Review Article

# Stepped Clinico-Investigative Diagnostic Algorithm [SCIDA] approach to Hypoglycemia in Adults – A Review

Dr. Gunanithi.K[1], Dr. Sridevi.S[2], Dr. Ashwini.D[3]

[1]Professor & HOD, Dept of Biochemistry, Tagore Medical College and Hospital, Affiliated to The Tamil Nadu Dr.MGR Medical University, Chennai, Tamilnadu
[2]Primary care Physician & Diabetologist, National Institute of Technical Teachers Training and Research Institute, Chennai, Tamil Nadu
[3]Assistant professor, Dept of Physiology, Govt Villupuram Medical College, Affiliated to The Tamil Nadu Dr.MGR Medical University, Chennai, Tamil Nadu

**Abstract**

Introduction: Hypoglycemia, a common condition encountered in medicine practice manifests as metabolic consequences due to low plasma glucose levels. It can have varied presentations ranging from subtle asymptomatic episodes to gross neurological deficits manifesting dramatically. SCIDA [Stepped Clinico-Investigative Diagnostic Algorithm] offers a systematic protocol-based attempt towards stepped algorithmic approach to the etiological diagnosis of Hypoglycemia at wholistic ease. Discussion: The sequential 7 steps in order includes 7 actions to be taken by the physician to arrive the diagnosis. It includes Suspect, Screen, Confirm, Classify, Categorize, Evaluate & Diagnose towards etiological cause of Hypoglycemia. Key criteria for hypoglycemia confirmation includes Whipple's triad. Among the types, Diabetic hypoglycemia is more common in clinical practice and mostly due to administration of exogenous insulin or oral hypoglycemic agent. On the other hand, Nondiabetic hypoglycemia is relatively rare but tends to occur due to varied causes and at times even multifactorial. It includes Insulinoma, MEN-1(Multiple Endocrine Neoplasia) associated islet cell tumors and many more. Conclusion: Physicians should maintain a high degree of suspicion towards its diagnosis as early treatment would result in prompt reversal of symptoms and its associated morbidity and mortality. This stepped approach provides a guide in history taking, examination findings and selection of relevant investigations to aid towards etiological diagnosis. SCIDA would further help to validate targeted treatment selection options beyond dextrose administrations, thereby preventing recurrences and thus improving quality of life among patients with hypoglycemia.

**Keywords:** Glucose homeostasis disorder, Insulin secreting tumor, Whipple's triad.

## 1. Introduction

Hypoglycemia, a common condition encountered in medicine practice manifested as metabolic consequences due to low plasma glucose levels. It can have varied presentations ranging from subtle asymptomatic episodes to gross neurological deficits manifesting dramatically. On a global scale, Incidence data of moderate hypoglycemia showed 13100 per 100000 patient per year and severe hypoglycemia showed 4800 per 100000 patient per year. [1-2] Incidence data of hypoglycemia among Indian population shows 16.51 per 1000 visits in the emergency departments. [3] Annual incidence of hypoglycemia in general ranges from 3.3 % – 13.5 %. [4] Hypoglycemia has been found to be associated with increased mortality rates [5] and morbidity rates [6-7]. Though hypoglycemia related to diabetes mellitus is common, mortality rates have even been reported to be higher among non-diabetic hypoglycemia too. [8] On the morbidity scale, Hypoglycemia causes increased risk of neurological abnormalities [9] like cognitive dysfunction, hemiparesis, pontine dysfunction and increased risk of cardiovascular complications [10] including arrhythmias and heart blocks along with increased risk of falls & fractures and can even lead to mental health abnormalities including anxiety, depression and FoH (fear of Hypoglycemia) on being recurrent.[11]

