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INSIDE

A Rare Delayed Presentation of Deep Venous Thrombosis Progressing to Renal Graft Vein Thrombosis.

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ABSTRACT

We present a case of diabetes and hypertension with history of diabetic retinopathy and nephropathy with chronic kidney disease stage-V (D) who underwent live related renal transplant. In post transplant period, he had delayed graft function which gradually recovered and was being planned for discharge. At the time he developed right lower limb deep venous thrombosis which rapidly progressed to graft vein stenosis in matter of hours and was successfully managed with thrombolysis, PTA and pulverization of the thrombus. The patient recovered good renal function and is presently having a serum creatinine of 1.7 mg/dl.

CONCLUSION

Timely and aggressive management of acute complications like graft vein stenosis can have favorable graft outcomes.

Mr. A, a known case of diabetes since 1998 and hypertension since 2012 with diabetic nephropathy and retinopathy with chronic kidney disease stage-V was on maintenance hemodialysis for past few months. He was being worked up for renal transplant with prospective donor being his wife. After a thorough pre-transplant work up and T and B cell CDC cross match negative his renal transplant surgery was performed. He was given ATG induction and kept on triple drug immunosuppression tacrolimus, mycophenolate mofetil and steroids. Total ischemic time was 25 minutes and warm ischemic time was 5 minutes. Post-operative he had urine output of less than 100 ml over next 2 hours. Ultrasound Doppler done showed good flows at hilum with RI of 0.77and 0.72. Local causes like catheter site obstruction were also ruled out. His urine output remained low and

hemodialysis was done at night. A clinical diagnosis of ATN was made and tacrolimus was withheld and second dose of ATG was given. Tacrolimus levels sent were 29.7 ng/ml. Over a period of next 2 days, his urine output improved. His tacrolimus levels started decreasing and gradually tacrolimus was reintroduced. On 7th postoperative day, his serum creatinine came down to 1.7 mg/dl. His blood sugars fluctuated and required insulin infusions intermittently. His urine culture grew E Coli sensitive to colistin and colistin and meropenem were added. He complained of breathlessness and was evaluated. Chest medicine opinion was also taken. CT thorax and Pulmonary function test done were within normal limits. Breathlessness gradually improved by fluid management only. He was planned for discharge on a serum creatinine of 1.8 mg/dl when he complained of mild pain in right lower limb. On examination, right lower limb had mild swelling. A venous doppler done was suggestive of deep venous thrombosis extending up to right external iliac vein but renal artery flows were normal. He was immediately started on low molecular weight heparin. In a period of few hours, he had a fall in urine output. A high clinical suspicion of graft vein thrombosis was kept and after explaining to family his CT venogram was done which showed deep venous thrombosis extending from right external iliac vein up to below knee including anastomoses of allograft renal vein. Cardiologist and vascular surgeons were involved and after discussing also possible options and there outcomes with the family TPA thrombolysis and vascular intervention was planned. On fluoroscopy thrombus filled right allograft renal vein and right common femoral vein without any flows to common iliac vein were detected. PTA and pulverisation of the thrombus was done and TPA was injected. TPA thrombolysis was continued post intervention and heparin infusion with regular monitoring of APTT was done. Post procedure urine output remained low and his hemodialysis was done. Gradually over next 24 hours his urine output improved. He required one more session of hemodialysis before his serum creatinine started decreasing. He was gradually shifted to acitrome. His INR was monitored and patient discharged on a serum creatinine of 2 mg/dl. He is on regular follow up and maintaining a serum creatinine of 1.7 mg/dl.

