Case Report

The Truth Lies Within the Gap

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Abstract

We report a case in which a post-analytical transcription error in serum osmolality led to diagnostic delay and unnecessary investigations in a patient with hyponatremia. The principal learning point of this case is that discrepancy between measured and calculated osmolality—the osmolal gap—was critical to detecting the error and redirecting the diagnostic evaluation. A 68-year-old female with a past medical history of hyperlipidemia, hypertension and type 2 diabetes mellitus, was scheduled for inpatient open reduction and internal fixation of a right distal radius fracture under the Hand surgery service. She was referred to the endocrinologists in view of persistent hyponatremia. Pre-operatively, her serum sodium was 128mmol/L (reference interval 135-145mmol/L), potassium was 4.2mmol/L (3.5-5mmol/L), urea was 4.8mmol/L (2.0-6.5mmol/L), random glucose was 5.2mmol/L (4.0-7.8mmol/L) and measured osmolality was 296 mOsm/Kg (275-300 mOsm/Kg). Records of her previous serum sodium trend were reviewed, and all were within the normal reference interval since 2011. In this case, the serum osmolality was within our laboratory's normal reference interval albeit at the higher end at 296 mOsm/Kg. The initial clinical impression at this point of time was thus normo-osmolar pseudohyponatremia. The clinical pathologist's opinion was requested, and the patient's serum sodium was further evaluated on the RAPIDLab® 1200 Systems Siemens blood gas analyser. This returned a similar sodium result of 128 mmol/L, excluding the possibility of electrolyte exclusion effect on the initial indirect ISE (Beckman Coulter, CA, USA). Upon further investigation, it showed that the osmolal gap was spuriously elevated (30mOsm/kg) due to a transcription error for the measured osmolality, hence confounding the true aetiology of hyponatremia. This case has shown that prompt communication between laboratory professionals and physicians will assist in resolving clinically discordant results. The osmolal gap, in particular, served as a useful adjunct in working up an ambiguous cause of hyponatremia.

Osmolality is an important laboratory marker reflecting the colligative property of a solution, measuring the solute concentration. It may be defined as the number of osmoles of solute per kilogram of solvent, and indicates the amount of solute particles dissolved in solution [1]. The osmolal gap (OG) is the difference between the measured osmolality (MO) and calculated osmolality (CO) and an increased gap suggests the presence of unmeasured, osmotically active particles. Where the MO is usually assessed by freezing point depression method on osmometers in most clinical chemistry laboratories, CO is determined using a number of osmolality equations [2]. A widely accepted and simple equation for CO initially proposed by Smithline et al, and used in our laboratory is: 2 x Sodium + Glucose + Urea (all in mmol/L) [3]. Hyponatremia remains the most common electrolyte disorder, seen in up to 30% of hospitalized individuals [4]. There are a multitude of potential aetiologies of hyponatremia, requiring meticulous work up by the physician and investigations often involve the determination of serum and urine osmolality as well as urine sodium.

The laboratory plays and important role in ensuring reliable results are reported and therefore interpreted correctly in such cases. Herein this report presents the evaluation of a surgical patient with hyponatremia posing a diagnostic conundrum to the clinical team, requiring a multi-disciplinary team approach with laboratory consult for successful resolution. Discussion of this case involves integrating clinical aspects of chemical pathology in the investigation of hyponatremia, osmolality concepts, as well as analytical understanding of the osmometer instrument.

Case Report

Case Description

A 68-year-old female with a past medical history of hyperlipidemia, hypertension and type 2 diabetes mellitus, was scheduled for open reduction and internal fixation of a right distal radius fracture. She was referred to the endocrinologists in view of persistent hyponatremia. Clinically, the patient was asymptomatic and did not report nausea, vomiting, giddiness, lethargy or any changes in her mental status. She indicated a recent increase in fluid intake from 500-800ml/day to 1000ml/day. At the time of admission, she was on Simvastatin 20mg, Lisinopril 10mg, Metformin 250mg. Pre-operatively, her serum sodium was 128mmol/L (reference interval 135-145mmol/L), potassium was 4.2mmol/L (3.5-5mmol/L), andom glucose was 5.2mmol/L (4.0-7.8mmol/L) and measured osmolality was 296 mOsm/Kg (275-300 mOsm/Kg). Records of her previous serum sodium trend were reviewed and all were within the normal reference interval since 2011. The patient demonstrated normal renal function with a serum creatinine of 66 µmol/L (reference interval 50–90 µmol/L) and urea of 4.8 mmol/L, indicating preserved renal function. These findings support the exclusion of renal insufficiency as a cause of hyponatremia.Physical examination revealed that she was clinically euvolemic with normal skin turgor, appearance of a slightly dry tongue but no pedal oedema or features suggestive of Cushing syndrome.

