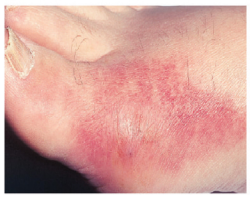
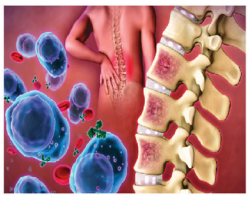
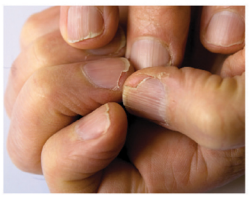
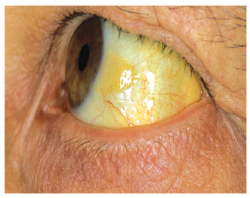

Clinical Cases in Biochemistry

As per the Revised Competency-based Medical Education Curriculum (NMC-2024)

2nd Edition

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JAYPEE

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Section

4 Vitamins

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Give Reasons Why

Case 4.1

Beri-Beri

COMPETENCY

- **BC8.1:** Describe the Biochemical role of vitamins in the body and explain the manifestations of their deficiency

Integration with Pediatrics

- **PE12.7:** Describe the causes, clinical features, diagnosis and management of deficiency of B complex Vitamins

Integration with Medicine

- **GM24.3:** Discuss and describe the etiology, causes, clinical manifestations, complications, diagnosis and management of common vitamin deficiencies

Case Scenario

A 60-year-old female presented at our emergency department with 1-week febrile sense, progressive dyspnea, and generalized edema. A few days prior to this presentation, she had experienced several loose stools with intermittent abdominal pain localized to the right upper quadrant for 4 months.

She denied smoking or using illicit drug use, but confessed excessive and protracted alcohol consumption and a dietary pattern restricted to intake of only carbohydrates; specifically, eating only cookies without any intake of essential nutrients.

Laboratory Findings Reveal

- Aspartate transaminase (AST)—34 IU/L (8–20)
- Alanine transaminase (ALT)—34 IU/L (10–40)
- Total bilirubin 0.8 mg/dL—(0.2–1.2)
- Electrolyte levels, thyroid function test and cardiac enzymes like creatinine kinase (CK), CK-MB, and troponin-I were all within the normal range

QUESTIONS

Q.1. What is the most probable diagnosis in this case?

Q.2. Enlist the factors which can precipitate this deficiency disorder.

Q.3. Which investigation you require to confirm your diagnosis?

Q.4. What will be your treatment plan?

ANSWERS

A.1. Patient is most probably suffering from beri-beri. It is a severe and chronic form of thiamine (vitamin B_1) deficiency, characteristic clinical signs and symptoms are shown in **Figure 4.1.1**.

