IMA-KAUVERY NEWSLETTER

kauvery hospital

Alwarpet, Chennai

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Hon. Secretary

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Hon. Treasurer

Dr. R. Balasubramaniyam
Editor

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Secretary Message

Dear colleagues,

Greetings from IMA Chennai Kauvery Alwarpet Branch.

On behalf of our branch I would like to congratulate Prof.Dr.K.Senthil and his Team for achieving a landslide victory in the Tamilnadu Medical Council Elections .

We congratulate our IMA TNSB state office bearers for the active participation in the Mahapanchayat at Delhi and also assure our fullest cooperation in all the steps taken against the NMC bill.

The monthly Academic meetings of our branch were held on 08/01/18,02/02/18 and 02/03/18 at Kauvery Hospital,chennai and were well attended.

We request all our members to join the IMA PPLSS and FSS schemes

Long live IMA
Yours in IMA service
Dr. S. Sivaram Kannan

Editor Message

Dear friends

Happy to meet you all through our IMA newsletter.
It is astonishing to know the kind of medical care that is being provided at Kauvery Hospital. It is truly of international standards.
I am sure you would agree with me , once you go through the newsletter.

I am thankful to all the consultants for sharing their patient 's case summaries.

your comments and feedback are welcome

With regards
Dr. R. Balasubramaniyam
Editor

PRIMARY AMENORRHOEA- COMPLETE ANDROGEN INSENSITIVITY SYNDROME

DR KARPAGAMBAL SAIRAM
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Consultant Obstetrician, Gynaecologist and Fertility Specialist

16 year old Ms X presented to the outpatient clinic with her parents for having not attained menarche and to discuss the need for hormonal support.

She is the second child to parents married consanguineously. Her mother's antenatal period was uneventful and was born by normal vaginal delivery. The neonate was diagnosed to have bilateral inguinal swellings at birth with female external genitalia.
The parents were counselled about the disorder of sexual development and Karyotyping was done to check for ANDROGEN INSENSITIVITY SYNDROME(AIS), formerly called as TESTICULAR FEMINISING SYNDROME. The Karyotyping report showed 46XY.

The sex of rearing for these patients is the based on the external genitalia. During embryogenesis , present of testicular tissue and sensitivity to androgens will develop into male phenotype with Wolffian ducts , but the absence of testicular tissue can develop into female external genitalia without Fallopian tubes and uterus(absent Mullerain Ducts).So the sex of rearing becomes that of external genitalia(female)
The parents were explained about the need for removal of the inguinal swellings(gonads-Testes) after age 10-12 years till adequate bone development. The parents were also explained about the risk of malignancy developing in the inguinal undescended testes and she will be amenorhoeic and cannot conceive.

She developed mild autistic features at age of 3 years and she joined a special school for therapy.Around the same time , the child developed breathlessness and was diagnosed with Tetrology of Fallot for which she underwent complete correction.Ms X also underwent gonadectomy at the age of 12 years and the histopathology showed testicular tissue.

Years passed she grew up to be good looking tall girl with typical female secondary sexual characters.(Peripheral conversion of androgens to estrogens) .She is now under the care of a gynaecologist and endocrinologist, Paediatric cardiologist.

There are three distinct clinical features of genetic disorders in this child-Androgen insensitivity syndrome, Tetralogy of Fallot and Autism. We consulted clinical geneticist to identify any syndromic association with all three features. Routine Karyotyping did not reveal any microdeletion, translocation or non disjunction. So we may have to do Complete Genomic Hybridisation (CGH- microarray) to identify the subtle genetic changes . After counseling, the parents were not keen on further testing , so we assume that Autism and Tetrology Of Fallot are sporadic and AIS is the main genetic disorder.

Management

After gonadectomy, these girls need hormonal support with estrogens to maintain female secondary sexual characters as the peripheral conversion of testosterone to estrogens is absent.
As they lack uterus and ovaries, conception is not possible. The vagina usually presents as a dimple or a blind ending pouch. They require vaginoplasty to lengthen the vagina and create adequate space for sexual activity which can be done by serial dilators or moulds. Mc Indoes vaginoplasty is one such procedure . The girl and her parents need to be counselled regarding the procedure and the need for long term followup. This procedure is performed with help of plastic surgeons who help in creating flaps for the neo vagina. In case of partial AIS, surgery can be done to correct ambiguous genitalia.

Hormonal Therapy

These girls require a continuous hormonal support in the form of estrogens either as ethinyl estradiol or estradiol valerate atleast till the age of natural menopause (47-51 years) There is no need for progesterone as there is no uterus. We started this girl on 2mg Estradiol valerate with annual monitoring of LFT and GTT.