When approaching the problem of hyponatremia, it is helpful to analyse serum osmolality to determine the underlying cause. In this case, the serum osmolality was within our laboratory's normal reference interval albeit at the higher end at 296 mOsm/Kg. The initial clinical impression at this point of time was thus normo-osmolar pseudohyponatremia possibly contributed by use of TCMs. The clinical team had instructed the patient to cease taking her TCMs, but the hyponatremia persisted. Further investigations revealed total cholesterol of 3.93mmol/L, triglyceride level 0.67 mmol/L and LDL-cholesterol 2.24mmol/L. A myeloma panel performed did not detect abnormal bands on serum protein electrophoresis, with a normal serum protein level of 72 g/L (65-82) including that of albumin and globulin. The laboratory's opinion was requested, and the patient's serum sodium was further evaluated on the RAPIDLab® 1200 Systems Siemens blood gas analyzer, which is a direct Ion selective electrode (ISE) method. This returned a similar sodium result of 128 mmol/L, excluding the possibility of electrolyte exclusion effect on the initial indirect ISE (Beckman Coulter, CA, USA).

Additional investigations were ordered for endocrine evaluation of hyponatremia including a thyroid function test (TFT) and 250mcg short synacthen test. TFT demonstrated normal free thyroxine of 15.2 pmol/L (8-16pmol/L), and TSH of 0.71 mIU/L (0.45-4.5mIU/L). An appropriate cortisol response to the short synacthen test with a peak cortisol of 609nmol/L one-hour post-administration excluded adrenal insufficiency.

Laboratory Troubleshooting and Intervention

The "normal" measured serum osmolality results now appeared to be a red herring. Pseudohyponatremia was unlikely in view of the investigations performed above. Our laboratory team first proceeded to calculate the osmolality, as the clinical picture and patient's laboratory results appeared to be discordant. The results were carefully scrutinized and the calculated osmolality, based on the formula of 2 x Na + Glucose + Urea (all in mmol/L) indicated an elevated osmolal gap of 30 mOsm/kg (normal <10mOsm/Kg). The increased osmolal gap was immediately alerted to the endocrinologists but there was no clinical evidence to suggest any ingestion of osmotically substance, such as ethanol. This led to the suspicion of possible error in the initial reported serum osmolality of 269mOsm/kg. In our laboratory, a bench top Advanced Micro-Osmometer Model 3320 is used for osmolality measurement in urine and serum. It utilizes the technique of freezing-point depression to measure the osmolality of the solutions. A volume of 20uL of sample is inserted into the osmometer through a sampler with a new sampler tip. After one minute, the result will be displayed, recorded onto an osmometer worksheet and manually typed

onto the laboratory information system (LIS). The workflow for our osmolality samples, including the sample in question, is that they are analyzed only once before reporting by the medical technologist on duty. The initial sample earlier in the day was retrieved and reanalyzed three times for osmolality where the repeated results were 269 mOsm/Kg, consistent across three reruns. The initial result (296 mOsm/kg) was thus amended to 269 mOsm/kg.

Core Teaching Point: Post-analytical Transcription error

The large discrepancy between the initial result and the corrected reruns pointed to a post-analytical transcription error as the likely cause. Such errors typically occur when laboratory results are inaccurately transcribed from the worksheet into the LIS, leading to incorrect reporting. The diagnosis was thus changed from normo-osmolar hyponatremia to hypo-osmolar hyponatremia, which in a setting of post-fracture pain, was consistent with the syndrome of inappropriate anti-diuretic hormone secretion (SIADH). This was further supported by a urine sodium of 60mmol/L as well as inappropriately elevated urine osmolality (322mOsm/kg). The patient's fluid intake was restricted to 1.5L/day and she was followed up in outpatient clinic, with her serum sodium subsequently returning to normal. The table below summarizes the patient's key investigations and results:

Table 1: Summary of patient's clinical feature, investigations and laboratory troubleshooting.

