



mMEDCON International

Round 1 MPSC: NEUROLOGY (35 points)

Case Study I: (12 points)

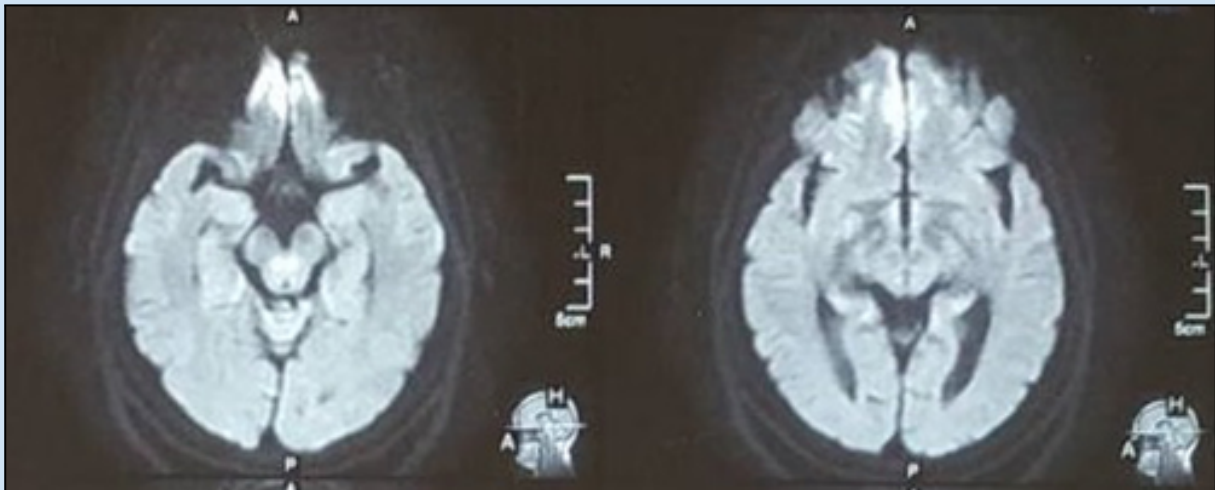
A 65-year-old woman was referred to the hospital with a 3-month history of behavioral, and personality changes along with progressive cognitive decline. She was in her usual state of health until 3 months ago when her family members noticed that she seemed to have easy forgetfulness and worsening functional impairment. The forgetfulness was initially related to daily care activities, dressing, and self-care and later progressed to the extent of not recognizing family members. Later, the patient developed stiffness in the entire body and started experiencing visual hallucinations, such as seeing random people in her surroundings. In the 2 weeks before the emergency room visit, her symptoms rapidly progressed, to the extent that she was unable to ambulate. There was also new onset urinary and fecal incontinence. She does not smoke or consume alcohol, and there is no significant past medical, surgical, or psychiatric history.

There is no family history of dementia or other neurological disorders. At the time of admission, she was mute and appeared ill. The tone was increased, and brisk reflexes were present across all extremities. Jaw jerk was prominent, and bilateral palmomental reflex was present. Continuous myoclonic jerks involving all extremities were noted. She was unable to ambulate secondary to worsening ataxia. The presence of forced eye gaze deviation was noticed at times. Paratonia was present in both upper limbs and lower limbs and bilateral extensor plantar response.

Routine laboratory investigations including serology for hepatitis B, HIV, and syphilis were all negative. Lumbar puncture for CSF evaluation along with culture revealed insignificant findings (total count: 2/cumm with only lymphocytes, sugar: 3.5 mmol/L and total protein 62 mg/dL, and ADA levels: 2.32 U/L). Thyroid hormones, vitamin B12, and folate levels were all within normal limits. Chest X-ray, electrocardiogram, and abdominopelvic ultrasound were otherwise unremarkable. EEG report showed continuous periodic complexes and generalized slow waves as shown in the picture below.



MRI of the Brain revealed high signal intensity in the bilateral frontal and occipital cortex as shown in the picture below. The involvement of the pulvinar nucleus of the bilateral thalamus with subtle T2 high signal giving a hockey stick appearance is also noted in the patient.





The autoimmune encephalitis panel (NMDA, AMPA-GluR1, AMPA-GluR2, GABA-B receptor antibody, LGI-1 antibody, and CASPR2 antibody) was negative.

- 1. What is the diagnosis and what symptoms and findings indicate this disease? List at least five.** (6pts → 1pt for the diagnosis, 5pts for the symptoms/findings)

- 2. What causes this disease? List and describe the types of this disease.** (4pts → 1pt for the cause, 3 pts for the types)