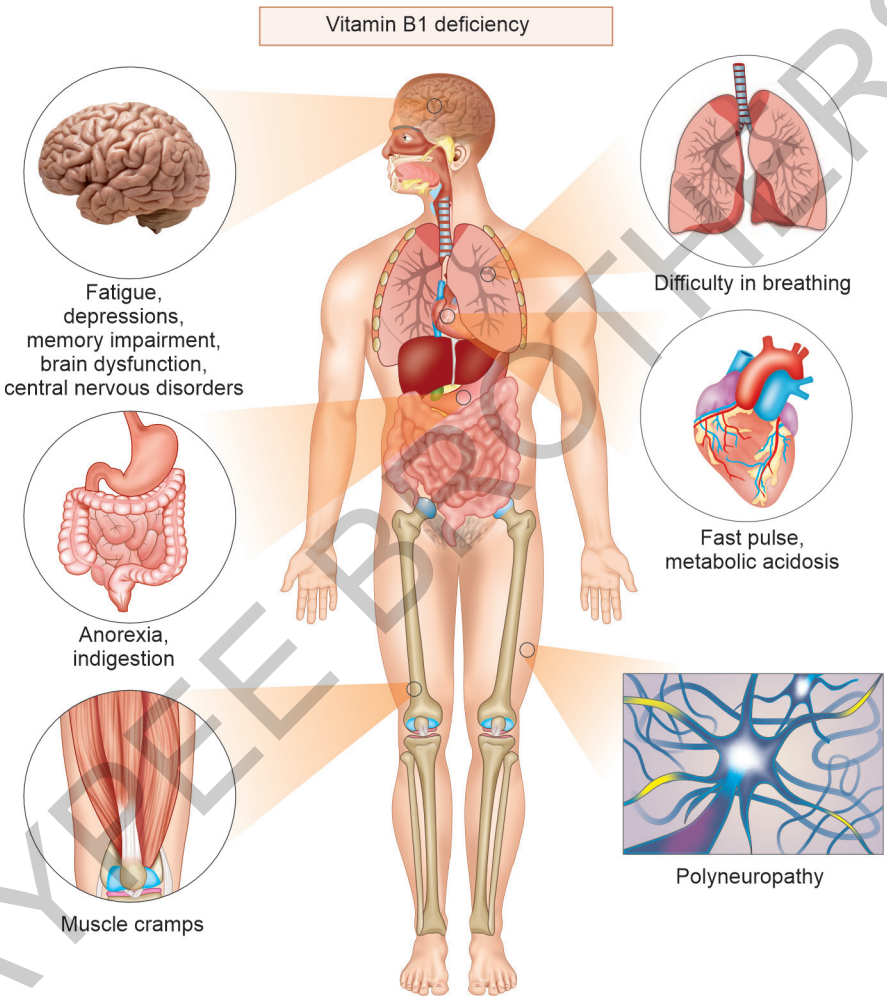

Fig. 4.1.1: Clinical presentation of vitamin B_1 deficiency.

The two main types in adults are:

Wet beri-beri: Wet beri-beri also known as shoshin beri-beri, cardiovascular beri-beri affects the cardiovascular system, clinical manifestation starts with vasodilation, tachycardia, a wide pulse pressure, sweating, warm skin, shortness of breath, and leg swelling and lactic acidosis. Later, heart failure develops.

Dry beri-beri: Dry beri-beri affects the nervous system, they affect predominantly the lower extremities, beginning with paresthesia's in the toes, burning in the feet (particularly severe at night), muscle cramps in the calves, pains in the legs. Continued deficiency worsens polyneuropathy and can result in muscle wasting.

Infantile beri-beri: Infantile beri-beri (thiamine deficiency) occurs mainly in infants breastfed by mothers with inadequate intake of thiamine, presents with sleeplessness, loss of appetite, vomiting, lactic acidosis, changes in heart rate, and enlargement of the heart.

A.2. Recommended daily allowance for thiamine is 1–1.5 mg/day and 0.5 mg/day additional per 1,000 kcal energy released from carbohydrate sources.

Precipitating Factors for Beri-Beri

Food habits: Diet consisting largely of highly refined carbohydrates as polished rice (oriental beri-beri), because thiamine is present in outer (aleurone) layers of cereals, diet-containing raw fish, raw shellfish, tea, coffee have thiaminase activity which destroy vitamin B_1.

Increased demand: Hyperthyroidism, pregnancy, lactation, strenuous exercise, or fever.

Decreased intake: Gastric or small intestinal resection, malabsorption syndromes hepatic insufficiency, diarrhea, hyperemesis gravidarum.

Interaction with other drugs: Alcohol, absorption is inhibited by thiamine analogues (such as metronidazole), diuretics (such as amiloride).

A.3. Laboratory investigations for confirmation of beri-beri are:

- Erythrocyte transketolase levels <0.75 (normal 0.75–1.30 IU/g Hb)
- Blood thiamine levels <2.5 (2.5–7.5 µg/dL)

A.4. Ensuring that dietary supplies of thiamine are adequate is important regardless of symptoms.

Start supplemental thiamine based on clinical manifestations.

- **For mild polyneuropathy:** 10 to 20 mg orally once a day for 2 weeks.
- **For moderate or advanced neuropathy:** 20 to 30 mg/day (as a single or divided dose), continued for several weeks after symptoms disappear.
- **For edema and congestion due to cardiovascular beri-beri:** 100 mg IV once-a-day for several days.

BIBLIOGRAPHY

1. Moonen M, Lancellotti P, Betz R, Lambermont B, Piérard L. Béribéri [Beri-beri]. Rev Med Liege. 2007;62(7-8):523-30.
2. Wiley KD, Gupta M. Vitamin B_1 thiamine deficiency. [Updated 2022 Jul 22]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2022. Available from: https://www.ncbi.nlm.nih.gov/books/NBK537204/

Case 4.2

Wernicke-Korsakoff Syndrome

COMPETENCY

- **BC8.1:** Describe the Biochemical role of vitamins in the body and explain the manifestations of their deficiency

Integration with Pediatrics

- **PE12.7:** Describe the causes, clinical features, diagnosis and management of deficiency of B complex Vitamins

Integration with Medicine

- **GM24.3:** Discuss and describe the etiology, causes, clinical manifestations, complications, diagnosis and management of common vitamin deficiencies

Case Scenario

A 60-year-old man presents with confusion, disorientation, and gait imbalance. Other than disorientation, bilateral paresis of gaze is noted. He has history of heavy alcohol intake for 15 years. He has progressive dementia. Physical examination shows weight loss and muscle wasting. There is no hepatomegaly.