Discussion

The incidence of androgen insensitivity syndrome is 2-5 per 1,00,000 Population.
Transmission-X linked recessive (only males are affected and females are carriers)

Embryology

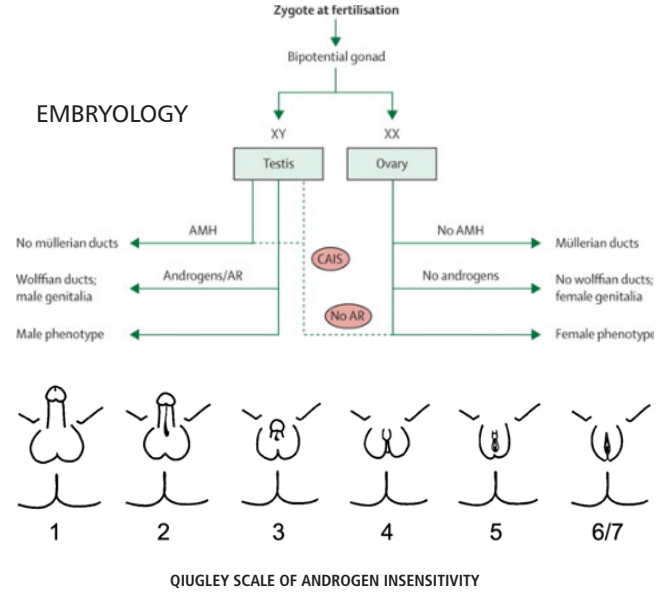
Classification

In 1996, Sinnecker classified AIS into 7 types based on the sensitivity to androgen receptors. Complete AIS denotes that the androgen receptor are totally insensitive to circulation androgens.
Partial AIS denotes varying degrees of sensitivity to androgens to androgen receptors and clinically manifests varies from female external genitalia to under-virilized male phenotype
QIUGLEY SCALE OF ANDROGEN INSENSITIVITY

Support groups

Various support groups are available to support these girls regarding the psychological and physical disabilities they would undergo.
AIS support group(AISSG, UK)
Rainbow network(Australia)
I personally encourage these girls to register and become members of these support group to share their emotions and feelings online.
It reassures them that they are not alone with these problems. There are many celebrities in different fields with AIS.

The management of these girls involve Multidisciplinary team involving Gynaecologist, Endocrinologist, Plastic surgeons, Counselors. The need of the hour is to have a national and regional registry of these Disorders of sexual development so that we can have our national statistics and formulate management guidelines and protocols.



QIUGLEY SCALE OF ANDROGEN INSENSITIVITY

A CASE OF BASILAR ARTERY OCCLUSION SUCCESSFULLY TREATED WITH TIMELY INTERVENTION

Dr. Sivarajan Thandeswaran
MBBS, MRCP (London)

Abstract
This article describes a case of acute ischemic stroke who presented with right sided weakness and complete opthalmoplegia. Initial NIHSS was 15 with hyper dense basilar artery sign on Plain CT Brain.
Angiogram revealed basilar artery occlusion with thrombus.
Intravenous thrombolysis initiated and bridged with mechanical thrombus aspiration. Basilar artery was successfully recanalized and patient achieved very good clinical outcome

Introduction
20% of the ischemic strokes occur in posterior circulation, of which Basilar artery strokes is the most devastating stroke, with fatality rate of almost 90% if left untreated. Involvement of the pyramidal tracts in pons causes a state of locked in syndrome which is characterized by quadriplegia, opthalmoplegia with preserved consciousness. Extension of infarct into adjacent brainstem structures further increases the mortality rate.

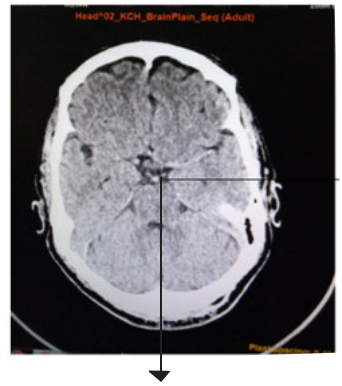
Case presentation
65 year old gentleman with past history of systemic hypertension, mitral valve replacement in 2013 for rheumatic heart disease and is on oral anticoagulant (warfarin) therapy. He also has history of TIAs twice (2012, 2017) in past which presented as slurring of speech and numbness of right upper and lower limb lasting for few hours.
Currently he was brought to emergency with complaints of sudden onset of numbness in right upper and lower limb at around 2.30am on 03/01/2018 which worsened within few minutes to complete paralysis. He also had slurring of speech with deviation of the angle of mouth to right side which started more or less at the same time.
On arrival to emergency at 6.30am, he was conscious, oriented with Glasgow coma scale of 14/15 (E4, V4, M6). CNS-marked slurring of speech and UMN facial palsy, right sided complete gaze palsy and right hemiplegia. Initial National Institute of Health Stroke Scale was 15. Hyperacute Stroke protocol was initiated