Clinical features	Basic investigations	Endocrine function testing	Lab troubleshooting
•Distal radius fracture •Euvolemic hyponatremia with no changes in neurological state •No weakness, fatigue, confusion or drop in Glasgow coma scale	•Na 128 mmol/L (135-145) •Serum osmolality 296 mmol/kg (275-300) •K 4.2 mmol/L (3.5-5) •Urea 4.8 mmol/L (2-6.5) •Creatinine 66 umol/L (50-90) •Total Cholesterol 3.93mmol/L; Triglyceride 0.67mmol/L; HDL-C 1 mmol/L; LDL-C 2.24 mmol/L •Glucose 5.2mmol/L	•8am serum cortisol 285 nmol/L (123-626) •Peak cortisol of 609nmol/L post short synacthen test •fT4 15.2 pmol/L (8-16pmol/L), and TSH 0.71 mIU/L (0.45-4.5mIU/L)	Direct ISE on blood gas analyzer •Similar result to indirect ISE, Na 128mmol/L • Unlikely pseudohyponatremia Calculation of osmolal gap •Elevated osmolal gap of 30 mOsm/kg • Informed endocrinologists to exclude potential causes of elevated osmolal gap Retrieved initial serum and re-analyzed serum osmolality x 3 • Likely true result of 269mOsm/kg transcribed wrongly as 296mOsm/kg Quality improvement measures instituted and will be explained in discussion

Discussion

Approach to Hyponatremia

Cumming et al have described the prevalence of hyponatremia in their cohort of elderly fracture patients to be 14.2% [5]. The aetiology of hyponatremia was found to be multifactorial in 72.7% of patients and the commonest factors include thiazide diuretics, dehydration, proton pump inhibitors, SIADH and mirtazapine. SIADH will be elaborated in further detail below. Biochemically, hyponatremia may be defined as a decreased plasma sodium concentration. In our laboratory, the lower limit of the reference interval is 135mmol/L, in line with recommendations from Biswas et al [6].

Hyponatremia is classified as hypo-osmolar, hyper-osmolar, normo-osmolar and hence the most important initial step is the determination of plasma osmolality [7]. Normo-osmolar hyponatremia in the presence of normal glucose and urea should alert one to the high likelihood of pseudohyponatremia attributed by the electrolyte exclusion effect. This occurs as a consequence of excess lipid molecules or proteins, which occupy the solid fraction of serum, leading to interference [8]. The causes an underestimation of sodium in indirect ISE analysers, as there is a predilution step involved and the indirect ISE assumes that the solid fraction of serum is 7%. Performing sodium analyses on a direct ISE analyzer will therefore negate the effect of pseudohyponatremia. In this case, a myeloma screen, a lipid panel as well as a direct ISE analysis of sodium excluded the possibility of pseudohyponatremia.

Hyper-osmolar hyponatremia exists when there are increased quantities of other solutes in the extracellular fluid. This leads to extracellular shifts of water, or intracellular shifts of sodium to maintain the osmotic balance between the extracellular fluid and intracellular fluid. Common causes include hyperglycemia, mannitol or glycine.

Hypo-osmolar hyponatremia is a "true hyponatremia" in a sense that there is excess loss of sodium (depletional hyponatremia) or an increased extracellular fluid volume (dilutional hyponatremia). Patients with depletional hyponatremia are often hypovolemic and exhibit clinical signs such as hypotension, tachycardia and reduced skin turgor. Severe mental confusion with seizures may occur at values less than 105mmol/L [7]. An important further investigation in this

scenario would be urinary sodium. If urinary sodium is low (<10mmol/L), this indicates proper renal function and that the source of sodium loss would likely be from the gastrointestinal tract or skin. Urinary sodium losses of >20mmol/L generally suggest that renal loss is more likely. Dilutional hyponatremia occurs as a consequence of excess water retention and typically manifests as oedema on physical examination. Causes of dilutional hyponatremia include renal failure, congestive heart failure, cirrhosis as well as nephrotic syndrome. Hypo-osmolar hyponatremia in the context of euvolemic volume status in the patient occurs in SIADH, primary polydipsia and endocrine disorders such as adrenal insufficiency and hypothyroidism. An algorithm being used in our laboratory is provided in Figure 1.