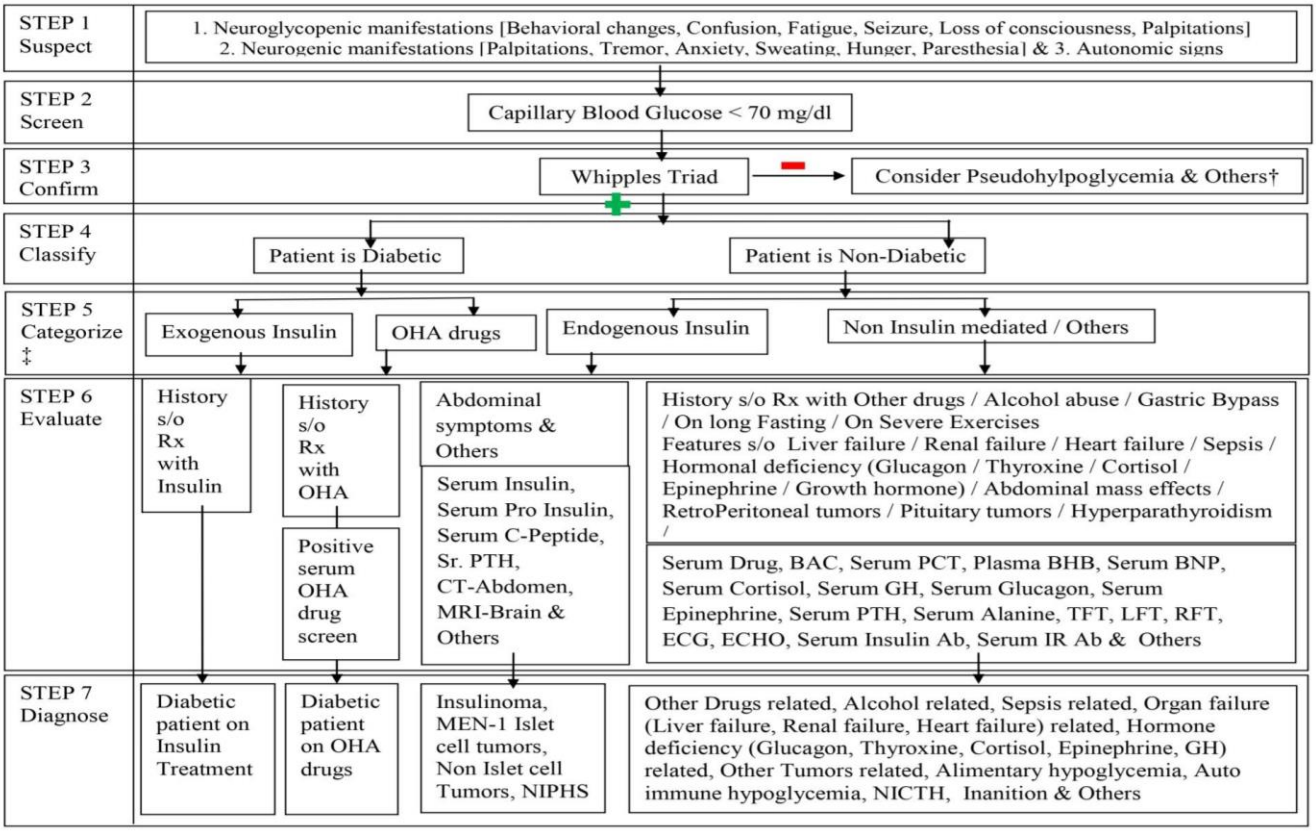
Among the types, Diabetic hypoglycemia is more common in clinical practice. Hypoglycemia is more frequent among Type 1 diabetes mellitus when compared to Type 2 diabetes mellitus. [12] The Most common cause of Diabetic Hypoglycemia is drug related including exogenous insulin administration [13] constituting 90% of those who receive it and treatment with insulin secretagogues like sulfonylurea and meglitinides.[14] One of the main reasons behind increased risk of hypoglycemia includes treatment guidelines moving towards stricter glycemic control as shown by ADVANCE (Action in Diabetes and Vascular Disease : Preterax and Diamicron Modified Release Controlled Evaluation)[15], VADT (Veteran Affairs Diabetes Trial) [16], ACCORD (Action to Control Cardiovascular Risk in Diabetes) [17] trials. On the other hand, Non-diabetic hypoglycemia is uncommon and even a rare presentation with estimated frequency of 36 per 10000 among in patient admissions and more likely among elderly population > 65 years age. [18].

Among the causes on non-diabetic hypoglycemia, other drug offenders, critical illness, alcohol use, organ failures, malnutrition are common. Tumors are a rare cause. [19] Among them, Insulinoma, a pancreatic islet beta cell neuroendocrine tumor remains a rare but important cause of non-diabetic hypoglycemia. Its incidence from 1-32 cases per million persons per year. [20] Insulinoma also is associated with 6-7% of Patients with MEN1 syndrome. [21] Much rare causes include Non insulinoma pancreatogenesis hypoglycemia syndrome (NIPHS), autoimmune hypoglycemia, [22] Least common considerations include pseudo hypoglycemia, artefactual hypoglycemia and malicious hypoglycemia. There are several algorithms in medical literature towards hypoglycemia diagnosis, yet SCIDA [Stepped Clinico-Investigative Diagnostic Algorithm] offers a systematic protocol-based attempt towards stepped algorithmic approach to the etiological diagnosis of Hypoglycemia at wholistic ease.