CT brain showed hyperdense basilar artery tip. In suspicion of basilar artery occlusion cerebral angiography was done which showed occlusion of V4 segment of left vertebral artery and short segment occlusion of basilar artery consistent with intra luminal thrombus. Interventional Radiologist was alerted and cathlab and anaesthetic team informed to receive patient for possible endovascular intervention.
As he was within window period and after checking for contraindications we proceeded with intravenous thrombolysis with actilyse (alteplase) and was taken to cathlab and Intra arterial mechanical thrombus aspiration was attempted and almost 100% of clot was retrieved using

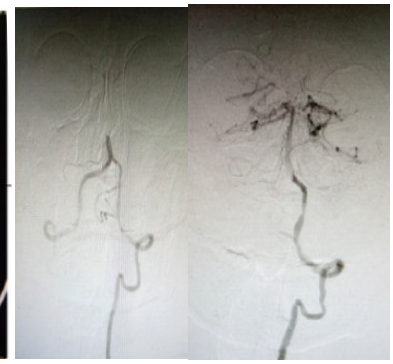
thrombo-aspiration technique and good flow achieved in vertebral and basilar arteries. Meanwhile alteplase infusion was completed simultaneously. Soon after aspiration of clot, his opthalmoplegia started recovering on Cath table itself and his right sided weakness also improved. He was transferred to HASU (hyper acute stroke unit) where close monitoring was done. He continued to improve neurologically. Repeat NIHSS was 5, being able to move both upper and lower limbs against gravity, speech was better, gaze palsy was completely improved, able to take feeds orally.
On the next day control CT revealed a small area of infarct in left upper pons and repeat cerebral angiography showed maintenance of basilar patency. Secondary prevention was initiated with antiplatelets, statins and LMWH. Rehabilitation with physiotherapy & speech therapy were commenced from day 2. By 72hrs he was able to transfer to chair and speak more clearly. At discharge on day 7 he was able to walk with minimal support. Followed up as outpatient and currently he was walking well without support and was able to resume his regular activities.
The outcome was outstanding as patient was brought in the optimal time window along with prompt diagnosis, facilities for imaging, good team work, early thrombolysis and fast access to cathlab and timely recanalization

Discussion :
Basilar artery runs along the ventral aspect of pons supplying it by the median pontine and transverse pontine arteries. It also supplies the superior and inferior aspects of cerebellum through AICA. In addition there are a number of perforating branches and collaterals supplying the medulla, midbrain and the adjacent structures. The key factor for determining the severity of stroke and the outcome depends on the blood flow in collaterals. Unfortunately these collaterals are very small to be picked up by angiography. Intravenous thrombolysis is extended only upto 4.5hrs but can be used upto 12hrs in basilar artery occlusion because without any attempt on recanalisation there is near total mortality. Despite extended time window with intravenous thrombolysis, the success rate of recanalisation is very abysmal at <10%. Hence in large vessel occlusion every effort should be made to remove the clot mechanically which has been approved internationally and is becoming a common practice worldwide. Recent studies have also showed that patients with BAO achieved good recovery from mechanical recanalization therapy even beyond 8 hours and upto 24hrs after onset of symptoms

Conclusion:
We conclude that any ischemic strokes should be recanalized as soon as possible, unless contraindicated. The earlier the recanalization, the best is the outcome & least is the disability. Any patients with atrial fibrillation and CHADS2-VASc score of more than 2, metallic valves, poor LV function with EF<33% who are at risk of LV thrombus should be adequately anticoagulated to prevent future risk of stroke. Physiotherapy and rehabilitation is also important for better outcome of stroke patients. Increasing the awareness of stroke in the community especially of 'newer treatment' possibilities that can nowadays reverse stroke is equally important.



Basilar artery dot sign is an appearance of increased attenuation in basilar artery consistent with intra luminal thrombus.



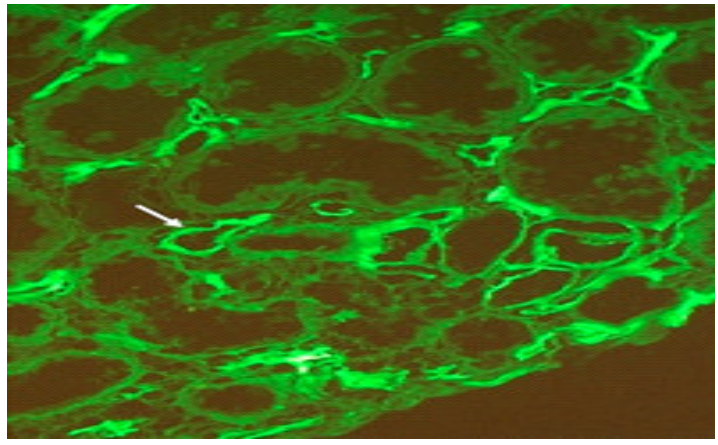
1 Angiogram showing occlusion of V4 segment of left vertebral artery and complete occlusion of basilar artery consistent with intra luminal thrombus.
2 Good flow seen in vertebral and basilar arteries following mechanical thrombus aspiration.