REVIEW

Renal allograft thrombosis may be responsible for 2–7% of early allograft losses in adults and up to 35% in children[1,2,3]. In a study conducted by Zilinska, ET AL out of 103 renal transplant patients (January 2008 to December 2009) studied they detected renal vein thrombosis in 3 cases (2.9%), artery thrombosis in 4 cases (3.9%), one time intrarenal pseudoaneurysm (1%) and renal artery stenosis in ten patients (9.7%) [4]. Most cases of renal allograft thrombosis occur early in the postoperative period with a peak incidence of 48 h. However, thrombus formation may be delayed until after the first week[1]. Thrombosis may initially involve the renal artery or more frequently the renal vein, but in some cases it is difficult to ascertain where the thrombosis originated. Predisposing factors for renal allograft thrombosis include

- Hypovolaemia
- Atherosclerosis
- Technique Error
- OKT3 (plus high-dose methylprednisolone)
- Antiphospholipid Antibodies
- High Dose Steroids
- Long Cold Ischaemia Time
- Delayed Graft Function Recovery
- Elderly Donors

Late allograft thrombosis has been defined as occurring later than 14 days postoperatively[1,5], but rarely renal artery thrombosis may develop a few months post transplant. Renal allograft vein thrombosis may be induced by renal vein kinking or by renal vein compression caused by lymphocele or other fluid collection, and often results from extension of deep vein thrombosis to the renal allograft vein[1,6]. A review of the USRDS data[1,7] found that in renal transplant recipients deep vein thrombosis had an incidence of 2.9 episodes/1000 persons year; the risk was greater for patients with renal insufficiency and with nephrotic syndrome, increased haematocrit, rejection, infection or factor V Leiden mutation. The prognosis is poor because many patients lose their graft function, but some may be rescued depending on the timeline of the diagnosis. Pulmonary embolism is a complication of renal vein thrombosis especially with deep vein thrombosis. Treatment with streptokinase or urokinase may be useful particularly in case of acute or partial vein thrombosis. Percutaneous mechanical thrombectomy and localized catheter-directed thrombolysis may also allow the return of kidney function in some patient[1,8].

DISCUSSION

As clear from above review our patient had some predisposing factors in form of an infection and delayed graft recovery. The above factors may have pre-disposed our patient to a thrombotic state vis-a vis all patients with these predisposing factors do not have thrombotic events. But early diagnosis and prompt and aggressive management led to resolution of the thrombosis and recovery of renal function.

CONCLUSION

Complications like renal graft vein thrombosis can present atypically and in late period as extensions of deep venous thrombosis but aggressive and timely intervention can have very satisfactory results and salvage renal graft.

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Crafting Nose to Increase Your Face Value!

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RHINOPLASTY (Nose Job & Reshaping)

Nose is the centerpiece of our face but it can become a problem if it becomes the centre of attention for others. Rhinoplasty aims at reshaping the nose to enhance its profile in tune with facial features. It is also possible to correct breathing problem during the same surgery, if needed.

IS IT RIGHT FOR YOU?

You may be conscious of your nose shape and size. Although in many cases this may be the nose that you may have been born with but sometimes it may be a result of trauma, cancer or previous surgery.

Rhinoplasty surgery can change nose size, in relation to the other facial structures, nose width at the bridge, nose profile, with visible humps or depressions on the bridge, nasal tip, that is large or bulbous, drooping, or too upturned, nostrils that are large, wide or upturned, nasal asymmetry and deviation

HOW IS IT DONE?

All patients are medically screened for fitness before surgery. The patient is asked to stop smoking completely for one month and avoid certain drugs like aspirin a week before surgery.

The procedure is performed under general anesthesia. Surgery of the nose is performed either using a closed approach, where incisions are hidden inside the nose, or an open approach, where a small portion of the incision is made across the columella (the narrow strip of tissue that separates the nostrils) is visible. Fortunately this scar almost becomes invisible with time.

Open approach is increasingly being used for tip corrections and complex rhinoplasties.

Surgery of the nose can reduce or augment nasal structures with the use of cartilage grafted from other areas of your body (septum, ear cartilage, rib cartilage) according to the requirement.