## 2. Discussion

The stepped algorithm as shown in Table-1 includes a systematic protocol that combines history considerations, clinical features and investigations into 7 steps. The sequential 7 steps in order includes 7 actions to be taken by the physician to arrive the diagnosis. It includes Suspect, Screen, Confirm, Classify, Categorize, Evaluate & Diagnose towards etiological cause of Hypoglycemia.

**Table 1. Stepped Clinico-Investigative Diagnostic Algorithm (SCIDA) Approach to Hypoglycaemia in Adults**




† - Includes True pseudo hypoglycaemia, Pseudo hypoglycaemia, Functional hypoglycaemia, Artifact/Spurious hypoglycaemia.

‡ - Categorization is based on quartet measurement including serum insulin, serum c-peptide, serum pro-insulin, serum OHA drug screen. OHA –Oral Hypoglycaemic Drugs, s/o – Suggestive Of, Rx – Treatment, PTH –Parathyroid hormone, CT – Computed Tomography, MRI – Magnetic Resonance Imaging, BAC – Blood Alcohol Concentration, PCT – Procalcitonin, BHB –Beta Hydroxy Butyrate, BNP – B-type Natriuretic Peptide, GH – Growth Hormone, TFT – Thyroid Function Test, LFT – Liver Function Test, RFT – Renal Function Test, ECG – Electrocardiogram, ECHO – Echocardiography, Ab – Antibody, IR Ab – Insulin Receptor Antibody, MEN –Multiple Endocrine Neoplasia, NIPHS – Non Insulinoma Pancreatogenesis Hypoglycaemia Syndrome, GH – Growth Hormone, NICTH - Non Islet Cell Tumour Hypoglycaemia

**STEPS**

**STEP 1 – Suspect Hypoglycemia**

First step includes maintaining a high degree of suspicion to possibility of Hypoglycemia whenever a patient presents mostly to emergency department with autonomic, neurogenic, neuroglycopenic features. It includes sweating, palpitations, shaking, hunger, confusion, drowsiness, odd behavior, speech difficulty, incoordination, headache, nausea (Edinburgh hypoglycemia scale) [23] Other neurogenic features including anxiety, paresthesia. tingling, and neuroglycopenic features including irritability, lethargy, changes in vision, speech abnormalities, seizures, cognitive abnormalities, confusion, feeling faint, feeling drowsy, loss of consciousness, headache, stroke like episodes like hemiplegia and even coma also should raise suspicion towards hypoglycemia. In addition, the autonomic signs including tachycardia, hypertension, pallor, diaphoresis, mydriasis and neuroglycopenic signs including transient & mostly reversible focal neurological deficits should be under consideration.

**STEP 2 – Screen Hypoglycemia**

Second step includes screening for evidence towards suspicion of Hypoglycemia by performing capillary blood glucose as point of care testing. The Normal reference range for blood sugar level (BSL) is 70-100 mg/dl. Capillary Blood Glucose (CBG) being considered as first line screening POCT(Point Of Care Testing) and hypoglycemia being considered if BSL falls below 70 mg/diet is the alert value defined by many global organizations including American Diabetes Association (ADA), American Association of Clinical Endocrinologists (AACE), Joint British Diabetes Society & Diabetes UK [24]. Continuous Glucose Monitoring [CGM] remains a better recommendation to screen and monitor subsequently but studies are lacking. The cut-off can be variable and to be kept flexible to even higher levels especially when a patient is a chronic uncontrolled diabetic patient.

**STEP 3 – Confirm Hypoglycemia**

Third step includes confirmation of Hypoglycemia, as most of its manifestations are nonspecific in nature. The diagnosis is usually confirmed by Whipple's triad. [25] including 3 criteria, first being symptoms consistent with hypoglycemia including neurologic manifestations, second being low plasma glucose levels and the third being relief of symptoms with subsequent glucose administration. Confirmation involves measurement of plasma glucose levels. Additionally, hypoglycemia can be ascertained into 3 levels as suggested by international hypoglycemia study group [26] based on the blood glucose levels, their implications and ability to treat or assistance requirement. Level 1/Hypoglycemia alert value [≤ 70 – 55mg/dl], Level 2/clinically significant hypoglycemia [≤ 54 - 41 mg/dl] and Level 3/severe hypoglycemia [≤ 40 mg/dl] indicates low plasma glucose levels with severe cognitive impairment. [27] Levels 1 and 2 are self-treatable, while Level 3 is not [28] and needs external assistance.