Laboratory Findings Reveal

- Blood glucose: 58 mg/dL (70–140 mg/dL)
- AST: 48 IU/L (8–20 IU/L)
- ALT: 38 IU/L (10–40 IU/L)
- CT head is negative for any tumor/injury

QUESTIONS

Q.1. What is the probable diagnosis? Explain the biochemical basis the cause of this condition.

Q.2. What is common cause for such a clinical presentation, how you can confirm your diagnosis?

Q.3. What is the plan of management of this patient?

ANSWERS

A.1. Patient is suffering from Wernicke-Korsakoff syndrome, precipitated due to deficiency of vitamin B_1. Clinical presentation of Wernicke-Korsakoff syndrome is summarized in **Figure 4.2.1**.

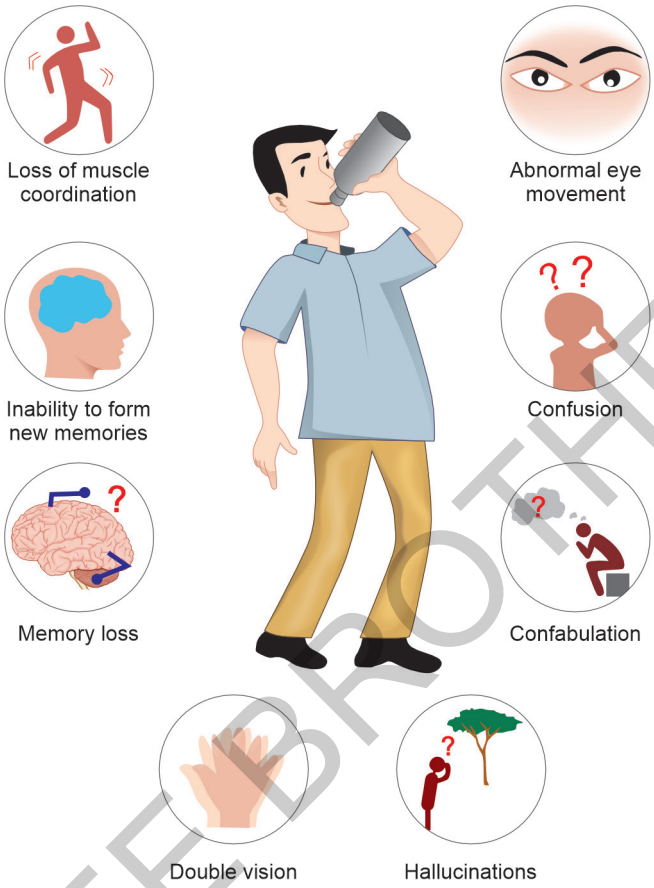

Fig. 4.2.1: Clinical presentation of Wernicke-Korsakoff syndrome.

Biochemical Basis of Clinical Signs and Symptoms

Mild thiamine deficiency presents with loss of appetite, fatigue, peripheral neuropathy, while is severe deficiency result in Wernicke encephalopathy—Korsakoff psychosis characterized by global confusion, ophthalmoplegia and ataxia (GOA).

Thiamin deficiency causes degeneration of peripheral nerves, thalamus, mammillary bodies, and cerebellum. Cerebral blood flow is markedly reduced, and vascular resistance is increased.

A.2. Chronic alcoholism with severe nutritional deficiency of thiamine is the most causes of such presentation, other common causes of thiamine deficiency.

Poor Intake

- Diets primarily high in polished rice/processed grains
- Chronic alcoholism
- Parenteral nutrition without adequate thiamine supplementation
- Gastric bypass surgery

Poor Absorption

- Malnutrition
- Gastric bypass surgery
- Malabsorption syndrome

Increased Loss

- Diarrhea
- Hyperemesis gravidarum
- Diuretic use
- Renal replacement therapy

Increased Thiamine Utilization

- Pregnancy
- Lactation
- Hyperthyroidism

Diagnosis of Wernicke-Korsakoff Syndrome

A full psychiatric assessment, and a comprehensive case history is essential. To rule out tumors, infarcts, and bleeding, computed tomography (CT) scanning and magnetic resonance imaging (MRI) may be needed.

Laboratory investigation for confirmation of diagnosis is blood thiamine level and erythrocyte transketolase activity.

A.3. Because most of the patients present with hypoglycemia, IV glucose can worsen thiamin deficiency, alcoholics and others at risk of thiamin deficiency should receive IV thiamin 100 mg before receiving IV glucose solutions.