External nasal splint is applied to support the nose as it begins to heal for approximately one week. Nasal packs inserted at the time of surgery are usually removed within 24-48 hours. It is not unusual to get black eyes and blocked nose feeling after this surgery but these settle nicely in 2-3 weeks time. Initial swelling subsides within few weeks. The overall risks of the procedure are minimal in healthy individuals.

RECENT ADVANCES

Correction of severely deviated nose has now been made possible with the usage of an advanced bio-resorbable plate, which supports for 3months and then gets absorbed.

All in all Rhinoplasty is one of the most complex of all plastic surgery procedures but in the hands of an expert it can give satisfying, predictable and pleasing results.

ICUs Should Always Function on Closed Basis

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"The occasion is piled high with difficulty, and we must rise high with the occasion"
Abraham Lincoln

BACKGROUND

Intensive care is an innately complex specialty calling for dedicated round the clock trained intensivists for clinical and administrative calls. The amount of data and inputs are overwhelming and perplex. Patients are seriously ill putting organs and life at stake. This calls for ownership of the unit and patient care plans. Quality comes with ownership and moral onus. The intensive care units (ICUs) must be used in the most efficient way, as they are the most integral, sensitive and happening part of hospital layout. High quality and cost effective performance can best be achieved when responsibility and management are given to those who have the special expertise.

Care, compassion, competence, communication, courage and commitment are traits of a good critical care unit. Physicians, called intensivists, are being specially trained to manage intensive care units (ICUs) and provide focused, high quality care to critically ill patients. The presence of specially trained intensivists in these ICUs has started a 'turf' war. ICU is one of the most demanding areas and competency goes beyond knowledge, procedural proficiency, skills and most important attitude of a single organ specialist.

MODELS OF ADMINISTRATION

Various models of care have been proposed and followed worldwide with huge variations from region to region as well as within a hospital. Aim of each model is to utilize critical care resources judiciously in a cost effective manner with best patient outcomes. There are pros and cons of each model. There are numerous variables which helps us decide on ICU model of care.

ICU ADMINISTRATION MODELS

ICUs can have an 'open' or 'closed' arrangement. 'Open' means the physician responsible for the patient admits the patient to the ICU and keeps the formal responsibility for the patient and his treatment. The intensivist is a consultant

without primary responsibility for the patient. A 'closed' format means the patient is admitted to the ICU and the responsibility for the patient and his treatment is transferred to the intensivist.

THE OPEN UNIT MODEL

SCCM definition: Any attending physician with hospital admitting privileges can be the physician of record and direct ICU care; the presence or absence of a dedicated intensivist physician and nursing unit directors; the presence of ICU dedicated house officers variable; the potential for duplication of services, the lack of a cohesive plan, and inconsistent night coverage.

Since the formative years of critical care units, this models of critical care delivery have been popular, the so-called 'open model' in which admitting physicians direct the care of their individual patients with the assistance of house staff who provide round-the-clock on-the-spot patient care. This is just an extension of the traditional makeshift arrangement that operates in the setting of non-critical illness. The physicians are not specially trained or experienced in critical care. They utilize the basic ICU doctor / Anaesthetist in airway care and in placing vascular lines, while depending on their own clinical approach that is usually lacking in critical care orientation. An intensivist, if available, is not involved directly in the care of most of the patients. Management centres around treatment strategies being set, not merely on ad hoc correction of physiologic derangements.

This model also does not fully address the crucial issue of directing team work and coordination of care 24 x 7. It also does not allow a system to evolve that would standardize the approach to all patients, as individual idiosyncrasies would need to be accommodated. Thus, this model is primitive and would arguably lead to chaos and confusion.

THE CLOSED UNIT MODEL

SCCM definition: An intensivist is the physician of record for all ICU patients; full time ICU directors (physician and nursing); house officers usually present and usually full-time dedicated to the ICU; all orders and procedures carried out by ICU staff; potential for improved efficiency and standardized protocols for care; potential to lock-out private physicians and increase physician conflict.