PATIENT WITH ACUTE POST KIDNEY TRANSPLANT GRAFT DYSFUNCTION

Dr. R. Balasubramaniyam
Sr Consultant Nephrologist

Mr.G, 24 years old male, was referred to us for post transplant management of his kidney dysfunction. He was diagnosed with chronic renal failure 2 years ago and after a brief period on dialysis, underwent his first kidney transplant (elsewhere) and he had severe rejection and kidney was removed within few weeks. After dialysis for few months he underwent another transplant (elsewhere) and he had kidney dysfunction again at the end of 2 weeks.
He had unmatched kidney transplantation and had positive

tissue cross match prior to transplantation. That was treated and then transplantation was performed. He underwent transplant kidney biopsy that was suggestive of acute cell and antibody mediated rejection. There were occasional oxalate crystals found on the interstitium. Clinical examination revealed well hydrated patient, who was mildly anemic, with good controlled blood pressure. His urine output was 200 ml/day. He was referred to us for further management.

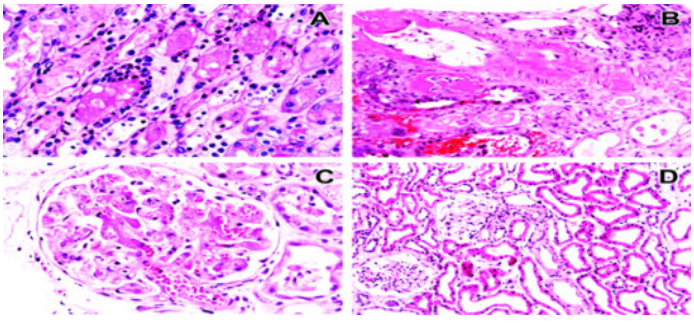


C4d deposits in the peritubular capillaries (Immunofluorescence).

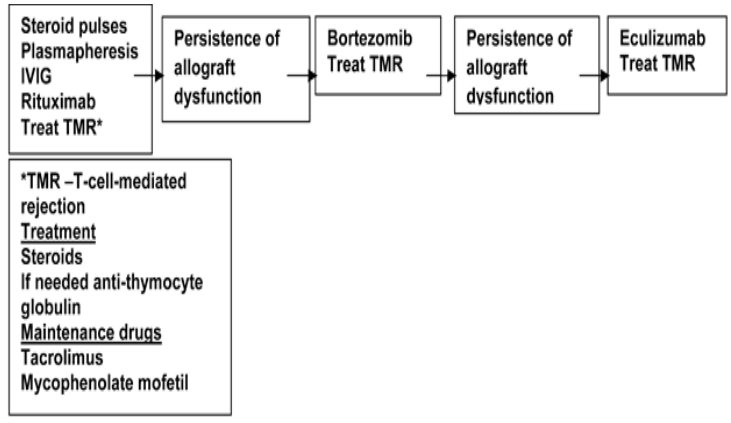
The acute graft rejection can be of 2 types: T – LYMPHOCYTE mediated rejection and antibody mediated rejection. The later is more severe and less treatment responsive. T cell mediated rejection principally involves tubulo interstitium and it responds to a greater extent to steroids. Whereas antibody mediated rejection involves glomeruli, peritubular capillaries and need much higher immuno suppressants.
Diagnostic criteria : The presence of Donor Specific Antibodies (DSA) , renal biopsy evidences and C4D immuno florescence staining are suggestive of antibody mediated rejection.
He was treated with IVIG (intra venous immunoglobulins), plasmapharesis and Bortezomib therapy along with hemodialysis. He gradually improved his urine output and his kidney functions recovered remarkably. He is off dialysis support now.

Risk factors for acute antibody mediated rejections:

- Previous transplant recipient
- Multiple blood transfusions
- Multiple pregnancies
- Positive pre transplant cross match
- HLA mismatched or ABO incompatible transplantations
- Inadequate immuno suppressions
- Non compliance to drugs Cadaveric renaltransplantation

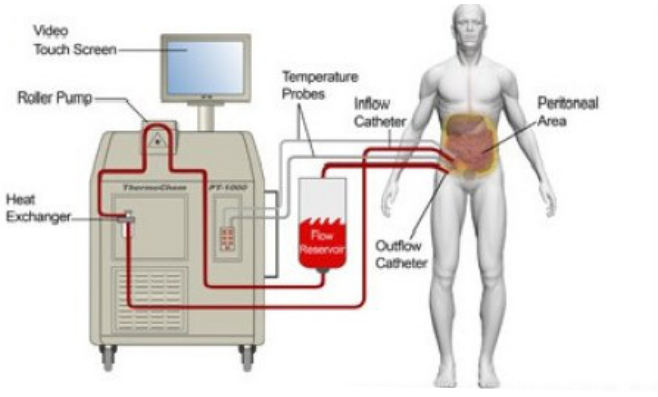


Histologic expressions of acute antibody-mediated rejection (AMR). (A) Margination of neutrophils in peritubular capillaries. (B) Arterial fibrinoid necrosis. (C) Thrombotic microangiopathy. The glomerulus contains intracapillary fibrin thrombi and also shows red blood cell stasis, suggesting poor perfusion. (D) Acute tubular injury. The proximal tubules appear dilated with flattening of the epithelium.