At this point, it is essential also to rule out laboratory artifacts like pseudo hypoglycemia, [29] functional hypoglycemia and spurious hypoglycemia at this point itself. True pseudo hypoglycemia seen in unrefrigerated samples of blood with large number of granulocytes, occur in leukemoid reactions, leukemia, administration of colony stimulating factors. Pseudo hypoglycemia is falsely low finger prick capillary glucose levels in patients with poor peripheral circulation, falsely low post prandial glucose levels in venous samples, typical symptoms present but with measured plasma glucose > 70 mg/dl. Functional hypoglycemia is seen during discontinuation of total parenteral nutrition. Spurious hypoglycemia is pre analytical artifact that delayed separation of plasma from blood cells, incomplete inhibition of glycolysis in tubes, excess glucose consumption by leukocytosis, erythrocytosis, thrombocytosis.

**STEP 4 – Classify Hypoglycemia**

Fourth step includes classification of Hypoglycemia into either Diabetic Hypoglycemia or Non-Diabetic Hypoglycemia. Diabetic hypoglycemia is more common while Nondiabetic hypoglycemia is relatively uncommon, especially during treatment initiations and titrations towards stricter glycemic controls among diabetic patients. Classification is important as most diabetic hypoglycemia are drug related and simple cessation of the offending drug and further down titration would suffice as optimal treatment. On the other hand, Nondiabetic hypoglycemia is relatively rare but tends to occur due to varied causes and at times even be multifactorial. It needs additional categorization and sometimes extensive evaluation through subsequent steps of the algorithm, so as to guide towards appropriate treatment protocols further.

**STEP 5 – Categorize Hypoglycemia**

Fifth step includes categorization based on the causative factor behind Hypoglycemia. It can be due to Insulin mediated (Exogenous Insulin, Oral hypoglycemic drugs, Endogenous Insulin) and Non-Insulin mediated / Others. Categorization is necessary to guide further treatment. Most times, history is always evident when Exogenous Insulin and Oral hypoglycemic agents remain the cause. In such cases, it's usually not necessary to proceed further down the algorithm. Simple cessation and future down titration of either exogenous insulin injection or oral hypoglycemic drug would do magical recovery. In others and in doubtful cases, categorization can be done on the basis of quartet measurement of serum insulin, serum C-peptide, serum proinsulin levels, serum oral hypoglycemic agent screen upon 72 hours fast which is considered the gold standard [30] for hypoglycemia evaluation. Alternative is collection of samples with 48 hours fast. [31] The quarter assessment could provide clues to the causative factor behind it. Normal reference range for serum insulin, serum C-peptide, serum proinsulin in fasting state is 2-25 µIU/mL, 0.9-1.8 ng/mL , 1.1-6.9 pmol/L respectively. Elevation of serum insulin levels alone (>3µU/ml) signifies Exogenous Insulin mediated hypoglycemia as the cause. Combined Elevation of serum insulin (>3µU/ml), serum C-peptide (≥0.2 nmol/L), serum Pro insulin (≥5 pmol/L) signifies Endogenous Insulin mediated hypoglycemia as the cause. A positive oral hypoglycemic drug screen along with combined elevations of serum insulin (>3µU/ml), serum C-peptide (≥0.2 nmol/L), serum Pro insulin (≥5 pmol/L) signifies oral hypoglycemic agent as the cause. [32] When all these tests prove negative, non-insulin mediated hypoglycemia / Other factors mediated hypoglycemia should be considered.

**STEP 6 – Evaluate Hypoglycemia**

Sixth step includes further evaluation for Non-Diabetic Hypoglycemia from clinical history, examination findings and appropriate laboratory and imaging studies.

