropping with plastic bag with gloves</td>
<td>80</td>
</tr>
<tr>
<td>Going to a public toilet</td>
<td>75</td>
</tr>
<tr>
<td>Touching the outside of a bin</td>
<td>70</td>
</tr>
<tr>
<td>Shaking hands with colleagues</td>
<td>65</td>
</tr>
<tr>
<td>Touching handles on a bus</td>
<td>60</td>
</tr>
<tr>
<td>Sitting in doctor’s waiting room</td>
<td>50</td>
</tr>
<tr>
<td>Touching old coins</td>
<td>30</td>
</tr>
<tr>
<td>Touching newspaper</td>
<td>20</td>
</tr>
<tr>
<td>Shaking hands with doctor</td>
<td>10</td>
</tr>
</tbody>
</table>
The course of CBT for OCD

Different stages

- Psychoeducation (nature, cbt model of OCD role of avoidance, role of homework, importance of family involvement
- Work about motivation (25% of patients refuse exposure)
- Cognitive therapy (overestimation responsibility and consequences)
- Exposure with response prevention
Indication

- Depends on the will and motivation of the patient
- The degree of severity of the disorder
- The comorbidity
- The type of OCD (mental rituals are less sensitive to CBT)
Results

- A treatment of OCD is considered as efficient when the improvement is around 25% or more on the Yale Brown obsessive compulsive scale (YBOCS).

- 20-30% significant improvement
- 40-50% moderate improvement
- 20-40% staying ill or even getting worse
OCD is a very disabling and complex disorder with different clinical symptoms and underlying factors.

Currently available treatments stay insufficient.

We need to know more about this disorder and better understand neurobiology and psychological mechanisms to improve our psychotherapy, pharmacology, and neurosurgical treatments.
Role of cortico-striato-pallido-thalamo-cortical neuronal circuits

- Obsessive compulsive behavior with pathologies consequent on damages of basal ganglia:
  - infection Group A beta-haemolytic Streptococcus:
    - Sydenham chorea
    - PANDAS Pediatric Autoimmune Neuropsychiatric Disorders Associated with Streptococcal Infections
  - Obsessional compulsive behavior caused by bilateral pallidostriatal necrosis after an encephalopathy caused by a wasp sting
  - Basal ganglia calcifications
- Damages of prefrontal cortex precisely orbitofrontal cortex

Neuroimagery contributes to confirm these dysfunctions in cortico-striato-pallido-thalamo-cortical neuronal circuits
Role of Neurotransmitters

- Serotonin

- Experience with clomipramine (tricyclic anti depressant the only with serotonin action) with patients suffering from depression and OCD, shows an improvement about OC symptoms
Genetic factors

- Twin and family studies confirm the role of genetic factor in OCD
- Monozygotics twin pairs > dizygotic twin pairs concordance for OC symptoms
  
  Nestadt, 2000

- Lifetime prevalence higher for OCD relatives compared with control relatives (11.7 % vs 2.7%)

- More risks of tourette syndrome in family with OCD

- A "significant association" to chromosome 9 near a gene called protein tyrosine phosphokinase (PTPRD).
Modification of brain activities

CBT:

Table 1  Psychotherapy effects in OCD

<table>
<thead>
<tr>
<th>Authors</th>
<th>Trial size/interventions and pre–post interval</th>
<th>Functional imaging technique</th>
<th>Post-treatment decreases</th>
<th>Post-treatment increases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baxter et al.24</td>
<td>N= 9 CBT, N= 9 fluoxetine, N= 4 healthy controls*, all 10 ± 2 weeks</td>
<td>FDG (fluoro-deoxyglucose)-PET, resting state, normalised data, no PVC</td>
<td>Responders: right caudate; correlation between right OFC, caudate and thalamus</td>
<td>None</td>
</tr>
<tr>
<td>Schwartz et al.25</td>
<td>N= 9 CBT, 10 ± 2 weeks</td>
<td>FDG-PET, resting state, normalised data, no PVC</td>
<td>Responders: caudate bilaterally; correlation between right OFC, caudate and thalamus</td>
<td>None</td>
</tr>
<tr>
<td>Nakatani et al.26</td>
<td>N= 22 CBT (some also received clomipramine), duration based on clinical improvement</td>
<td>Xenon-enhanced CT (measures rCBF), resting state</td>
<td>Right head of caudate</td>
<td>None</td>
</tr>
<tr>
<td>Nakao et al.29</td>
<td>N= 6 CBT, N= 4 fluvoxamine, 12 weeks</td>
<td>fMRI during Stroop task and symptom provocation</td>
<td>Bilateral OFC, DLPFC, ACC (symptom provocation)*</td>
<td>Bilateral parietal cortex, cerebellum (Stroop task)*</td>
</tr>
</tbody>
</table>

*Control groups are only listed where a second measurement after an interval comparable to the treatment period was obtained, not if they only served for comparison of pretreatment effects (as, e.g., in Nakatani et al.26).

PVC: partial volume correction.

*For some parts of the PET data analysis, data were pooled with those from Baxter et al.24.