For Wernicke-Korsakoff syndrome, thiamin 50 to 100 mg IM or IV twice a day must usually be given for several days, followed by 50 to 100 mg orally once a day until a therapeutic response is obtained. Anaphylactic reactions to IV thiamin are rare. Symptoms of ophthalmoplegia may resolve in a day; improvement in patients with Korsakoff psychosis may take 1 to 3 months. Recovery from neurologic deficit is often incomplete in Wernicke-Korsakoff syndrome.

BIBLIOGRAPHY

1. Akhouri S, Kuhn J, Newton EJ. Wernicke-Korsakoff syndrome. [Updated 2022]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2022. Available from: https://www.ncbi.nlm.nih.gov/books/NBK430729/
2. Covell T, Siddiqui W. Korsakoff syndrome. [Updated 2022 Jul 20]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2022 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK539854/

Case 4.3

Pellagra

COMPETENCY

- **BC8.1:** Describe the Biochemical role of vitamins in the body and explain the manifestations of their deficiency

Integration with Pediatrics

- **PE12.7:** Describe the causes, clinical features, diagnosis and management of deficiency of B complex Vitamins

Integration with Medicine

- **GM24.3:** Discuss and describe the etiology, causes, clinical manifestations, complications, diagnosis and management of common vitamin deficiencies

Case Scenario

A 69-year-old woman with alcohol use disorder was brought to the emergency department with progressive disorientation, and disorganized speech. Two weeks before emergency episode patient had a history of watery diarrhea. On admission the patient was dehydrated, pale, lethargic, disoriented, she displayed symmetrical scaly rash with hyperpigmentation and hyperkeratinization in exposed areas of the skin.

QUESTIONS

Q.1. What is the most probable diagnosis, explain the biochemical basis of clinical signs and symptoms?

Q.2. How you confirm your diagnosis?

Q.3. What are the causative agents/precipitating factors of such clinical presentation?

Q.4. What is the line of management?

ANSWERS

A.1. Patient is most probably suffering from pellagra (vitamin B_3 deficiency disorders). Pellagra is an Italian word meaning rough skin. Major symptoms of pellagra are:

- **Dermatitis:** Early stages of deficiency is characterized by red erythema around ankles and face which on persistent deficiency may cause dark pigmentation around neck known as Casal's necklace.

- Diarrhea
- Dementia
- Death

Active form of niacin NAD+ and NADP+ are required as co-enzymes for more than 200 enzymes. Neurological symptoms in pellagra may be due to a serotonin deficiency caused by decreased tryptophan availability to the brain.

A.2. Confirmation of diagnosis of pellagra can be done by:

- Typical clinical sign and symptoms **(Fig. 4.3.1)**
- Dietary/medication history
- Blood level of vitamin B3 (niacin) found low
- Low levels of urinary excretion of N-methylnicotinamide and pyridone indicates niacin deficiency.

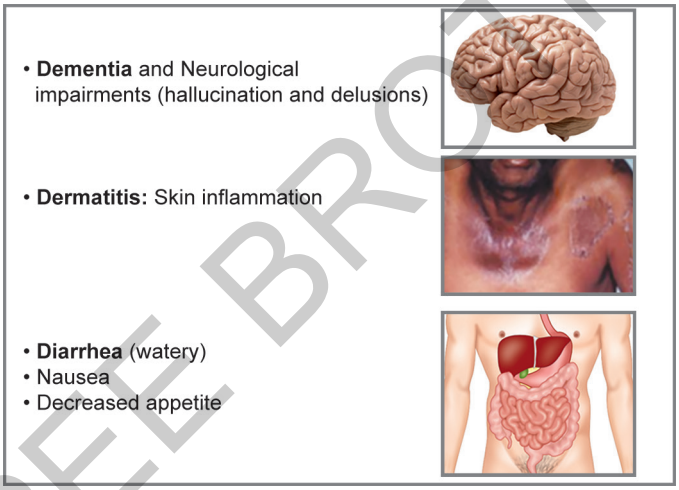

Fig. 4.3.1: Clinical presentation of pellagra (vitamin B_3) deficiency.

A.3. There are multiple causes of niacin deficiency:

- **Dietary deficiency of tryptophan:** Approximately 60 mg of tryptophan synthesizes 1 mg niacin.
- **Consumption of maize as staple diet:** In maize niacin is present in a bound form, which is unavailable for functioning.
- **Consumption of sorghum (jowar) as staple diet:** Sorghum is rich in leucine, which inhibits enzyme quinolinate phosphoribosyl transferase (QPRTase). This enzyme is required to convert niacin into active form NAD+.
- **Deficiency of pyridoxal phosphate (PLP) (vitamin B6):** This is the co-enzyme for kynureninase, one of the enzymes of tryptophan metabolism require for synthesis of niacin from tryptophan.
- **Antitubercular drug INH (isoniazid):** It inhibit formation of PLP.