A closed system implies that patient care is either directed or co-managed by an intensivist. The patients are in effect placed under the charge of an intensivist who delivers round-the-clock cover through suitably trained physician staff. He provides leadership in the management of individual patients, organizes 'on call' cover, puts in place infection control and quality care practices, staff training and clinical research. While leading the critical care service, the intensivist physician should have no competing clinical responsibilities which are possible only in a closed format.

THE TRANSITIONAL UNIT MODEL

SCCM definition: An intensivist director, trainees, and intensivist team are present as locally available; standard policies and procedures usually present; shared co-managed care between ICU staff and private physician; encourages optimal communication between ICU staff and community physician; may reduce physician conflict; ICU staff is the final common pathway for orders and procedures; potential for confusion and conflict regarding who has final authority and responsibility for patient care decisions.

Patients are admitted under the care of an internist, family physician, surgeon or other primary consultant, with intensivists available providing expertise via elective consultation. Intensivists may play a de facto primary role in the management of some patients, but only within the discretion of the primary consultant. Mandatory intensivist consultation may or may not be in place. Round-the-clock full time consultant intensivist cover may or may not be available.

The model that may be most acceptable is a team model in which primary physicians are integrated into day-to-day decision making. This would integrate the core values represented by both models.

MIXED ICU MODELS

In practice, the above models overlap to a considerable extent. Thus some literature avoids attempting to characterize ICUs in terms of these models and focus instead on the level of involvement of intensivists in patient care regardless of the organizational model. This involvement may consist of daily ICU rounds by an intensivist (thus including 'closed model ICUs' and 'intensivist co management'), ICU directorship by an intensivist (possibly including examples of all 3 models above), or simply the presence of a full-time intensivist in the ICU (also including examples of all 3 models.)

INTENSIVIST MODELS

ICU management may include all of these models. These models are contrasted with the open ICU model, in which an intensivist generally does not participate in the direct care of a significant proportion of the ICU patients.

BENEFITS OF CLOSED ICU MODEL

Multidisciplinary team concept does not just revolve around critical care team but encompasses primary physician, other specialist and various other health care providers as stake holders. It is vital to include specialist as and when required. ICU team has the obligation to cooperate and communicate in a closed loop with other HCP and family. Primary physician often does not have right knowledge and skill required for treating a seriously ill patient in ICU. He often apportions management to several single organ specialists.

This results in fragmented, non-cohesive treatment strategies. In this scenario the patient receives more tests and medication than he may need. There is little accountability with no physician assuming full responsibility for the patient. Families of patient also may receive contradictory and confusing opinions. In the closed model, the intensivist assumes full responsibility for treatment planning and integrates the inputs from several specialists. In the latter model, consultations are more meaningful and efficacious. The chances of protocols and standard operating procedures being developed is enhanced in this model.

Multidisciplinary approach is characterised by:

- Medical and nursing directors with authority and co-responsibility for ICU management
- Medical, respiratory therapy and nursing collaboration in a team approach
- Use of standards protocols and guidelines to assure consistent approach to medical, nursing, and technical issues
- Dedication to coordination and communication for all aspects of ICU management
- Emphasis on practitioner certification, research, education, ethical issues, and patient advocacy.
- The team dynamics in a multidisciplinary team is an essential precondition to ascertain the high quality of care, with the necessary reliability, promptness, and adaptation to the various demands. Creating a good team spirit depends very much on the social competence of the directors.

TIMELINESS

With a persistent trend of increasing ICUs volumes every year, services are intensifying. Unstable patient physiology, anxious family and a panicked primary physician all calls for an immediate consult while dealing with a seriously ill patient. Timeliness of care has been a focus of critical care. SCCM maintains that the Right Care, Right Now™ is best provided by an integrated team of dedicated experts directed by a trained and present physician credentialed in critical care medicine (an intensivist), also referred to as the multi professional team model. Timely and personal intervention by an intensivist reduces mortality, reduces length of stay, and decreases cost of care.

