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Scenario 1

Lizzo was a second year uni student when she first began having problems. She would go days without sleeping and had difficulty concentrating at uni. She started spending a lot of money on strange online shopping purchases (e.g. 18 miniature shrek figurines). When her parents discovered the problems, they brought her in for evaluation. Lizzo did not feel that anything was wrong. She felt that she had just made several poor decisions, like anyone her age. You are Lizzo’s GP and they have persuaded her to come in to see you.

1. **What will you look for on history and examination to support a diagnosis of a manic episode?**

DIGFAST

D – distractibility

I – irresponsibility or loss of social inhibitions (e.g. reckless behaviour, aggressiveness, hostility)

G – grandiosity or heightened self-esteem

F – flight of ideas or racing thoughts

A – increased goal directed activity (sexually, at work or socially)

S – decreased need for sleep

T – talkativeness/pressured speech

PLUS significant dysfunction (for mania) or not significant dysfunction (for hypomania)

1. **What are some complications of acute mania?**
* Psychosis
* Suicidality
* Marked functional impairment
* Damage to reputation and assets
1. **How is mania treated acutely and long term?**

Acute mania: treat with atypical antipsychotics (olanzapine, quetiapine) – lithium can be used when mania is mild and patient is not agitated

Long term: lithium

Lizzo is prescribed lithium as a mood stabilizer. The GP explains that lithium has a ‘narrow therapeutic index’ and this means she will have to have regular monitoring of the drug.

1. **What does this mean and what kind of monitoring will need to be done?**

The therapeutic index is the LD50/ED50 where the ED50 is the potency (dose required to produce 50% of the maximum possible response) and the LD50 is the dose that is lethal in 50% of the test population. The higher the lethal dose in comparison to the effective dose, the safer the drug. For lithium, the narrow therapeutic index means that the lethal dose is close to the effective dose.

Lithium levels and renal function are the key things to monitor. Lithium causes nephrotoxicity and lithium toxicity is also caused by impaired renal function if the kidneys are unable to clear the lithium.

Scenario 2

Geoff Schwartz is a 23 year old man who is brought to the GP by his sister who is concerned about his increasingly bizarre behaviour. She says he talks about voices no one else can here and the voices tell him to ‘make more tables about the menstrual cycle’. He also believes that ‘the renin-angiotensin-aldosterone system is watching him’. His sister is very interested in the possibility that he is having a psychotic episode.

1. **She asks you ‘what are the main categories of symptoms that define psychosis and some examples of each?’**

|  |  |  |
| --- | --- | --- |
| **Positive symptoms** | **Negative symptoms** | **Disorganized** |
| Hallucinations (auditory is most common type)IllusionsDelusions | Flat affectAlogiaAnhedoniaApathy  | Loose associationsWord saladNeologisms Tangential speech |

1. **Discuss the difference between an illusion vs delusion vs hallucination.**

Delusion: fixed, false beliefs which cannot be corrected by logic and are not consistent with the culture and education of the patient

Hallucination: false sensory perceptions experienced without real external stimulus e.g. seeing a goblin sitting next to you, when nothing is actually there

Illusions: Misperception of real external stimulus. E.g. looking at a cloud formation and seeing a goblin

Geoff is diagnosed with schizophrenia and is commenced on olanzapine. What class of drug does olanzapine belong to?

1. **List 3 side effects of olanzapine you would monitor for in Jeff.**
* Metabolic side effects very common with second generation antipsychotics: dyslipidaemia, weight gain, hyperglycaemia and diabetes mellitus 🡪 need to monitor waist circumference, fasting glucose, lipid profile and blood pressure
* Anticholinergic side effects: dry mouth, constipation, urinary retention
* Cardiovascular events: QT prolongation and cardiomyopathy
* Sexual side effects: reduced libido, erectile dysfunction, anorgasmia)
* Sedation: due to antihistamine action
* Hyperprolactinaemia
	+ Dopamine secretion from the hypothalamus inhibits prolactin secretion 🡪 anti-dopaminergic activity therefore removes the inhibition of prolactin secretion. More on this in P4P
* + many more - antipsychotics are a pharmacological nightmare

Two weeks after Geoff’s acute psychosis, he comes to see the doctor because of difficulty with movements and a tremor. Neurological examination shows a shuffling gait, increased tone in the upper extremities and a tremor of the hands which improves with activity. The mental status examination is normal.

1. **What is going on here and what is the mechanism?**

This is drug-induced pseudoparkinsonism (bradykinesia, muscle rigidity, resting tremor) caused by anti-dopaminergic activity of antipsychotic drugs. NB: typical antipsychotics are thought to be worse for extrapyramidal side effects but they can occur with all antipsychotics

1. **How are you going to manage these symptoms?**

The first step would be to stop or reduce the dose of the antipsychotic. If this doesn’t work, consider centrally-acting muscarinic antagonists, e.g. benztropine.

1. **A patient presents to ED with some stuttering in their speech, numbness in their right leg and intermittent hemiballistic movements of their left arm. Apart from the above signs, their neurological examination is normal. The work up includes bloods, lumbar puncture, CT and MRI head, spine, an EEG and nerve conduction studies. No organic cause can be found to explain the symptoms. Which of the following is the most likely diagnosis?**
2. Somatic symptom disorder
3. Hypochondriasis
4. Multiple sclerosis
5. Malingering
6. **Conversion disorder**

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NB: The newer term for conversion disorder is ‘functional neurological disorder’.

1. **These are the three most common causes of dementia, fill out the table:**

|  |  |  |  |
| --- | --- | --- | --- |
|  | **Alzheimer’s** | **Vascular dementia** | **Lewy body dementia** |
| Natural history | Steady chronic decline | Stepwise decline or can be more insidious if small vessels affected | Variable – steady decline or more abrupt |
| Major clinical features | Classical picture is impaired memory (short term first, then longer term), cognition and speech | Cognitive impairment + focal neurological deficits Risk factors are the same as those for CVA | Visual hallucinations and Parkinsonism (LB dementia is a Parkinson-plus syndrome) |
| Radiographic findings | Diffuse cortical atrophy | Evidence of prior ischaemia e.g. periventricular hypodensities  | No noteworthy findings |
| Pathology | Amyloid beta plaques and neurofibrillary tangles composed of tau protein | Necrotic brain tissue  | Lewy bodies composed of alpha-synuclein  |

NB: Parkinson’s disease and Lewy body dementia share a common pathology and exist on a spectrum – the patient’s presentation depends on where the Lewy bodies deposit first (i.e. in substantia nigra or the cortex/frontal lobe)

1. **What are the three key differentials for an elderly patient presenting with cognitive impairment?**
* Depression, dementia and delirium – know how to compare and contrast these
1. **What if the patient had urinary incontinence and ataxia, as well as the cognitive impairment?**
* Normal pressure hydrocephalus - most common reversible cause of dementia. ‘Wet, wacky and wobbly’
	+ Gait disorder
	+ Dementia
	+ Urinary incontinence
1. **What if the patient was a chronic drinker, had ophthalmoplegia, was confused and ataxic? How could this progress if this was untreated?**
* This patient has Wernicke encephalopathy (reversible condition caused by alcohol-related thiamine deficiency)
* If untreated this would lead to Korsakoff syndrome: the main features are confabulation (unconscious production of fabricated memories to fill in real ones they forgot) with amnesia (long term memory usually preserved)
* The former is reversible, the latter is not!

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