- **Hartnup's disease:** This is a autosomal recessive disease characterized by defect in absorption of tryptophan from intestine.
- **Carcinoid syndrome:** Neuroendocrinal tumor of gastrointestinal tract, argentaffinoma—a tumor which secretes large amounts of the hormone serotonin from tryptophan. So, most of the tryptophan is used for serotonin synthesis leads to deficiency of niacin.

A.4. Management of Pellagra

Pellagra is treated by niacin. The WHO has recommended niacin to treat pellagra at 300 mg in divided doses for up to 4 weeks. Mucositis and diarrhea will improve within days, and neuropsychiatric changes improve in the first week. Skin changes may take a while to resolve totally.

For Prevention of Pellagra

Recommended dose for adults—10–15 mg/day.

(The WHO recommended intake is 6.6 niacin equivalents per 1,000 kcal for adults and children of 6 months or older).

BIBLIOGRAPHY

1. Pitche PT. Pellagre et érythèmes pellagroïdes [Pellagra]. Sante. 2005;15(3):205-8.
2. Prabhu D, Dawe RS, Mponda K. Pellagra a review exploring causes and mechanisms, including isoniazid-induced pellagra. Photodermatol Photoimmunol Photomed. 2021;37(2):99-104.

Case 4.4

Folic Acid Deficiency

COMPETENCY

- **BC8.1:** Describe the Biochemical role of vitamins in the body and explain the manifestations of their deficiency

Integration with Pediatrics

- **PE12.7:** Describe the causes, clinical features, diagnosis and management of deficiency of B complex Vitamins

Integration with Medicine

- **GM24.3:** Discuss and describe the etiology, causes, clinical manifestations, complications, diagnosis and management of common vitamin deficiencies

Case Scenario

A 26-year-old woman (gravida 4, para 3) was admitted at 31 weeks gestation with worsening fatigue and shortness of breath on exertion over a duration of 1 month. She reported about anorexia, pregnancy-related nausea, but denied any fever, night sweats or itching. There was no history of alcohol excess or any dietary restriction. All her previous pregnancies had been uneventful. On examination, she was pale and exhausted with no palpable lymphadenopathy or splenomegaly.

Laboratory Findings Reveal

Macrocytosis with a severe pancytopenia:

- Hemoglobin—7.2 g/dL (11.5–16.5 g/dL)
- MCV (mean cellular volume)—104 fL (80–100 fL)
- Platelets—15 × 10^9/L (150–400 × 10^9/L)
- Neutrophils—0.5 × 10^9/L (1.7–7.5 × 10^9/L)
- Vitamin B_{12} level—110 pg/mL (191–663 pg/mL)
- Serum folate—2.7 ng/mL (4.6–18.7 ng/mL)

QUESTIONS

Q.1. What is the probable diagnosis of patient? What may be the possible causes for this clinical presentation?

Q.2. Explain the biochemical basis of clinical signs and symptoms.

Q.3. How will you confirm the diagnosis?

Q.4. What will be the plan of management for this patient?

ANSWERS

A.1. Biochemical basis of clinical presentation in folic acid deficiency **(Fig. 4.4.1)**.

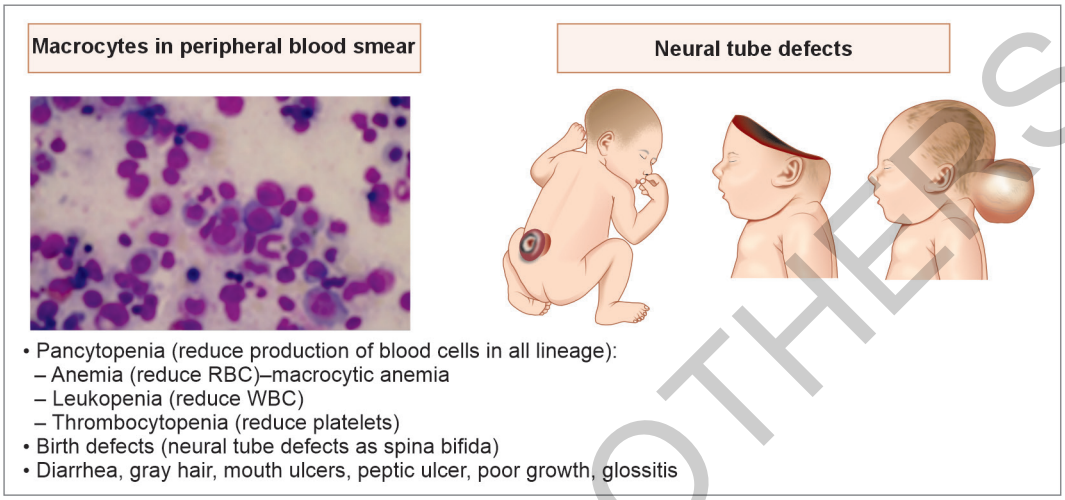

Fig. 4.4.1: Clinical presentation of folic acid deficiency.

