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MD (Pathology & Microbiology)
Chairman & Consultant Pathologist

Dr. Yugam Chopra
MD (Pathology)
CEO & Consultant Pathologist

NAME	**: Baby. PIHU**		
AGE/ GENDER	: 8 YRS/FEMALE	**PATIENT ID**	: 1547830
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REFERRED BY	:	**REGISTRATION DATE**	: 13/Jul/2024 01:59 PM
BARCODE NO.	: 01513078	**COLLECTION DATE**	: 13/Jul/2024 02:08PM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	**REPORTING DATE**	: 13/Jul/2024 02:50PM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA CANTT		

Test Name	Value	Unit	Biological Reference interval

CLINICAL CHEMISTRY/BIOCHEMISTRY

ALKALINE PHOSPHATASE (ALP)

ALKALINE PHOSPHATASE: SERUM *by PARA NITROPHENYL PHOSPHATASE BY AMINO METHYL PROPANOL*	355	U/L	0.0 - 500.0

DR.VINAY CHOPRA
CONSULTANT PATHOLOGIST
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Test Name	Value	Unit	Biological Reference interval
CREATININE			
CREATININE: SERUM *by ENZYMATIC, SPECTROPHOTOMETRY*	0.52	mg/dL	0.40 - 1.20

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Test Name	Value	Unit	Biological Reference interval

CALCIUM

CALCIUM: SERUM *by ARSENAZO III, SPECTROPHOTOMETRY*	9.76	mg/dL	8.50 - 10.60

INTERPRETATION:-

1.Serum calcium (total) estimation is used for the diagnosis and monitoring of a wide range of disorders including diseases of bone, kidney, parathyroid gland, or gastrointestinal tract.
2. Calcium levels may also reflect abnormal vitamin D or protein levels.
3.The calcium content of an adult is somewhat over 1 kg (about 2% of the body weight).Of this, 99% is present as calcium hydroxyapatite in bones and <1% is present in the extra-osseous intracellular space or extracellular space (ECS).
4. In serum, calcium is bound to a considerable extent to proteins (approximately 40%), 10% is in the form of inorganic complexes, and 50% is present as free or ionized calcium.
NOTE:-Calcium ions affect the contractility of the heart and the skeletal musculature, and are essential for the function of the nervous system. In addition, calcium ions play an important role in blood clotting and bone mineralization.

HYPOCALCEMIA (LOW CALCIUM LEVELS) CAUSES :-

1.Due to the absence or impaired function of the parathyroid glands or impaired vitamin-D synthesis.
2. Chronic renal failure is also frequently associated with hypocalcemia due to decreased vitamin-D synthesis as well as hyperphosphatemia and skeletal resistance to the action of parathyroid hormone (PTH).
3.***NOTE:-*** A characteristic symptom of hypocalcemia is latent or manifest tetany and osteomalacia.

HYPERCALCEMIA (INCREASE CALCIUM LEVELS) CAUSES:-

1.Increased mobilization of calcium from the skeletal system or increased intestinal absorption.
2.Primary hyperparathyroidism (pHPT)
3.Bone metastasis of carcinoma of the breast, prostate, thyroid gland, or lung.
NOTE:-Severe hypercalcemia may result in cardiac arrhythmia.

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Test Name	Value	Unit	Biological Reference interval

PHOSPHOROUS

PHOSPHOROUS: SERUM	4.55	mg/dL	2.30 - 4.70

by PHOSPHOMOLYBDATE, SPECTROPHOTOMETRY

INTERPREATION:-

1.Eighty-eight percent of the phosphorus contained in the body is localized in bone in the form of hydroxyapatite. The remainder is involved in intermediary carbohydrate metabolism and in physiologically important substances such as phospholipids, nucleic acids, and adenosine triphosphate (ATP).
2.Phosphorus occurs in blood in the form of inorganic phosphate and organically bound phosphoric acid. The small amount of extracellular organic phosphorus is found exclusively in the form of phospholipids.
3.Serum phosphate concentrations are dependent on meals and variation in the secretion of hormones such as parathyroid hormone (PTH) and may vary widely.

DECREASED (HYPOPHOSPHATEMIA):-

1.Shift of phosphate from extracellular to intracellular.
2.Renal phosphate wasting.
3.Loss from the gastrointestinal tract.
4.Loss from intracellular stores.

INCREASED (HYPERPHOPHATEMIA):-

1.Inability of the kidneys to excrete phosphate.
2.Increased intake or a shift of phosphate from the tissues into the extracellular fluid.

SIGNIFICANCE:-

1.Phosphate levels may be used in the diagnosis and management of a variety of disorders including bone, parathyroid and renal disease.
2.Hypophosphatemia is relatively common in hospitalized patients. Levels less than 1.5 mg/dL may result in muscle weakness, hemolysis of red cells, coma, and bone deformity and impaired bone growth.
3.The most acute problem associated with rapid elevations of serum phosphate levels is hypocalcemia with tetany, seizures, and hypotension. Soft tissue calcification is also an important long-term effect of high phosphorus levels.
4.Phosphorus levels less than 1.0 mg/dL are potentially life-threatening and are considered a critical value.