Possible treatment options for Antibody mediated rejection

Our patient responded to plasmapharesis, Bortezomib and IVIG. Renal transplantation involves close monitoring of the kidney functions, and graft dysfunction could happen at anytime. This has multiple causes and that needs proper identification and appropriate management.



Hyperthermia intraperitoneal chemotherapy

ANAESTHETIC MANAGEMENT IN HYPERTHERMIC INTRAPERITONEAL CHEMOTHERAPY (HIPEC)

HIPEC
Hyperthermia intraperitoneal chemotherapy is a highly concentrated, heated chemotherapy treatment that is delivered directly to the abdomen during surgery.

Indication : Advanced surface spread of cancer only within the abdomen.

How is it done : following cyto-reductive surgery heated sterilized chemotherapy solution is delivered at 41-42 degrees Celsius to the abdomen for approx. one and half hours to penetrate and destroy remaining cancer cells.

Case Discussion
A 54 year old female with no comorbidities .TAH with BSO done for granulosa cell tumour 9 years back now came with recurrence of tumour with peritoneal metastasis. Pre-operative evaluation done and blood products were reserved.

Anaesthesia and monitoring:
- Thoracic epidural + general anaesthesia.
- Advanced hemodynamic monitoring- PiCCO (pulse contour cardiac output monitoring)
- Temperature monitoring
- End tidal carbon dioxide level monitoring.

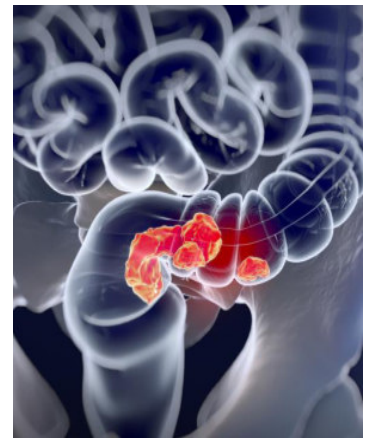
Anaesthetic challenges
End organ perfusion -Fluid management, adequate urine output (1ml/kg/hour), MAP at 70mmHg

Hemodynamic stability – cardiac output (global end diastolic volume); extra vascular lung water index) were monitored for goal directed management of fluids and pressors.

Arterial blood gas analysis: to monitor signs of tissue hypo perfusion (decrease in pH, base excess, serum lactate levels).

ANAESTHESIA MANAGEMENT
Fluidmanagement consisted of both crystalloids (ringer lactate, plasmalyte-A) and colloids. Maintaining renal function by avoiding hypovolemia, hypotension and nephrotoxic drugs hemodynamic optimization by maintaining cardiac output, tissue perfusion and oxygenation.Throughout the procedure urine output was maintained at 40-50 ml/hour. Blood loss is estimated and replaced with packed cells. Besides blood loss patient when exposed to extreme changes in body temperature both hypo and hyperthermia, suffer from metabolic acidosis, calcium and magnesium depletion. Before HIPEC patient was pre-medicated with antiemetics, warming devices were switched off and hemotherm set at temperature 18 degree Celsius. Measures were taken to maintain body temperature less than 37 degree Celsius by using cold saline, ice packs. Infusion of cisplatin 140mg given at temperature of 41-42 degree Celsius over one and half hours. The maximum temperature while HIPEC was 36.7 degree Celsius. Significant hypercarbia was present and managed with hyperventilation. Serial arterial blood gases were done.Serum lactate went up to 8mmol/l during HIPECmanaged with aggressive fluid therapy and acidosis corrected to baseline with bicarbonate infusion and calcium gluconate.
Post 600 minutes of anaesthesia time patient was electively ventilated for 3 hours and warmed weaned and extubated in intensive care unit.

Post-operative pain managed with epidural analgesia and fentanyl infusion.