Folic acid is essential in one carbon metabolism. As active form of folic acid, tetrahydrofolic acid acts as carrier of one carbon moieties for different metabolic reaction **(Table 4.1.1)**.

Table 4.1.1: One carbon moieties and their corresponding folic acid carrier.

Group	*Structure*	*Carried by*
Formyl	—CHO	N^5–formyl—THFA and N^{10}–formyl—THFA
Formimino	—CH=NH	N^5–formimino—THFA
Methenyl	=CH–	N^5, N^{10}–methenyl—THFA
Hydroxymethyl	—CH_2OH	N^{10}–hydroxymethyl—THFA
Methylene	—CH_2–	N^5, N^{10}–methylene—THFA
Methyl	—CH_3	N^5–methyl—THFA and methylcobalamin

One carbon metabolic reactions are essential in:

- Purine ring synthesis (C2 and C8 of purine ring)
- Pyrimidine synthesis (conversion of deoxyuridine monophosphate to deoxythymidine phosphate dUMP to dTMP)
- Transmethylation reaction
- Synthesis of choline, creatine, epinephrine

Deficiency of folic acid result in derange one carbon metabolism leads to:

- **Reduced DNA synthesis:** It affects rapidly diving cells like bone marrow (causes pancytopenia) intestinal mucosa (GIT upset) hair (hair loss). In pregnancy reduced DNA synthesis result in birth defects especially neural tube defects.

- **Hyperhomocysteinemia:** Accumulation of homocysteine result in increased risk of cardiovascular accidents.

A.2. Most probable diagnosis of this patient is folic acid deficiency.

Possible causes of such nutritional deficiency:

- **Increased demand:** Pregnancy, lactation
- **Decreased uptake:** Dietary deficiency, malabsorption, intestinal disorders (coeliac, sprue, gluten enteropathy) or intestinal resection
- **Drugs:** Anticonvulsant (phenytoin, phenobarbitone), alcohol, oral contraceptives
- **Folate trap:** Deficiency of vitamin B_{12} results in deficiency of free tetrahydrofolic acid **(Fig. 4.4.2)**.

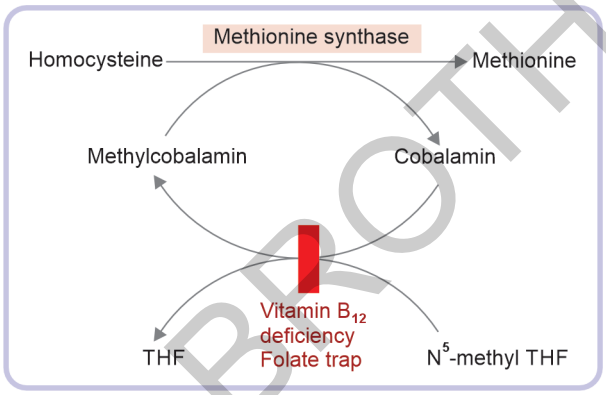

Fig. 4.4.2: Folate trap.

A.3. Confirmation of folic acid deficiency can be done by estimation of serum blood folic acid levels and histidine load test/FIGLU excretion test. During the metabolism of histidine FIGLU (formiminoglutamic acid) is synthesized as an intermediate which gives its formimino group to folic acid, but in case of folic acid deficiency, this process does not occur resulting in excretion of FIGLU in urine.

A.4. Therapeutic dose in 1 mg/day orally and always should be in combination with vitamin B_{12}.

Folic acid should never give alone in macrocytic anemia, as it will definitely decrease the symptoms of anemia but aggravate neurological symptoms of vitamin B_{12} deficiency.

BIBLIOGRAPHY

1. Donnelly JG. Folic acid. Crit Rev Clin Lab Sci. 2001;38(3):183-223.
2. Prabhu D, Dawe RS, Mponda K. Pellagra a review exploring causes and mechanisms, including isoniazid-induced pellagra. Photodermatol Photoimmunol Photomed. 2021;37(2):99-104.