REVENUE MODEL

Care delivered by a multi professional team optimizes care for patients, improves conditions for healthcare providers, and boosts the financial performance of the hospital. In a closed model the revenue is consolidated ensuring transparency and clarity. This helps critical care develop as independent financial unit.

Intensivist management in the care of critically ill patients*

Study Setting	Study Year	ICU Type	Study Design, Outcomes	Intensivist Intervention	Mortality Relative Risk Reduction (%)	
					ICU	Hospital
Closed ICU Model						
Tertiary care, urban, teaching hospital; patients with septic shock; historical control[33]	1982-1984	MICU	Level 3, Level 1	Closed	NA	23
Teaching hospitals (n=2); two study designs using historical and concurrent controls[18]	1992-1993	MICU	Level 3, Level 1	Closed	NA	Retrospective: 19 (p=NS) Prospective: 26 (p=NS)
Tertiary care, urban, teaching hospital; historical control[32]	1993-1994	MICU	Level 3, Level 1	Closed	NA	-38 (p=NS)† O/E 13‡
Tertiary care, urban, teaching hospital; historical control[34]	1995-1996	SICU	Level 3, Level 1	Closed	58	50§
Mixed ICU models						
ICUs (n=16) with different characteristics; cross-sectional[16]	1989-1992	Pediatric MICU SICU	Level 3, Level 1	Mixed	RRR 25¶ OR 1.5**	NA
ICUs (n=39) with different characteristics; cross-sectional. Patients with abdominal aortic surgery[38]	1994-1996	SICU	Level 3, Level 1	Mixed	NA	OR 3.0§§
ICUs (n=31) with different characteristics; cross-sectional. Patients with esophageal resection[14]	1994-1998	SICU	Level 3, Level 1	Mixed	NA	RRR 73¶ OR 3.5**
ICUs (n=39) with different characteristics; cross-sectional. Patients with hepatic resection[15]	1994-1998	SICU	Level 3, Level 1	Mixed	NA	RRR 81¶ OR 3.8**
Community teaching hospital; historical control[40]	1992-1994	MICU	Level 3, Level 1	Open	29	28
Co-managed ICUs						
Tertiary care ICU in a teaching children's hospital[16]	1983-1984	Pediatric MICU SICU	Level 3, Level 3	Co-manage	48 (p=NS)	NA
Tertiary care, Canadian teaching hospital; historical control[39]	1984-1986	SICU	Level 3, Level 1	Co-manage	52	31
Tertiary care, urban, teaching hospital; cross-sectional comparison (concurrent control)[31]	1994-1995	SICU	Level 3, Level 1	Co-manage	NA	32 (p=NS)

* ICU indicates intensive care unit; MICU, medicalintensive care unit; Mixed, mixed intensivist model (including daily ICU rounds by an intensivist, the presence of a full-time intensivist, open units with comanagement and closed units with mandatory consultations or only intensivist management); NA, not available as outcome (was not evaluated); NS, not stastically significant; and SICU, surgical intensive care unit.

† Negative value indicates an increase in relative risk of mortality.

‡ O/E is observed to expected mortality ratio based risk adjustment

§ Hospital mortality measured 30-days after discharge

QUALITY & RESEARCH

When an intensivist is available in an administrative role in the ICU providing benchmarks, clinical research, and standardization of care, the data suggest that length of stay, cost of care, and treatment complications can be reduced.

PERCEIVED POTENTIAL FOR HARM

The potential for harm resulting from intensivist management is unclear. Concerns raised in the literature about intensivist managed ICUs include the loss of continuity of care by primary care physicians, insufficient patient-specific knowledge by the intensivist, reduced use of necessary sub-specialist consultations, and inadequate CCM training of residents who formerly managed their own ICU patients. Perhaps more worrisome is the impact that adoption of this practice would have on physician staffing and workforce requirements. Without a substantial increase in the numbers of physicians trained in CCM, projected increases in the ICU patient population over the next few decades will result in a significant shortfall in the intensivist workforce.