Endogenous insulin being the reason behind hypoglycemia, the causes to be considered includes Insulinoma, MEN-1 related Islet cell tumors, NIPHS (Non insulinoma pancreatogenesis hypoglycemia syndrome) and also non-islet cell tumors with ectopic insulin production. Non-Islet cell tumors include bronchial carcinoma, ovarian carcinoma, teratoma, small cell carcinoma cervix. In such cases, history of abdominal symptoms including abdominal pain and abdominal mass effects, bone pain, muscular symptoms and organ specific symptoms need to be enquired. Further evaluation involves interpretation of obtained results of serum Insulin levels, serum Pro Insulin levels, serum C-peptide levels during categorization and performing additional investigations including serum parathyroid hormone (PTH) levels, and abdominal imaging studies (CT-abdomen) and neuro imaging studies (MRI-Brain), EUS (Endoscopic Ultra Sonography), selective arterial calcium stimulation test (SACST) and organ specific imaging and cancer specific investigations depending on the presenting scenarios. Ancillary investigations that document hyperinsulinism includes plasms BHB, plasma BCAA, plasma FFA levels. Genetic studies for MEN remain optional done only if Insulinoma is diagnosed, as it is commonest pancreatic endocrine manifestation in MEN-1.

Non-Insulin / Other factors being the reason behind hypoglycemia, drugs are among the most common cause as is alcohol. Drug offenders include fluoroquinolones, pentamidine, quinine, ACE inhibitors, ethanol, salicylates, and beta blockers. Other causes include ethanol abuse, sepsis, organ failures, hormone deficiencies, alimentary hypoglycemia, autoimmune hypoglycemia, NICTH and others. History needs to be enquired includes usage of other glucose lowering drugs / medications, alcohol abuse, excessive exercise, prolonged fasting, past history of medical ailments including liver disease, renal disease, heart disease, pituitary disease, parathyroid disease, associated auto immune diseases and any hormonal deficiency (Glucagon / Thyroxine / Cortisol / Epinephrine / Growth hormone) states. Examination findings to be watched for includes general examination, vital signs assessment and disease specific findings and any signs of sepsis. Additional investigations to be done on-need basis to support the history and examinations findings includes serum drug levels screen, Blood Alcohol Concentration (BAC), serum Procalcitonin (PCT), plasma beta hydroxyl butyrate (BHB) / urine ketones, serum lactate, serum B-type natriuretic peptide (BNP), serum cortisol levels, serum Growth Hormone (GH) levels, serum glucagon, serum epinephrine, serum alanine, serum Insulin antibody (Ab), serum Insulin Receptor (IR) Ab, Thyroid Function Tests (TFT), Liver Function Test (LFT), Renal Function Tests (RFT), Electrocardiogram (ECG), Echocardiogram (ECHO), serum electrolytes Arterial blood gas analysis (ABG), serum IGF-I, serum IGF-II, serum IGF-II:IGF-I ratio. Though Inborn errors of metabolism presenting as hypoglycemia seems to be obvious in childhood itself, the IEM panel of tests that needs to be considered includes urine reducing substances, urine dicarboxylic acid, serum free fatty acids, serum creatine kinase, plasma amino acids, serum lipid profile, serum uric acid & serum carnitine levels as last resort when search towards etiological evaluation ends in vain among adults.

**STEP 7 – Diagnose Hypoglycemia**

Seventh and Final step involves arriving at the final etiological diagnosis of Hypoglycemia from the interpretation of the previous 6 steps of the SCIDA algorithm. Diabetic hypoglycemia is more common among patients with type 1 diabetes mellitus and late-stage type 2 diabetes mellitus where the patient seems to be put on strict glycemic control. Diabetic Hypoglycemia due to Exogenous Insulin and Oral hypoglycemic drugs are most obvious from history taking through the narrative of family members and by evaluation of medical records of the patient. Mostly presents as recent treatment initiation or treatment titration with insulin and oral hypoglycemic agents and always accompanied by too few food intake, delayed meals, skipped meals, altered food pattern to liquids and semi solids & too much exercise.