Hyperthermia intraperitoneal chemotherapy

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RARE BUT A SIZEABLE CAUSE FOR COMA

Dr. Bhuvaneshwari Rajendran
Sr. Neurology and Neurophysiology

Myxedema coma is a rare medical emergency which is a state of severe hypothyroidism that is characterized by decreased mental status, hypothermia, bradycardia and symptoms related to slowing of multiple systems. It has a very high mortality rate of 25 to 60% even with best treatment. We report two cases of hypothyroidism who presented to our hospital with altered sensorium. Although both patients were initially evaluated for other problems, thyroid function tests were impaired with high thyroid stimulating hormone (TSH) and very low T3, T4.
A definitive diagnosis of myxedema coma was made and patients were treated with high thyroxine supplementation along with steroids, following which they improved dramatically.
Case one : 55 year old male with background history of diabetes, dilated cardiomyopathy with poor LV dysfunction and Atrial fibrillation presented with altered sensorium. There was history of RTA in 2009 with head injury and subdural hematoma (SDH) requiring burr-hole evacuation. He was also diagnosed to have hepatitis C related chronic liver disease with portal hypertension in 2014.
Family gave a history of recent hospital stay for giddiness managed with IV fluids. On arrival, he was in deep stuporous state, with GCS of 7/15, pupils-1.5 mm and reacting to light. Vitals were stable. In view of the chronic liver disease he was initially suspected to have hepatic encephalopathy and his serum Ammonia was high with history of constipation. However, his Liver function studies including coagulation profile were essentially normal. And on adding laxatives to reduce his serum ammonia did not improve GCS.
So further evaluation for other causes of drowsiness like infection and other metabolic conditions was commenced. CT brain imaging only revealed age related changes and post op changes of old SDH. EEG suggested widespread slowing with superimposed triphasic waves suggesting encephalopathy mostly of metabolic origin.
Detailed history from family revealed that he was diagnosed with hypothyroidism in 2015 but did not get treated. He had increased sleep, lethargy, tiredness, dryness of skin for the past 3 months, gained weight. Recently, he was having difficulty in understanding things and registering new events. TSH done was 117.83 U/ml (normal level-0.3 – 5.0 U/ml), with total T3-52.5 (normal range- 80-200ng/dl), total T4-1.8 (normal range- 4.6-12ng/dl). He was treated with Eltroxin 300 micrograms. Gradually there was marked improvement in his mental status.
Case two : 74 year old gentleman with history, of hypertension, Parkinsonism, chronic CVA with residual left sided weakness and SDH in 2016 post fall, which was treated conservatively. He is also a known case of Hypothyroidism, on treatment with tablet Eltroxin 100 micrograms long term. He has been less active, more bed bound since 1 year, dependent for all daily activities. He also had history of recurrent hyponatremia.He presented with history of altered sensorium of 10 days duration., GCS of 7-8/15 (E1-2M5V1)and accelerated hypertension (BP – 200/90 mm/Hg), respiratory rate of 30/min and temperature was normal. ECG showed sinus brady cardia (HR – 44/ min). Family gave the history of lethargy, for which they discon tinued tablet Eltroxin as they assumed that lethargy is due to thyroxine supplements. CT brain did not show any new insult. ECHO showed adequate LV function, no effusion. In view of the history of hypothyroidism and hyponatremia, laboratory investigations such as electrolytes and thyroid function tests were done. As suspected patient had low sodium of 116 mmol/L and TSH was abnormally high at 205 IU/ml. T3 – 2.6 ng/dl (reference range: 80 - 200) T4 – 1.30 g/dl (reference range: 5.1 – 14.1). A definitive diagnosis of Myxedema coma was made. In this patient, Myxedema coma was precipitated by withdrawal of thyroid supplements. He had clinical features such as altered sensorium, bradycardia, constipation & abdominal distention, drastic weight gain, hyponatremia, pleural effusion which were indicative of severe hypothyroidism.
Discussion : These cases illustrate the importance of doing thyroid function tests in the assessment of altered sensorium in patients with history of hypothyroidism. It is important to rule out myxedema coma when a patient has unexplained neurological or psychiatric features as it is a medical emergency with high mortality rates. Myxedema refers to deposition of mucopolys accharides in the dermis, resulting in thickening of the skin. Myxedema coma refers to severe hypothyroidism with physiological decompensation and occurs in patients with long-standing, undiagnosed hypothyroidism as seen in our patient. It is usually precipitated by infection, cerebrovascular disease, heart failure, trauma, or drugs like Lithium or Amiodarone. It can also be the presentation of autoimmune thyroiditis . [1, 2, 3]
Patients of myxedema coma usually present with mental confusion, disorientation, depression, hallucinations , poor memory, hypothermia, dry skin, weight gain, decreased deep tendon reflexes. [1, 2, 3, 4] There is a high mortality incidence and thus these patients require ICU care with continuous cardiac monitoring as these patients are prone to arrythmias.Cardiac complications include reduced effect of beta-adrenergic receptors, increased cate cholamines, and increased syst emic vascular resistance, leading to diastolic hypertension and a narrowed pulse pressure. Dec rease in plasma volume and capillary permeability can lead to fluid accumulation in tissue spaces and pericardial effusion. These patients also show reduced central respiratory drive leading to hypoventilation. Another physiological parameter noted in these cases is hyponatremia due to increased effect of anti-diuretic hormone and impaired water excretion. Myxedema coma is associated with a higher risk of bleeding caused by coagulopathy related to an acquired von Willebrand syndrome (type 1) and decrease in V, VII, VIII, IX, and X clotting factors.Myxedema coma carries high mortality rates from 25 to 60% and death is usually due to respiratory failure, sepsis or gastrointestinal bleeding. So high index of suspicion is required and once established immediate treatment is required to save lives. Therapy includes immediate thyroid hormone replacement along with glucocorticoids and other supportive measures. [5, 6] Thyroid hormonsupplementation can be given either oral or intravenous and there is no real consensus about the ideal route of therapy.
As the gastrointestinal absorption is poor in these patients, they are usually treated with a loading dose of 300-600 micrograms of levothyroxine (T4), followed by a daily dose of 50-100 micrograms intravenously. Also there is a debate about whether T4 or T3 supplementation is better. There is data that T3 is quicker to act and is the active component of thyroid hormone. T3 therapy is given as bolus of 5-20 micrograms intravenously and to be continued at a dosage of 2.5-10 micrograms every 8 hours depending on the patient's age and coexistent cardiac riskfactors. [5, 6, 7] Rapid correction precipitates arrhythmias or MI. Thus the treatment should be under constant monitoring.
Our patient was treated with high dose of oral thyroxine 300 mcg for the first 3 days followed by 100 to 200 mcg with regular monitoring of the T4, T3 and TSH levels. This regime was recommended byendocrinologist. Hydrocortisone 100 mg every 8 hours was administered initially till patient started to show some clinical improvement. There was gradual improvement in the GCS and patient became more and more alert. Both patients were discharged with regular high dose thyroxine and will be followed up regularly.