Clinical aspects of SIADH

SIADH is the commonest cause of euvolemic hyponatremia in hospitalized patients [10]. It occurs as a result of unsuppressed secretion of the antidiuretic hormone (ADH, arginine vasopressin or AVP) resulting in inappropriately concentrated urine and therefore a reduced urine volume [11]. In most patients with SIADH, ingestion of water fails to adequately suppress ADH hence urine remains concentrated. This causes inappropriate and excessive free water retention, increasing total body water and subsequently dilutional hyponatremia. Subsequently the phenomenon is compounded by the body's response to water retention by suppressing renin-angiotensin-aldosterone axis, thus resulting in even greater renal sodium wasting [10]. As a result, a high urinary sodium excretion will be observed. The causes of SIADH can generally be divided into five main causes as described by Hannon et al in the European Journal of Endocrinology [12]. These include (1) Malignancies such as small cell lung cancer, sarcoma, lymphoma, gastrointestinal tract and pancreatic tumors; (2) Drugs commonly implicated are selective serotonin reuptake inhibitors, carbamazepine and tricyclic antidepressants; (3) Pulmonary causes such as Pneumonia, tuberculosis and vasculitis; (4) Intracranial pathology for example meningitis, gliomas, abscesses, and subarachnoid haemorrhage; (5) Miscellaneous causes for example post-operative pain, acute intermittent porphyria and Guillain-Barre syndrome.

For this case, in the absence of any drug aetiology, liver/renal impairment, or endocrinopathies such as thyroid/adrenal dysfunction, our team attributed her hyponatremia SIADH to being pain induced. The pathogenesis of pain leading to SIADH has been previously described by Mavani et al [13]. Arginine vasopressin, or ADH, is reported to have significant effects on pain perception in the central nervous system via the receptors V1a and V1b located within the neuronal cells and hypothalamus. In response to painful stimuli, central and peripheral ADH levels are reportedly increased to act as a potent analgesia, thereby increasing the body's pain threshold and reducing the nociceptive signals [14]. Post-fracture pain was identified as the precipitating cause of SIADH in this patient, in keeping with the known neurogenic stimulus for ADH release.

Biochemical and Clinical Diagnosis of SIADH

Based on the clinical guidelines from the European Society of Endocrinology, essential biochemical criteria for SIADH include low serum osmolality <275mOsm/kg, Urine sodium >30mmol/L with normal dietary salt intake and inappropriately elevated urine osmolality >100mOsm/kg. In addition, there the patient should be clinically euvolemic with the exclusion of adrenal, thyroid, or renal insufficiency with no recent use of diuretic agents [15]. These are summarized in table 2 below. Management SIADH includes initial measures such as fluid restriction and hypertonic saline. Drug treatments may be rendered for more severe SIADH including demecleocycline, an ADH inhibitor and Vaptans, which are ADH receptor antagonists. Earlier identification of the transcription error would have avoided unnecessary investigations, shortened the diagnostic evaluation, and enabled earlier initiation of targeted management with fluid restriction and pain control. In this case, our patient responded well to fluid restriction, pain control of the fracture, and close monitoring of serum sodium.

Table 2: Criteria for Diagnosis of SIADH.

Clinical Criteria	Biochemical Criteria	Exclusion cases
• Clinically euvolemic – No clinical signs of tachycardia, decreased skin turgor or dry mucous membranes – No oedema or ascites • No recent use of diuretics	• Serum Na <135mmol/L • Low serum osmolality <275 mOsm/kg • Urine Na >30mmol/L • Urine osmolality > 100 mOsm/kg	• No adrenal insufficiency • No hypothyroidism • No renal insufficiency

Clinical consequence – delay in care and unnecessary investigations

Of note, it is important to mention that our patient fulfilled the clinical criteria of SIADH but her diagnosis was confounded by an erroneous laboratory result. This had led to unnecessary further investigations and delay to timely diagnosis and appropriate management. For example, the normoosmolar hyponatremia led the clinical team down the rabbit hole of pseudohyponatremia which included work-up for lipids, myeloma screen and direct ISE testing. Thankfully there were no adverse outcomes and interventions in her case, but this underscores the fact that an estimated 70 percent of medical decision-making predicates on reliable laboratory test results [16]. As laboratory professionals, any discordant results with the clinical picture mandates us to do due diligence to thoroughly investigate the laboratory processes involved. In this case, our laboratory professionals picked up the discrepancy between measured and calculated osmolality in a timely fashion.