Case 4.5

Vitamin B_{12} Deficiency

COMPETENCY

- **BC8.1:** Describe the Biochemical role of vitamins in the body and explain the manifestations of their deficiency

Integration with Medicine

- **GM24.3:** Discuss and describe the aetiology, causes, clinical manifestations, complications, diagnosis and management of common vitamin deficiencies

Integration with Pediatrics

- **PE12.7:** Describe the causes, clinical features, diagnosis and management of deficiency of B complex Vitamins

Case Scenario

A 59-year-old man with 6 months history of progressive loss of memory, disorientation, apathy, paranoid delusions, gait difficulties with falls, and urinary incontinence. He had suffered similar episode 3 years before, with a complete remission. Paleness and icterus in the sclera had been noted from last few weeks, the vegetarian patient had not consumed any food of animal origin for many years. Besides, his family only rarely ate fresh fruits or vegetables.

Laboratory Findings Reveal

Complete blood count shows pancytopenia, peripheral blood smear shows megaloblasts (large RBC) with hypersegmented neutrophils.

- Hemoglobin: 8 g/dL (12–16 g/dL)
- Vitamin B_{12}: 38 pg/mL (191–663 pg/mL)

QUESTIONS

Q.1. What is the probable diagnosis of patient? What may be the possible differential diagnosis for this clinical presentation?

Q.2. Explain the biochemical basis of clinical signs and symptoms.

Q.3. How will you confirm the diagnosis?

Q.4. What will be the plan of management for this patient?

ANSWERS

A.1. Patients is most probably suffering from vitamin B_{12} deficiency vitamin B_{12} (cobalamin) is a water-soluble vitamin that is derived from animal products such

as red meat, dairy, and eggs. Intrinsic factor is a glycoprotein that is produced by parietal cells in the stomach and necessary for the absorption of B_{12} in the terminal ileum. Once absorbed, B_{12} is used as a cofactor for enzymes that are involved in the synthesis of DNA, myelin sheath and metabolism of odd chain fatty acids. As a result, B_{12} deficiency can lead to hematologic and neurologic symptoms. B_{12} is stored in excess in the liver. Clinical presentaion of vitamin B_{12} deficiency is summarized in **Figure 4.5.1**.