NOTE: Phosphorus has a very strong biphasic circadian rhythm. Values are lowest in the morning, peak first in the late afternoon and peak again in the late evening. The second peak is quite elevated and results may be outside the reference range

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Test Name	Value	Unit	Biological Reference interval

MAGNESIUM

MAGNESIUM: SERUM *by XYLIDYL BLUE, SPECTROPHOTMETRY*	1.9	mg/dL	1.70 - 2.100

INTERPRETATION:-

1.Magnesium along with potassium is a major intracellular cation.
2.Magnesium is a cofactor of many enzyme systems. All adenosine triphosphate (ATP)-dependent enzymatic reactions require magnesium as a cofactor. 3.Approximately 70% of magnesium ions are stored in bone. The remainder is involved in intermediary metabolic processes; about 70% is present in free form while the other 30% is bound to proteins (especially albumin), citrates, phosphate, and other complex formers. The serum magnesium level is kept constant within very narrow limits. Regulation takes place mainly via the kidneys, primarily via the ascending loop of Henle.

INCREASD (HYPERMAGNESIA):-Conditions that interfere with glomerular filtration result in retention of magnesium and hence elevation of serum concentrations.
1.Acute and chronic renal failure.
2.magnesium overload.
3.Magnesium release from the intracellular space.
4.Mild-to-moderate hypermagnesemia may prolong atrioventricular conduction time. Magnesium toxicity may result in central nervous system (CNS) depression, cardiac arrest, and respiratory arrest.

DECREASED (HYPOMAGNESIA):-
1.Chronic alcoholism.
2.Childhood malnutrition.
3.Malabsorption.
4.Acute pancreatitis.
5.Hypothyroidism.
6.Chronic glomerulonephritis.
7.Aldosteronism.
8.Prolonged intravenous feeding.

NOTE:-
Numerous studies have shown a correlation between magnesium deficiency and changes in calcium-, potassium-, and phosphate-homeostasis which are associated with cardiac disorders such as ventricular arrhythmias that cannot be treated by conventional therapy, increased sensitivity to digoxin, coronary artery spasms, and sudden death. Additional concurrent symptoms include neuromuscular and neuropsychiatric disorders.

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MBBS , MD (PATHOLOGY)

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Test Name	Value	Unit	Biological Reference interval

CREATININE PHOSPHOKINASE-MB (CPK-MB)

CPK-MB - SERUM	2.94	ng/mL	0.0 - 5.0

by EFIA (ENZYM FLUORESCENT IMMUNOASSAY)

Interpretation:-

1.Alternative name of Creatine Kinase (CK) is Creatine phospho-kinase(CPK).
2.Creatine Kinase (CK) is a dimeric enzyme composed of two types of monomer sib-units (i.e. M-Muscular & B-Brain),which combine to form three distict CK isoenzymes.
a).CK-BB(CK-I), is produced primarily by brain , lungs and smooth muscles , and enters the blood only on injury to these organs like cerebrovascular accidents or pulmonary infarctions.
b).CK-MB (CK-II), is produced primarily by heart muscle;
c).CK-MM (CK-III) , is produced primarily by skeletal muscle.
3).Normally very little CK is found circulating in the blood. Elevated levels indicate damage to either muscle or brain possibly from a myocardial infarction, muscle disease, or stroke.
4).CK levels are reduced in first half of pregnancy, and increased in second half of pregnancy.

Increased:-
Physiological:-
1.Strenuous physical activity .
2.New Born.
Pathological :-
1.Myocardial & pulmonary infarction
2**.** Accident and recent surgery.
3. Drugs:- Statins.
4.Convulsions & brain tumour.
5. Myopathies
6.Malignant hyperthermia
7.Hypothyroidism & Hyperthyroidism

5).CK-MB (CK-II) levels increase significantly 4-6 hours following a myocardial infaction and peak at around 12-24 hours after the infarct. The levels return to normal , in case of no further myocarial damage, after 24 to 48 hours . Hence the increased levels of CK-MB along with elevated levels of total CK is a good indicator of myocardial infarction.
6).For diagnosis of MI with high sensitivity and specificity , serial sampling over a period of 8 to 12 hours is required . For accurate diagnosis of myocardial infarction, CK-MB activity along with total CK should be measured. If the total CK activity is raised and CK-MB contributes mare than 6% of the total activity, then myocardial infarction is considered highly probable.