Osmolality – The Truth lies in the Gap and the discovery of Transcription error

When screening patients for the presence of exogenous osmotically active substances such as ethylene glycol and ethanol, the presence of an increased osmolal gap is important. This is calculated by subtracting calculated osmolality from measured osmolality. Choy et al have documented up to 37 different formulae for calculated osmolality, but detailed comparisons are beyond the scope of this report [1]. Nonetheless, a common and widely accepted formula adoped by my laboratory remains the Smithline-Gardner formula which uses 2 x Sodium + Glucose + Urea (All values in mmol/L) [3]. A normal osmolal gap should typically be less than 10 mOsm/Kg and an elevated gap indicates presence of unmeasured, osmotically active molecules such as ethanol, mannitol or hyperlipidemia. Calculated osmolality and osmolal gaps are not reported in our laboratory at present. Importantly, the key diagnostic clue in this case was the unexpectedly elevated osmolal gap, which prompted re-evaluation of the measured osmolality. Recognition of this discrepancy between measured and calculated osmolality triggered repeat analysis of the stored serum sample and ultimately led to the identification of a post-analytical transcription error. This case therefore highlights the value of the osmolal gap not only as a screening tool for unmeasured osmoles, but also as a practical internal consistency check for detecting laboratory errors when results appear discordant with the clinical picture. As described above, the elevated osmolal gap of 30 mOsm/kg prompted the laboratory to relay this information to the clinicians to first exclude intoxicating substances. Upon confirmation that this was clinically unlikely, we then proceeded to retrieve the initial serum from our refrigerated stockyard for analyses as we now suspected a wrong initial result. Based on a study by Bezuidenhout et al [17], it was concluded that serum and plasma osmolality were stable for up to 36 hours. The Advanced micro-osmometer utilizing the freezing-point depression method had been shown by Koumantakis to have excellent analytical precision (within-run Coefficient of variation of 0.59%, between-day Coefficient of variation of 0.58%). Moreover, based on our external quality assessment subscription, internal quality control processes and instrument maintenance charts there was no issue with our instrument. Reanalysis of the serum revealed that in all likelihood, there was a post-analytical transcription error of 296 mOsm/kg instead of 269 mOsm/kg which led to the result being retracted and amended. Data from Plebani's group has indicated that errors in the post-analytic phase, including that of transcription errors, have a relative frequency of 12.5-20% within the total testing process [18]. These were especially prevalent in scenarios where manual keyboard entry resulted in error, or analytical instruments that were not adequately interfaced with laboratory information systems and this was an apt description of our case in question.

Key Incident Monitoring and Management System (KIMMS)

More recently, Badrick et al have reported valuable data from the Australasian Quality improvement programme known as the Key Incident Monitoring and Management System (KIMMS) [19]. KIMMS records incidents known as process defects as well as episodes to calculate incident rates for pre- and post-analytical errors, thereafter using a failure mode effects analysis (FMEA) to assign a quantifiable risk and harm to the incident. A relevant category to our case was "Result retraction or correction" which described the occasion when result(s) have been modified because of an error after release in any form by the laboratory, and included data entry errors. Under KIMMS, result retraction or correction events are recognised as high risk–harm incidents, as they involve the potential dissemination of incorrect diagnostic information that may influence clinical decision-making, patient management, or treatment pathways. Among the top ten categories, KIMMS data from 2008-2016 showed that "result retraction or correction" was the tenth highest with 91,895 (3.09%) of incidents. However, its weighted risk-harm metric was the second highest ranking at 4,502,855 (12.01%). This indicates that a retracted laboratory report based on post-analytical error confers a high patient safety threat, which may result in further unnecessary investigations or treatments to a wrong diagnosis.

Allowable limits of difference and duplicate testing

As osmolality is tightly controlled by the body, it would be challenging to apply the commonly used analytical goal formula of $CVa < 0.5*CVi$ because the biological variability of osmolality reported in literature ranges from only 1.0-1.3% [20]. One option to mitigate total error would be to apply allowable limits of performance, which will allow my laboratory to assess its performance based on consensus