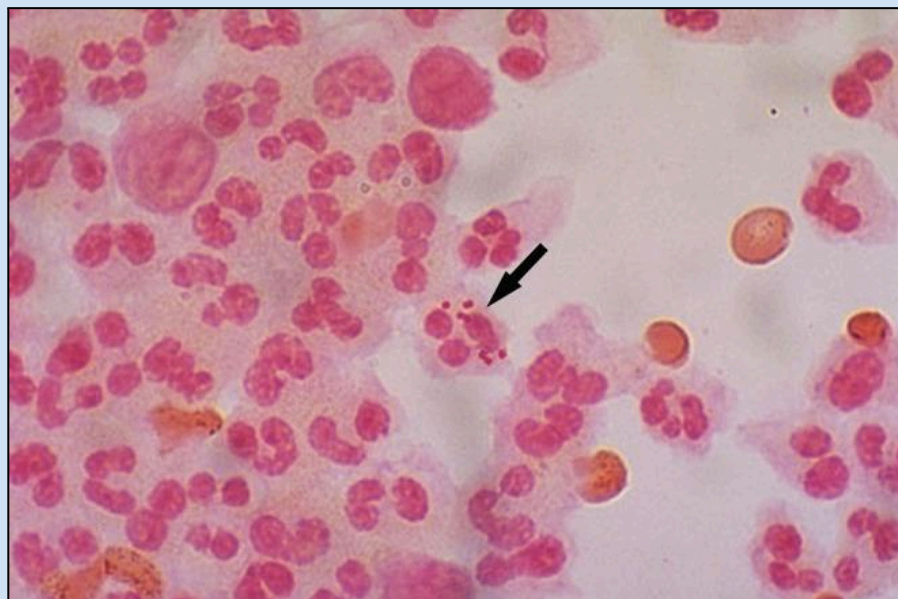
- 3. Is this disease treatable? What is the patient's prognosis?** (2 pts → 1pt for whether the disease is treatable, 1 pt for the prognosis)

Case Study II: (16 points)

An 18-year-old college student who lives in the campus dormitory presents to the emergency department with a fever and a stiff neck that began a few hours earlier. He also describes a headache and nausea that started at the same time. He has not seen a physician since the age of 13. His social history reveals recreational marijuana use and periodic alcohol ingestion. Physical examination reveals a well-nourished individual who is confused and in moderate distress. Vital signs are a temperature of 39.4 °C, heart rate of 120 bpm, blood pressure of 95/60 mm Hg, and respiratory rate of 20 breaths per minute. Cardiac examination reveals a normal S1 and S2 with regular rhythm without murmurs, heaves, or gallops. Lungs are clear to auscultation. The abdomen is soft, and non-tender with positive bowel sounds and no masses on palpation. A petechial rash is noted on his trunk. His skin is cold and clammy. Kernig and Brudzinski signs are positive. He is oriented to person and place but not time and has no focal neurological deficits. The remaining examination is within normal limits.

- 1. What are the Kernig and Brudzinski Signs, and what further testing is indicated for the patient?** (7pts - > 5 pts for testings, 2 pts for signs description)

An initial CBC is remarkable for a white blood cell (WBC) count of $18,000/\text{mm}^3$ ($4,500\text{-}11,000/\text{mm}^3$) and platelet count of $90,000/\text{mm}^3$ ($150,000\text{-}400,000/\text{mm}^3$). The PT is 17 seconds (11-15 seconds), INR is 1.4 (0.9-1.1), aPTT is 38 seconds (25-40 seconds), and the d-dimer is 1,100 ng/mL (<500 ng/mL). Following an unremarkable head CT scan, an LP is performed with an opening pressure of 250 mm H₂O (70-180 mm H₂O). The CSF has a yellowish, cloudy appearance. Results of the CSF analysis show a WBC count of $10,000$ cells/ mm^3 ($0\text{-}5$ cells/ mm^3), 99% neutrophils; protein 200 mg/dL (20-45 mg/dL); and glucose of 20 mg/dL (45-65mg/dL). The CSF gram stain below shows gram-negative diplococci. CSF culture, CSF-PCR panel, and blood cultures are pending.



Four hours later, a repeat CBC is remarkable for a WBC count of $22,000/\text{mm}^3$ and a platelet count of $40,000/\text{mm}^3$. Repeat coagulation studies are remarkable for a PT of 25 seconds, aPTT of 45 seconds, INR of 2.4, and a d-dimer of 3,500 ng/mL. The fibrinogen level is 0.1 g/L (0.2-0.4 g/L).



- 2. Given the patient's age and laboratory results, what is the diagnosis? What organism is the most likely culprit? Characterize the organism and list the most common causative agents in neonates and elderly patients. (5 pts -> 1pt for the diagnosis, 1pt for the organism, 1pt for its description, 2 pts for the causative agents)**

 - 3. How should this disease be treated? What are some possible complications of the acute form of this disease? (4pts -> 2pts for the treatment, 2 pts for the complications)**
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Case Study III: (7 points)

A woman in her 80s with a 5-year history of worsening motor symptoms presented to the Movement Disorders Clinic. Her initial symptoms included difficulty walking that progressively worsened alongside the presence of choreic movements, involving head bobbing and extremity tremors. There were no symptoms of numbness, urinary incontinence or memory impairment (mild forgetfulness was noted and attributed to age). No significant alcohol intake history was elicited.

Family history was significant for a sister with HD that developed in her 40s, with no relevant history in parents or grandparents. Her children were not known to have developed any signs of Huntington chorea and have not received genetic testing to date.

On general physical examination, there was no distress or abnormality identified. Her neurological assessment revealed mild abnormal movements in her mouth/face, and upper and lower extremities. Asymmetric bradykinesia was present on the left greater than right side as well as mild symmetrical rigidity. Her stance was normal with a slow gait and mild shuffling. Long tract signs were absent, along with a negative Romberg sign, no cerebellar dysmetria or truncal ataxia. Genetic testing revealed a 28-repeat CAG expansion in the huntingtin allele on chromosome 4.

- 1. What is the patient's diagnosis? Describe the pathophysiology of this disease. (2pts -> 1pt for the diagnosis, 1pt for the pathophysiology)**

2. **What is the clinical presentation of this disease, and how does it change based on its stages?** (3pts → 1pt for clinical presentation in each stage)

3. **What scale is widely recommended for the assessment of the severity of motor signs in this disease? Describe it briefly.** (2pts → 1pt for the name of the scale, 1pt for the description)