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Test Name	Value	Unit	Biological Reference interval
ENDOCRINOLOGY			
FREE THYROXINE (FT4)			
FREE THYROXINE (FT4): SERUM *by ECLIA (ELECTROCHEMILUMINESCENCE IMMUNOASSAY)*	1.05	ng/dL	0.70 - 1.50

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Test Name	Value	Unit	Biological Reference interval

THYROID STIMULATING HORMONE (TSH)

THYROID STIMULATING HORMONE (TSH): SERUM	0.619	µIU/mL	0.60 - 5.50

by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)
3rd GENERATION, ULTRASENSITIVE

INTERPRETATION:

AGE	REFFERENCE RANGE (µIU/mL)
0 – 5 DAYS	0.70 – 15.20
6 Days – 2 Months	0.70 – 11.00
3 – 11 Months	0.70 – 8.40
1 – 5 Years	0.70 – 7.00
6 – 10 Years	0.60 – 5.50
11 - 15	0.50 – 5.50
> 20 Years (Adults)	0.27 – 5.50
PREGNANCY	
1st Trimester	0.10 - 3.00
2nd Trimester	0.20 - 3.00
3rd Trimester	0.30 - 4.10

NOTE:-*TSH levels are subjected to circardian variation, reaching peak levels between 2-4 a.m and at a minimum between 6-10 pm. The variation is of the order of 50 %. Hence time of the day has influence on the measured serum TSH concentration.*

USE:- TSH controls biosynthesis and release of thyroid harmones T4 & T3. It is a sensitive measure of thyroid function, especially useful in early or subclinical hypothyroidism, before the patient develops any clinical findings or goitre or any other thyroid function abnormality.

INCREASED LEVELS:

1.Primary or untreated hypothyroidism, may vary from 3 times to more than 100 times normal depending on degree of hypofunction.
2.Hypothyroid patients receiving insufficient thyroid replacement therapy.
3.Hashimotos thyroiditis.
4.DRUGS: Amphetamines, Iodine containing agents and dopamine antagonist.
5.Neonatal period, increase in 1st 2-3 days of life due to post-natal surge.

DECREASED LEVELS:

1.Toxic multi-nodular goitre & Thyroiditis.
2.Over replacement of thyroid harmone in treatment of hypothyroidism.
3.Autonomously functioning Thyroid adenoma
4.Secondary pituatary or hypothalmic hypothyroidism
5.Acute psychiatric illness
6.Severe dehydration.
7.DRUGS: Glucocorticoids, Dopamine, Levodopa, T4 replacement therapy, Anti-thyroid drugs for thyrotoxicosis.
8.Pregnancy: 1st and 2nd Trimester

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Test Name	Value	Unit	Biological Reference interval

LIMITATIONS:

1.TSH may be normal in central hypothyroidism, recent rapid correction of hyperthyroidism or hypothyroidism, pregnancy, phenytoin therapy.
2.Autoimmune disorders may produce spurious results.

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VITAMINS

VITAMIN D/25 HYDROXY VITAMIN D3

VITAMIN D (25-HYDROXY VITAMIN D3): SERUM *by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)*	57.3	ng/mL	DEFICIENCY: < 20.0 INSUFFICIENCY: 20.0 - 30.0 SUFFICIENCY: 30.0 - 100.0 TOXICITY: > 100.0

INTERPRETATION:

DEFICIENT:	< 20	ng/mL
INSUFFICIENT:	21 - 29	ng/mL
PREFFERED RANGE:	30 - 100	ng/mL
INTOXICATION:	> 100	ng/mL

1.Vitamin D compounds are derived from dietary ergocalciferol (from plants, Vitamin D2), or cholecalciferol (from animals, Vitamin D3), or by conversion of 7- dihydrocholecalciferol to Vitamin D3 in the skin upon Ultraviolet exposure.
2.25-OH--Vitamin D represents the main body resevoir and transport form of Vitamin D and transport form of Vitamin D, being stored in adipose tissue and tightly bound by a transport protein while in circulation.
3.Vitamin D plays a primary role in the maintenance of calcium homeostatis. It promotes calcium absorption, renal calcium absorption and phosphate reabsorption, skeletal calcium deposition, calcium mobilization, mainly regulated by parathyroid harmone (PTH).
4.Severe deficiency may lead to failure to mineralize newly formed osteoid in bone, resulting in rickets in children and osteomalacia in adults.

DECREASED:
1.Lack of sunshine exposure.
2.Inadequate intake, malabsorption (celiac disease)
3.Depressed Hepatic Vitamin D 25- hydroxylase activity
4.Secondary to advanced Liver disease
5.Osteoporosis and Secondary Hyperparathroidism (Mild to Moderate deficiency)
6.Enzyme Inducing drugs: anti-epileptic drugs like phenytoin, phenobarbital and carbamazepine, that increases Vitamin D metabolism.

INCREASED:
1. Hypervitaminosis D is Rare, and is seen only after prolonged exposure to extremely high doses of Vitamin D. When it occurs, it can result in severe hypercalcemia and hyperphophatemia.

CAUTION: Replacement therapy in deficient individuals must be monitored by periodic assessment of Vitamin D levels in order to prevent hypervitaminosis D

NOTE:-*Dark coloured individuals as compare to whites, is at higher risk of developing Vitamin D deficiency due to excess of melanin pigment which interefere with Vitamin D absorption.*

*** End Of Report ***

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MBBS , MD (PATHOLOGY)