Among non-diabetic hypoglycemia, differentiation between endogenous insulin mediated hypoglycemia and non-insulin mediated hypoglycemia remains an enigma for which the quarter assessment would provide clues to the causative factor. C peptide and insulin are secreted in equimolar amounts, and so C peptide serves as a good marker of endogenous insulin production. [33] The diseases related to endogenous insulin mediated hypoglycemia includes Insulinoma, MEN-1 related Islet cell tumors, NIPHS (Non insulinoma pancreatogenesis hypoglycemia syndrome) and also non-islet cell tumors with ectopic insulin production. Insulinoma presents persistent hyper insulinemia and recurrent fasting hypoglycemia. Diagnostic criteria for Insulinoma includes plasma glucose ≤55 mg/dl, serum insulin ≥ 3 U/mL, serum C peptide ≥ 0.6 ng/mL, serum pro insulin ≥ 5 pmol/L, serum beta hydroxybutyrate ≤2.7 mmol/L, on a 72-hour fasting sample.[34] Appearance on CT-abdomen includes a small, well circumscribed, solid mass that enhance with contrast. [35]. Other laboratory features of hyperinsulinism include low plasma BHB, low Plasma Branched chain amino acid levels and low Plasma FFA levels as hyperinsulinism suppress ketosis and helps to differentiate functional hyperinsulinism due to IGF-II and IR-Ab. Normal reference range for plasma BHB, plasma FFA are < 0.3 mmol/L, 0.1-0.9 mmol/L respectively. BCAA measurements include plasma valine, leucine and isoleucine and their reference range includes 146-370 µmol/L, 74-196 µmol/L, 35-104 µmol/L. Genetic studies would be positive for Insulinoma in MEN-1, as Insulinomas found associated with para thyroid tumors and pituitary tumors with female preponderance at fourth to sixth decade age.

NIPHS also called diffuse adult-onset nesidioblastosis is a very rare type of post prandial hyper insulinemic hypoglycemia due to generalized islet hyperplasia and islet hypertrophy. [36]. NIPHS usually have positive selective arterial calcium stimulation test for multiple pancreatic vascular territories on SACST. It additionally helps also in differentiation of insulinoma and NIPHS. Non-Islet cell tumors are usually diagnosed with more organ specific findings and relevant tumor markers as applicable.

Among non-diabetic hypoglycemia, non-insulin mediated causes are varied and can be multiple. Ethanol abuse remains a significant cause. BAC levels typically get elevated to 100-300 mg/dl in acute intoxication. [37] Ethanol causes hypoglycemia by inhibition of gluconeogenesis as it depletes NAD because of its metabolism to acetaldehyde and then to acetic acid by alcohol dehydrogenase and aldehyde dehydrogenase. Change in redox state [increase in NADH-NAD ratio] causes block in gluconeogenic pathway, decreased conversion of TCA cycle metabolites to oxaloacetate, decreased conversion of lactate to pyruvate, decreased conversion of glycerophosphate to DHA. Ethanol also inhibits cortisol and growth hormone responses.

Among critical illness, sepsis remains the most common cause. Though sepsis still remains a clinical diagnosis, serum PCT levels seem to be elevated and > 0.5 ng/ml to aid diagnosis. [38] Sepsis causes increased glucose utilization by muscle, macrophage rich tissue like liver, spleen. Cytokines such as TNF & IL-6 increase glucose utilization, reduce hepatic responsiveness, hepatic hypoperfusion, and inactivation of catecholamines by superoxide anions. Sepsis also leads to cytokine induced inhibition of gluconeogenesis with nutritional glycogen depletion, hepatic hypoperfusion, renal hypoperfusion.

Organ failures as a cause of hypoglycemia are less common but still renal failure, liver failure and cardiac failure are to be considered when there are renal parameters, liver parameters and cardiac biomarkers respectively. Hypoglycemia in hepatic disease is due to decreased glycogen stores with reduced glycogenolysis and reduced hepatic gluconeogenesis. Hypoglycemia in renal failure is due to cachexia, calorie deprivation with protein restriction, reduced glucose turnover, diminished gluconeogenesis from alanine, reduced alanine turnover, reduced glycemic responses to glucagon due to impaired glycogenolysis, impaired renal gluconeogenesis. In renal failure, reduced clearance of insulin

and reduced mobilization of gluconeogenic precursors. Hypoglycemia in uremia is due to failure to produce substrates from peripheral tissues, notably alanine for hepatic gluconeogenesis. Hypoglycemia in cardiac failure due to hepatic congestion, hypoxia, inanition, gluconeogenic precursor limitation. Raised blood lactate levels inhibit gluconeogenesis.