OPPORTUNITIES FOR IMPACT

Currently, a minority of ICUs in India utilizes the intensivist model of ICU management. Intensivists are even less frequently found in non-teaching and tier II cities hospitals. The potential impact of the intensivist model is far-reaching. Intensivists save lives and costs. By working toward team care, hospitals may achieve a successful closed ICU model, and patients may realize the benefits of spending less for healthcare and living longer. To achieve this model, physician and hospital leaders must form a close partnership and work as closed unit.

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Pouring 'Salt' Over Troubled Waters: Hypernatremia and Diarrhea in Children

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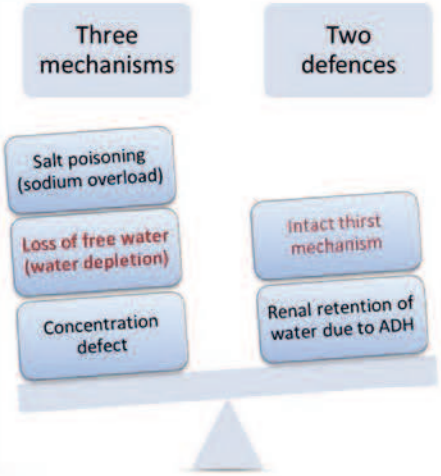
Diarrheal disease is the most common killer of children worldwide claiming close to 1 million lives every year. The introduction of oral rehydration therapy and implementation of hygiene and water sanitation have considerably reduced the burden of diarrheal disease. Widespread public knowledge about the importance of hygiene and ORS have at least prevented the serious complications of acute dehydration. But the silent and insidious complications of electrolyte abnormalities continue to contribute to deaths. It is imperative for doctors to be aware of and actively seek for these complications during treatment.

One such abnormality is hypernatremia. This is becoming relatively common and can cause devastating illness against a background of diarrhea. We have had several such cases in our unit over the last few years requiring prolonged PICU care, with unfavorable outcomes including death. Most of these cases occur during winter and rotavirus is the most common cause. Prompt recognition and ongoing surveillance for all children with diarrhea can prevent this potentially fatal occurrence.

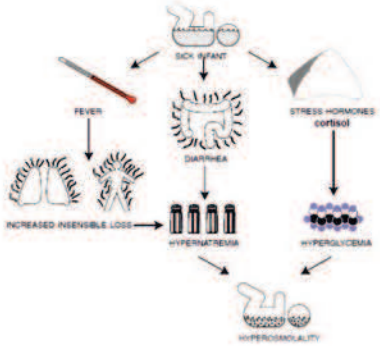
Hypernatremia is defined as a serum sodium level >145mmol/L. A level >170mmol/L is deemed severe with life-threatening consequences. Hypernatremia can be present in up to 12.5% of children hospitalized for diarrhea with dehydration. The cause of hypernatremia is free water depletion from the body. Hypernatremia occurs whenever there is an imbalance between salt intake on the one hand and water intake on the other: either excess salt or poor water intake can result in hypernatremia. A minor cause is a defect in the renal concentrating mechanism as occurs in diabetes insipidus. Often, poorly prepared ORS solutions are to blame, for example, it is common to encounter caregivers using ORS salts undiluted; not using the recommended proportion of water; preparing small volumes; all of which alter the sodium content in the resultant solution.