CONTINUOUS RENAL REPLACEMENT THERAPY

Dr.Balaji Kirushnan
Consultant Nephrologist

Acute kidney injury is very common in the intensive care unit (>50%). More than 60% of all the acute kidney injury patients require dialysis and acute kidney injury is an independent risk factor for overall mortality. The various modes of dialysis in the ICU are intermittent hemodialysis, continuous renal replacement therapy, peritoneal dialysis and hybrid therapies. CRRT is defined as any extracorporeal blood purification therapy intended to substitute for impaired renal function over an extended period of time and applied for or aimed at being applied for 24 hours /day.

The various principles used in CRRT are

1 SCUF – Slow Continuous Ultrafiltration

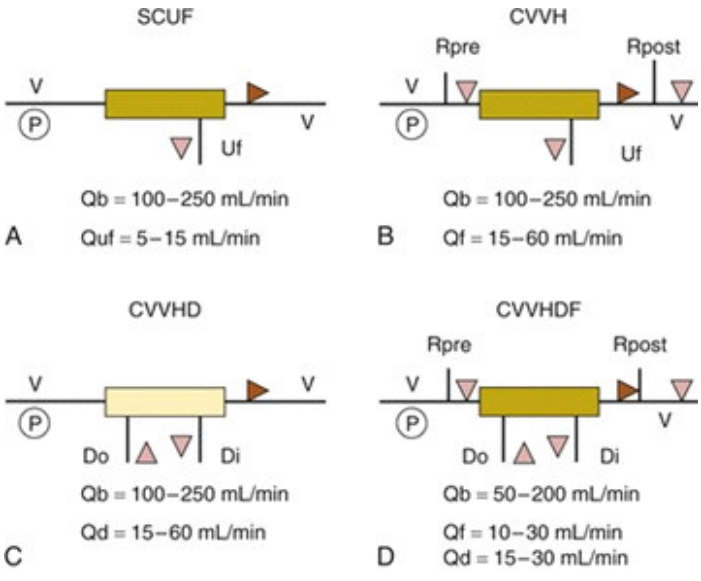
2. CVVHD – Continuous Veno-Venous Hemodialysis

3. CVVHF – Continous Veno-Venous Hemofiltration

4. CVVHDF – Continous Veno-Venous Hemodialfiltration

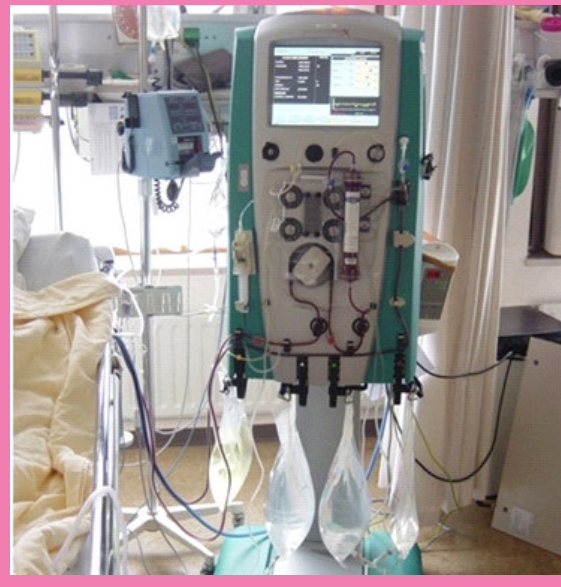
There has been ongoing debate of initiation of intermittent dialysis versus continuous hemodialysis in the ICU. Randomized controlled trials and Meta analysis have demonstrated no difference when either of them are used. However the use of CRRT has proven beneficial in certain sub populations. The advantages of CRRT are