Alimentary hypoglycemia, a significant cause for reactive hypoglycemia is due to rapid dumping of carbohydrates in upper small intestine causing rapid transit of large osmotic load through the gut causing rapid fluid shift and marked autonomic response and excess insulin release by intestinal factors Glucagon like peptide (GLP-1), Glucose dependent insulinotropic peptide (GIP) and rapid rise in blood glucose. History would be obviously evident with post-bariatric or post-major gastric surgery [gastrectomy, vagotomy, pyloroplasty, gastrojejunostomy, laparoscopic Nissen fundoplication, billroth II procedure, Roux-en-Y gastric bypass.

Auto immune hypoglycemia / Auto immune insulin syndrome / Insulin autoimmune syndrome is a rare auto immune disorder with circulating insulin antibodies attributed to dissociation of insulin-insulin antibody immune complexes releasing free insulin. It also occurs when antibodies directed against insulin receptors with insulin mimetic action. Laboratory investigations reveal presence of Insulin auto antibodies (IAA) titer, high serum insulin levels, presence of macro insulin while serum pro insulin and C-peptide levels remain low with reversed Insulin: C-peptide ratio > 1. The normal Insulin: C-peptide ratio is < 1. [39]. Uncommonly, Type B Insulin resistance due to insulin receptor antibodies (IR-A) rarely also can present with hypoglycemia. Laboratory investigations reveal presence of serum IR-A. It is accompanied by hyperadiponectinemia, hypotriglyceridemia, acanthosis nigricans and other associated autoimmune diseases.

Hormones including Glucagon, Cortisol, Epinephrine, Thyroxine and Growth hormones play role in blood glucose regulation. Hormonal deficiencies as such might not be the primary reason behind hypoglycemia but can be an additional factor or even a rare cause that needs consideration. Excluding diabetes associated autonomic neuropathy, epinephrine deficiency and glucagon deficiency is extremely rare. Cortisol concentration < 18-20 mcg/dL at time of hypoglycemia is suggestive of ACTH deficiency or primary adrenal insufficiency. Hypoglycemia in cortisol deficiency is due to impaired gluconeogenesis and glycogen depletion. GH concentration < 7 ng/mL during a hypoglycemic episode is suggestive of GH deficiency.

Non-Islet Cell Tumor Hypoglycemia [NICTH], a paraneoplastic syndrome secondary to non-islet cell tumors seen in retroperitoneal fibrosarcoma, hepatocellular carcinoma, adrenocortical carcinoma, renal cell carcinoma, gastrointestinal tumors, lymphoma, leukemia. [40] Pro IGF-II secreted by these tumors results in hypoglycemia. Extra pancreatic neoplasms secrete large forms of insulin like growth factor II [big IGF-II]. Tumor releases incompletely processed Insulin like growth factor IGF-II [big IGF-II]. Laboratory findings include undetectable Serum Insulin levels and will be low < 5 µU/mL, and C-peptide levels but high circulating levels of big IGF-II.IGF-I levels < 100 ng/mL, IGF-II levels > 275 ng/mL and IGF-II : IGF-I ratio > 3:1 is suggestive of NICTH.[41]

Certain milder variants of inborn errors of carbohydrate metabolism including glycogenolytic and gluconeogenetic enzymes, FAOD, Aminoacidopathy, Myopathy can present in early adolescence and adulthood, that needs special consideration when the etiology remains clueless and the laboratory reports of IEM panel shows positive. Other less common considerations of hypoglycemia include starvation, inanition, after strenuous exercise and after marathon. Hypoglycemia here is due to multiple factors like high rates of glucose utilization, depletion of hepatic glycogen stores, loss of whole-body fat stores, limitation of amino acid substrates and thereby depletion of gluconeogenic precursors.

## 3. Conclusion

Hypoglycemia is a common condition encountered in medical practice presenting with autonomic, neurogenic and neuroglycopenic features. The manifestation spectrum varies from being subtly asymptomatic to gross and dramatic neurological deficits. Physicians should maintain a high degree of suspicion towards its diagnosis as early treatment would result in prompt reversal of symptoms and its associated morbidity and mortality. SCIDA offers a 7 stepped diagnostic algorithm approach that includes suspect, screen, confirm, classify, categorize, evaluate and diagnose hypoglycemia. At the outset, though diabetes related hypoglycemia seems to be more common in practice, non-diabetes related hypoglycemia still remains a possibility among rare presentations. This step approach provides a guide in history taking, examination findings and selection of relevant investigations to aid towards etiological diagnosis.

SCIDA would further help to validate targeted treatment selection options beyond dextrose administrations, thereby preventing recurrences and thus improving quality of life among patients with hypoglycemia.

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