The two important defences against hypernatremia are an intact thirst mechanism and renal retention of water under the effect of

ADH. Infants are especially susceptible because they cannot indicate thirst and caregivers often cannot recognize thirst cues; with the result that hypertonic ORS solutions are given to the infant when in fact, he was thirsty and needed plain water! All dehydrated children produce maximally concentrated urine under the effect of ADH which is also secreted in response to pain, nausea or vomiting, or even emotional stress. This response to ADH is critical in preventing hypernatremia by retaining water. Again, this response may be blunted in infants leading to free water depletion. It should also be remembered that infants have a large volume of extracellular fluid (ECF) in the skin and connective tissue which is where most of the water depletion occurs.



As is well known, rotavirus causes the most severe form of gastroenteritis in children. Unlike other causes of diarrhea, rotavirus and other enteric viruses lead to hyposmolar fluid loss into the stools causing a net free water deficit which contributes to hypernatremia. Giving hyperosmolar or other proprietary ORS preparations can be detrimental both by delivering a high sodium load to the gut and glucose mal-absorption causing further osmotic fluid losses. A study from Sweden in 2010 highlighted an increase in the incidence of hypernatremia due to rotavirus diarrhea. However it could not be attributed to an increased virulence or prevalence of rotavirus. Instead the culprit was a commercial maltodextrin based ORS which caused a four-fold increase in the glucose concentration in the gut contents, leading to an osmotic free water loss. Similarly, in our country, it is a common practice to feed infants with boiled, skimmed cow milk which causes osmotic, low-salt diarrhea contributing to water loss and resultant hypernatremia. In addition, administration of antibiotics to children with viral diarrhea often complicates the picture by worsening fluid losses in stools.



Once hypernatremia sets in, there is a series of adaptive changes to help protect the tissues. Water shifts from the intracellular compartment to the ECF resulting in cell shrinkage; in the brain, this can amount to almost 10-15% of brain volume. Over the next 1-3 days, the cells retain sodium, potassium, amino acids, and short chain fatty acids (idiogenic osmoles) which restore the intracellular osmolarity, and by the end of one week, the cells regain up to 98% of the water lost.

Mostly complications from hypernatremia result from cell shrinkage, causing

- Hemorrhage from tearing of bridging veins in the brain
- Vascular thrombosis and infarction from increased viscosity causing cerebral & limb ischemia and demyelination

Systemic effects of hypernatremia include:

- Hemodynamic Instability
- Hyperglycemia, Metabolic Acidosis
- Acute Kidney Injury
- Encephalopathy: Irritability, Drowsiness, Seizures, Rigidity, Myoclonus
- Rhabdomyolysis

Mortality from hypernatremia can be as high as 15-40%, mostly in the acute cases. This is independent of the severity of hypernatremia. Acute kidney injury remains a serious cause of morbidity, often requires dialysis, and contributes to long PICU stay. Multi-organ dysfunction from dehydration, hyperviscosity, and infection also contributes to mortality.

Optimal management of hypernatremia depends on close observation and meticulous fluid administration to prevent complications. All children with diarrhea requiring hospitalization have to be screened for hypernatremia upon admission. Urine output must be measured accurately.

The basic principle is to replace the free water lost: this is calculated from the formula,

Free water deficit (mL) = 0.6 x body weight x [(serum sodium/ 140)–1]

This is aimed to gradually reduce the serum sodium level to 140mmol/L. Rapid replacement of fluid can push water into shrunken cells leading to edema and cell death. Most deaths and serious encephalopathy are a result of cerebral edema from overzealous fluid administration.

Oral replacement of free water is safer and more efficient. Up to 90% of the children can be treated with this route. Even though oral solutions are hypotonic they do not cause hyponatremia as the amount of water absorbed from the gut is proportionate to the degree of hypernatremia.

In a severely dehydrated child, rapid expansion of ECF volume can be achieved by infusing isotonic fluids like Ringer's lactate over 2-4 hours. Subsequent infusion of fluids will be guided by the serum sodium level. Slow infusion of isotonic fluid is recommended to achieve a gradual fall in serum sodium not to exceed 0.5mmol/L/hour. The type and amount of fluid are guided by the serum sodium levels and urine output.