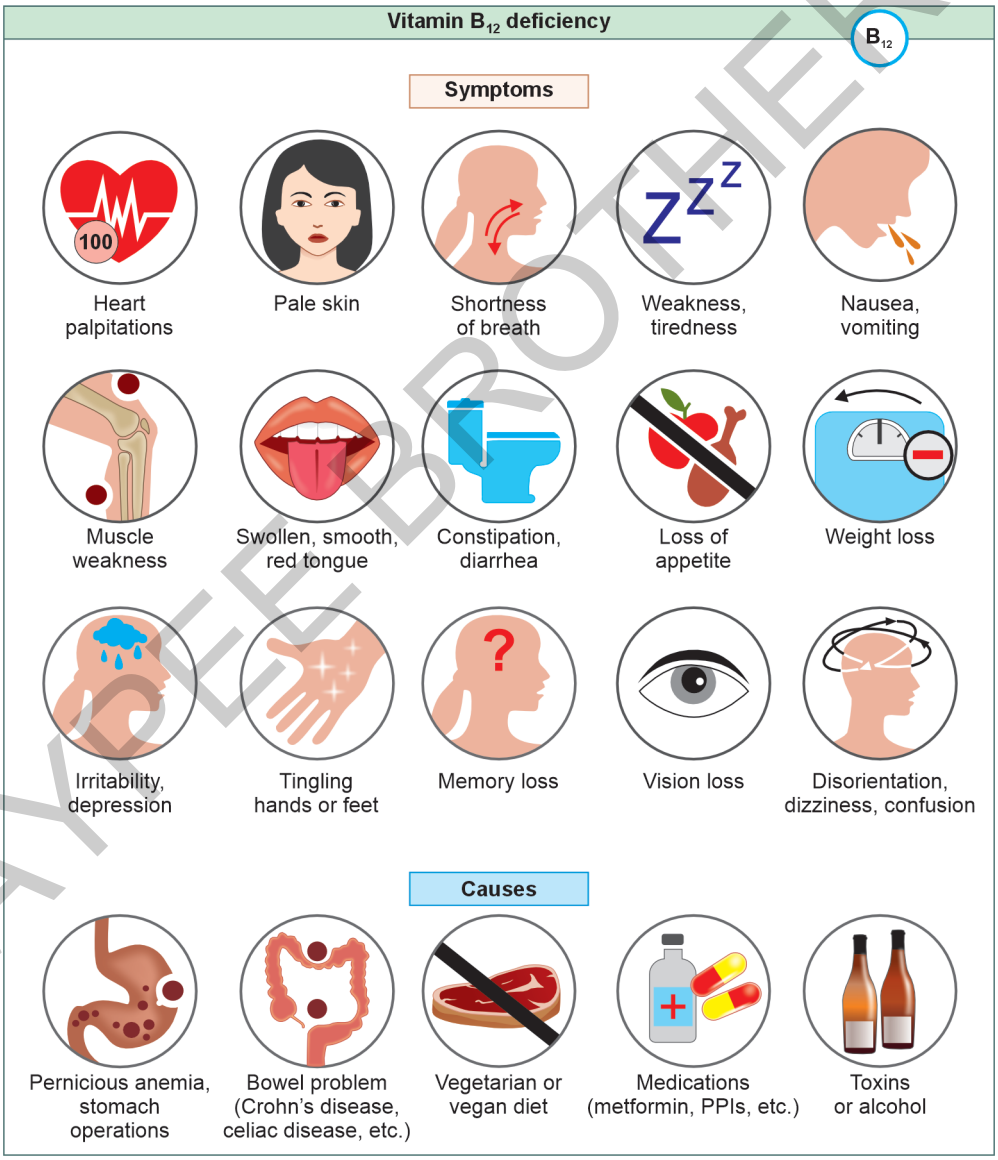

Fig. 4.5.1: Clinical presentation of vitamin B_{12} deficiency.

Vitamin B_{12} deficiency is primarily of three types:

1. **Autoimmune:** Autoimmune conditions like pernicious anemia where antibodies against intrinsic factor are produced. It will result in an inability of B_{12} to be absorbed by the terminal ileum.
2. **Malabsorption:** Patient with history of gastrointestinal surgery, gastric bypass surgery may be at risk for developing a B_{12} deficiency because parietal cells in the stomach produce intrinsic factor. In patients with normal intrinsic factor production, any damage to the terminal ileum, such as surgical resection due to Crohn's disease, will impair the absorption of B_{12} and lead to a deficiency. Other conditions like celiac disease or infection with the tapeworm *Diphyllobothrium latum*, may also result in a B_{12} deficiency.
3. **Dietary insufficiency:** Although vitamin B_{12} can be stored in the liver, but patients who have followed a strict vegan diet for long time may develop a B_{12} deficiency from a lack of dietary intake.

Differential diagnosis of vitamin B_{12} deficiency:

- Lead toxicity
- Syphilis
- HIV myelopathy
- Multiple sclerosis

A.2. Biochemical basis of clinical presentation is as follows:

- Decreased activity of methylmalonyl CoA mutase (isomerase) (vitamin B_{12} act as co-enzyme) leads to accumulation of methylmalonyl acid, as it cannot convert into succinyl-CoA resulting in metabolic acidosis, CNS depression.
- Decreased activity of methyltransferase (vitamin B_{12} act as co-enzyme) leads to:
  - **Demyelination:** Methylation of phosphatidylethanolamine and phosphatidyl-choline is inadequate resulting in defective myelin sheath of nerves and neurological lesions.
  - **Homocystinuria:** Homocysteine cannot be converted into methionine leads to accumulation of homocysteine which predispose to increase cardiovascular events.
- **Folate trap:** Vitamin B_{12} deficiency results in trapping of folate in methyl THFA form resulting in deficiency disorders of folate co-exist.

A.3. Assessment of vitamin B_{12} deficiency can be assess by examination of peripheral blood smear and/or bone marrow morphology which shows megaloblastic anemia.

Other investigations are:

- Direct estimation of vitamin B_{12} levels in serum normal levels >145 pg/mL, depend on age and gender of person.
- Indirect methods are urinary levels of methylmalonyl acid and homocysteine levels, FIGLU excretion test.

A.4. Patients present with clinical presentation of macrocytic anemia should be treated with folic acid, vitamin B_{12} combination. Therapeutic dose of B_{12} is 500 to 1,000 micrograms by intramuscular injections.

BIBLIOGRAPHY

1. Ankar A, Kumar A. Vitamin B_{12} deficiency. [Updated 2022 Aug 12]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2022. Available from: https://www.ncbi.nlm.nih.gov/books/NBK441923/
2. Langan RC, Goodbred AJ. Vitamin B_{12} Deficiency: Recognition and Management. Am Fam Physician. 2017;96(6):384-9.

Clinical Cases in Biochemistry

Salient Features

- Thoroughly revised as per the latest CBME curriculum by NMC (2024).
- Clinical cases are an essential component of both internal and summative assessment for MBBS exams as per new CBME curriculum led by NMC.
- Clinical cases are illustrated in a manner that it will also be helpful in NExT Pre-PG, Foreign Medical Graduate entrance, and medical licensing examinations.
- Simple and comprehensible language for easy understanding.

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