*Because of the small N, data for the CBT and fluvoxamine groups were not analysed separately.
<table>
<thead>
<tr>
<th>Study (Year)</th>
<th>N (Age)</th>
<th>Study Duration</th>
<th>Study Design</th>
<th>Outcome</th>
<th>% Improvement from Baseline on Active Treatment for OC Symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bolton et al (1983)</td>
<td>15</td>
<td>12–18 yr</td>
<td>Open trial CBT + drug or other treatment</td>
<td>7 (47%) asymptomatic</td>
<td>NR</td>
</tr>
<tr>
<td>(1995)</td>
<td>2–48 mo</td>
<td>F/U: 9–14 yr</td>
<td></td>
<td>6 (40%) much improved</td>
<td></td>
</tr>
<tr>
<td>March et al (1994)</td>
<td>15</td>
<td>8–18 yr</td>
<td>Open trial CBT + drug or other treatment</td>
<td>6 (40%) asymptomatic</td>
<td>50%</td>
</tr>
<tr>
<td></td>
<td>22 wk</td>
<td>F/U: 3–21 yr</td>
<td></td>
<td>3 (20%) much improved</td>
<td></td>
</tr>
<tr>
<td>Scahill et al (1996)</td>
<td>7</td>
<td>11–16 yr</td>
<td>Open trial BT + drug</td>
<td>7/7 showed clinically significant reduction in CY-BOCS score</td>
<td>61%</td>
</tr>
<tr>
<td>(1997)</td>
<td>9–15 wk</td>
<td>F/U: 3 mo</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Weaver and Roy (1997)</td>
<td>57</td>
<td>7–19 yr</td>
<td>Open trial CBT + drug</td>
<td>39 (68%) responders</td>
<td>60%</td>
</tr>
<tr>
<td></td>
<td>4 wk</td>
<td>F/U: 6–60 mo</td>
<td></td>
<td>(CY-BOCS score &lt;16)</td>
<td></td>
</tr>
<tr>
<td>Franklin et al (1998)</td>
<td>14</td>
<td>11–17 yr</td>
<td>Open trial CBT + drug or other treatment</td>
<td>12 (80%) responders</td>
<td>67%</td>
</tr>
<tr>
<td></td>
<td>16–18 sessions in 1–4 mo</td>
<td>F/U: 9 mo</td>
<td></td>
<td>(&gt;50% CY-BOCS score)</td>
<td></td>
</tr>
<tr>
<td>Fischer et al (1998)</td>
<td>15</td>
<td>12–17 yr</td>
<td>Open trial GCBT + drug</td>
<td>CY-BOCS score:</td>
<td>32%</td>
</tr>
<tr>
<td></td>
<td>7 wk</td>
<td>F/U: 6 mo</td>
<td></td>
<td>32% posttreatment,</td>
<td></td>
</tr>
<tr>
<td>(2001)</td>
<td></td>
<td></td>
<td></td>
<td>50% at 6-mo F/U</td>
<td></td>
</tr>
<tr>
<td>Thienemann et al (2001)</td>
<td>18</td>
<td>13–17 yr</td>
<td>Open trial GCBT + drug or other treatment</td>
<td>9 (60%) responders</td>
<td>25%</td>
</tr>
<tr>
<td></td>
<td>14 wk</td>
<td>F/U: 6 mo</td>
<td></td>
<td>(&gt;50% CY-BOCS score)</td>
<td></td>
</tr>
<tr>
<td>Waters et al (2001)</td>
<td>7</td>
<td>10–14 yr</td>
<td>Open trial CBT</td>
<td>6 (88%) responders</td>
<td>60%</td>
</tr>
<tr>
<td></td>
<td>14 wk</td>
<td>F/U: 3 mo</td>
<td></td>
<td>(no longer met diagnostic criteria for OCD)</td>
<td></td>
</tr>
<tr>
<td>Benazon et al (2002)</td>
<td>16</td>
<td>8–17 yr</td>
<td>Open trial CBT</td>
<td>7 (44%) asymptomatic</td>
<td>50%</td>
</tr>
<tr>
<td></td>
<td>12 wk</td>
<td>F/U: 3 mo</td>
<td></td>
<td>10 (60%) responders</td>
<td></td>
</tr>
<tr>
<td>(2002)</td>
<td></td>
<td></td>
<td></td>
<td>(CY-BOCS score)</td>
<td></td>
</tr>
<tr>
<td>Piacentini et al (2002)</td>
<td>42</td>
<td>5–17 yr</td>
<td>Open trial CBT + drug</td>
<td>33 (79%) responders</td>
<td>45%</td>
</tr>
<tr>
<td></td>
<td>12.5 sessions</td>
<td>F/U: 3 mo</td>
<td></td>
<td>(CSI-S-c)</td>
<td></td>
</tr>
<tr>
<td>Barrett et al (2004)</td>
<td>77</td>
<td>7–17 yr</td>
<td>Randomized parallel study ICBT vs GCBT vs control</td>
<td>21/24 (88%) responders in ICBT condition, 22/29 (76%) responders in GCBT condition</td>
<td>65% for ICBT 60% for GCBT</td>
</tr>
<tr>
<td>(2005)</td>
<td>14 wk</td>
<td></td>
<td></td>
<td>0/24 (0%) responders in control condition</td>
<td></td>
</tr>
</tbody>
</table>

OC = obsessive compulsive; mo=months; F/U=follow-up; yr=years; CBT=cognitive-behavioral therapy; NR=not reported; wk=weeks; BT=behavioral therapy; CY-BOCS=Children's Yale-Brown Obsessive-Compulsive Scale; GCBT=group cognitive-behavioral therapy; OCD=obsessive-compulsive disorder; CGI-S=Clinical Global Impression-Severity; ICBT=individual cognitive-behavioral therapy; vs=versus.