Neither sodium content of the intravenous fluids nor the rate of fall of serum sodium have been shown to be independently associated with adverse outcome. The risk factors for cerebral edema include:

- Initial rapid fluid bolus
- Rate of initial bolus >10ml/kg/h
- Overall rehydration rate >6ml/kg/h\
- Severe hypernatremia (>170mmol/L)

SUMMARY

- Hypernatremia must be actively sought for in any child with rotavirus diarrhea or acute diarrhea during winter months
- Encourage parents to give plain water to infants with diarrhea in addition to ORS
- Avoid use of maltodextrin and other proprietary ORS preparations except low-osmolarity ORS recommended by the WHO
- Prepared electrolyte/ORS solutions like Pedialyte are safer and ensure correct composition
- Oral replacement of water in hypernatremia is safe and effective
- Meticulous fluid administration can prevent adverse outcomes

WELCOME TO THE TEAM

Dr. Arun Saroha

Senior Consultant & Head of Unit
Neurosurgery
Max Super Speciality Hospital, Saket

EDUCATION

- MBBS, Rabindra Nath Tagore (RNT) Medical College, Udaipur
- MS (General Surgery)
- M.Ch (Neurosurgery) PGIMER, Chandigarh
- Visiting Fellow: Singapore General Hospital, Singapore

EXPERIENCE

- Over 10 years of rich experience (post M.Ch) in Neurosurgery with special interest in Spine Surgery
- Previous Assignment - Head – Neurosurgery (Spine and Brain), Artemis Hospitals, Gurgaon

ACCOMPLISHMENT / AWARDS

- Performed 1000's of Neurosurgeries (Spine & Brain)
- Awarded 'Best Spine Surgeon' by Big Research Awards at New Delhi 2013
- Best Scientific Paper Award on 'Brain Tumor Research' at NSICON, 2003
- Awarded 'Best Resident for Critical Care in Neurosurgery at PGIMER' 2002
- Best Scientific Paper at IASO Conference, 1996

AREAS OF INTEREST

- Degenerative Spine Disorders
- Brain Tumors
- Disc Replacements

MEMBERSHIPS

- North American Spine Society – NASS
- AO Spine – Asia Pacific (Delegate)
- American Association of Neurological Surgeons - AANS
- Neurological Society of India - NSI
- Neurological Spine Society of India - NSSI
- Association of Spine Surgeons of India - ASSI
- Neurotrauma Society of India
- Association of Surgeons of India - ASI
- Walter E Dandy Neurological Society
- Paediatric Neurosurgery
- Spine Trauma & Complex Spine Fixations

Dr. Sonia Naik

Senior Consultant & Head of Unit II
Obstetrics & Gynaecology
Max Super Speciality Hospital, Saket

EDUCATION

- MBBS, Bhopal University, MP
- MD (Obstetrics & Gynaecology), Pandit Jawahar Lal Nehru Medical College, Raipur
- Post Doctoral Training in Obstetrics & Gynaecology, RML Hospital, Delhi
- Short Term RCOG Course on Labor Ward Management, RCOG, London
- Training in Hysteroscopy & Laparoscopy, Bradford Royal Infirmary, UK
- Training in Infertility, Reliance Life Sciences, Mumbai

EXPERIENCE

- Consultant, Sitaram Bhartiya Institute of Science & Research, Delhi

ACCOMPLISHMENT / AWARDS

- Instrumental in starting Mother & Child Programme at Sitaram Bhartiya
- Organizing secretary, Foetal Medicine Society CME
- Editor, Bulletin of Foetal Medicine Society

AREAS OF INTEREST

- High Risk Obstetrics
- Laparoscopic and Hysteroscopic Procedures
- Adolescent Gynaecology
- Infertility

MEMBERSHIPS

- FOGSI
- ISAR (Indian Society of Assisted Reproduction)
- Foetal Medicine Society of India
- Indian Menopause Society