analytical performance goals. An available external quality assurance (EQA) programme, which my laboratory also subscribes to, would be the RCPAQAP General Serum Chemistry program. This has published extensive biochemistry data including that of osmolality. As outlined in the article by Jones et al [21]. such supporting information will allow users to determine if assays are meeting the requisite quality standard to diagnose or monitor patients within or between laboratories. For the use of serum or urine osmolality in diagnosis, the basis of limits given by RCPAQAP are that of minimal total error (bias and precision), in particular +/- 8 up to 266mOsm/L or 3% if more than 266 mOsm/L. Repeat testing, such as the one proposed by Deetz et al, involves automatically repeating laboratory tests when values exceed a critical threshold or trigger other automated rules such as a delta check in order to improve the accuracy and reliability of results [22]. A viable strategy that could be proposed would be to support osmolality analyses with duplicate testing to ensure the allowable limits of performance are not exceeded to mitigate error, and to provide warning of clinically important changes in assay performance. If these analytical performance limits were exceeded, it would then prompt investigation into one's quality controls and reporting processes and trigger repeating testing in order to troubleshoot potential error. If this had been performed for our case, we would have been able to pick up a discordant osmolality of an erroneous 296 mOsm/L versus a correct 269 mOsm/L result (difference of 27Mosm/L or 10% difference) and this would have saved the patient from unnecessary further investigations.

This case highlights several important considerations and learning points. If the result of an osmolal gap were readily visible to the clinicians on the electronic medical records, they could have significantly reduced the time taken to diagnose this patient. This could also potentially eliminate the need to order other redundant tests, resulting in cost savings. As such, our laboratory is considering the implementation of reportable calculated osmolality and osmolal gaps for all patients using programmable formulae on the laboratory information system (LIS).

Corrective and Preventive measures

We reviewed our current laboratory practice for medical technologists reporting osmolality results. Two medical technologists on duty are designated to perform osmolality testing. The first technologist performs the direct analyses and writes the result on an osmolality worksheet, whilst the second technologist verifies the results and transcribes the results onto the LIS. We suspect that the first technologist had transcribed the result wrongly onto the worksheet and the second technologist simply validated this result instead of checking on the osmometer screen. We improved our laboratory processes in order to reduce post analytical transcription error rate and enhance patient safety. The second technologist now has to verify the result directly displayed on the osmometer and on the osmolality worksheet before validating the result. Staff also were reminded and went through retraining to ensure competency in transcription of results into the LIS.

There was a need to review whether this was the appropriate osmometer model for our laboratory. Although our current purchased osmometer has not reached the end of its life-cycle, this case provided impetus for considering other models which would better suit our needs. The daily samples for urine/serum osmolality received by our laboratory remains more than 10 samples a day, with both adult and paediatric patients requiring osmolality determination. To reduce manual intervention, a consideration would be an osmometer from Advanced Instruments with integrated barcode scanner, automated liquid handling as well as middleware integrated with our LIS, known as A20 Advanced Automated osmometer [23]. This will be proposed during our next procurement process.

The summarized steps taken to mitigate and prevent further such occurences

1. Dual medical technologist verification at osmometer before transcription into the LIS
2. Retraining of staff to ensure competency in manual processes such as transcription for all analysers which are not interfaced
3. Proposal for automated osmolal gap calculation
4. Reviewing Osmometer instruments during our next procurement to include models which have the option of interfacing with the LIS

Conclusion

Taken together, this case has shown that prompt communication between laboratory professionals and physicians will assist in resolving clinically discordant results. It further emphasizes the importance of carefully examining pre-analytical, analytical and post-analytical laboratory processes for abnormalities. The osmolal gap, in particular, served as a useful adjunct in working up an ambiguous cause of hyponatremia. Reliable laboratory results must not be taken for granted, and chemical pathologists maintain an important role in bridging clinical details with technical laboratory aspects.

Declaration of Conflict of Interests and Disclosures

All authors declare that they do not have any conflicts of interest and nothing to disclose.

Ethical Approval

This incident report constituted part of our Laboratory's continuous quality improvement where errors and root cause analyses are performed. Ethics approval was sought from our local institutional review board and exemption was provided (2023/00104). Best efforts have been made to ensure anonymity.

Credit Author Statement

ST: Conceptualization, Methodology, Formal analysis, Investigation, Writing - Original Draft, Writing - Review and Editing

AY: Data Curation, Validation, Visualization, Writing - Review and Editing

SSaw: Conceptualization, Resources, Supervision, Writing - Review & Editing

SSethi: Supervision, Writing – Review and Editing

Funding and Data availability statement

This case report received no specific grant from any funding agency in the public or commercial sectors. The data that support the findings are stored on a password-protected computer owned by our hospital due to sensitive patient information. Access to the data is restricted in accordance with institutional policies to protect patient privacy. Researchers interested in the data may contact the corresponding author, subject to institutional and ethical approvals."

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