- Better preservation of cardiovascular function and maintenance of hemodynamic stability
- Prevents the surge in intra cranial pressure associated with intermittent therapies
- Effective in clearance of middle molecules
- Useful in removal of immunomodulatory subs tances in sepsis like endotoxin, interleukin-1, etc



- Permits protein rich nutritional support with a neutral nitrogen balance preventing protein
- Various poisoning
- Can be performed at bedside.
- Continuous mode, can be extended for days till the patient becomes stable.

It is useful in hemodynamically unstable patients, patients with acute liver failure with renal failure, acute brain injury patients and post operative cardiac patients.
The disadvantages of CRRT are that it requires continuous anticoagulation, filter change due to clotting, dedicated staff to analyze alarms and change fluid bags, patient immobility and lack of transfer to imaging facilities, Hypophosphatemia and loss of trace elements.
Early CRRT in the intensive care unit has shown mortality benefits when to compared to conventional indications of late start dialysis.
Each CRRT is a short term therapy lasting for 3-5 days depending on the clinical condition of the patient and duration of the filter without clotting.



CRRT machine – at Kauvery Hospital

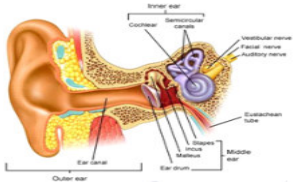
DIZZY AND GIDDINESS-A MERRY-GO-ROUND EFFECT- IS IT

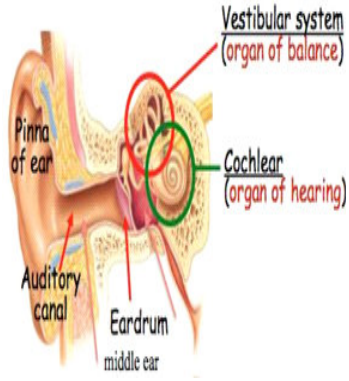
Dr.Sundari
Sr.ENT consultant

Dizziness-Giddiness-lightheadedness-Vertigo is not a disease but rather a symptom that can be result from a huge variety of underlying disorders of brain and inner-ear in some cases, or no disorder at all. Vertigo is a sense of rotation, rocking, or the world spinning, experienced even when someone is perfectly still. It can be experienced as a spinning sensation similar to riding a merry-go-round, sometime feeling of swaying to one side or being on a small boat in a stormy sea. Movement of the head or body, like rolling over in bed, can escalate or worsen the symptoms. The symptoms are different from light headedness or a sense of fainting. Many people experience associated nausea or vomiting.

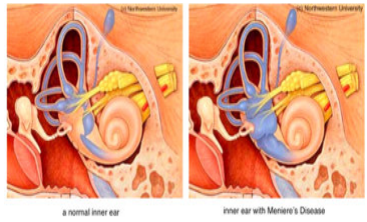
Symptoms: Vertigo can be a symptom of other conditions, and it can also have its own set of related symptoms. These include

1) Balance problems and general feeling of faintness, lightheadedness sense of rotational dizziness with motion sickness
2) Nausea and vomiting
3) Tinnitus (abnormal ringing sound in ears
4) Feeling of fullness in the ear
5) Headache





Causes:
There are a number of different causes of vertigo. Vertigo can be defined based upon whether the cause is peripheral or central. Central causes of vertigo arise in the brain or spinal cord while peripheral vertigo is due to the result of an imbalance in the inner ear.



Vertigo can also be caused by or related to:

Migraine headaches - a severe headache that's usually felt as a throbbing pain at the front or on one side of your head, which is especially common in younger people

Head injuries or trauma

Ear surgery followed by a peri-lymphatic fistula, a tear in one or both of the membranes separating the middle and inner ear causing leakage of inner ear fluid into the middle ear

Labyrinthitis or Vestibular neuronitis: This is an inflammation of the inner ear labyrinth and vestibular nerve, usually due to a viral infection

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BPPV : This is due to disturbance in the otolith particles of crystals of calcium carbonate within inner ear fluid that touch the sensory hair cells inside the semicircular canals during movement. They stimulate the vestibular nerve to send information to the brain about a person's position

Meniere's disease : A buildup of excess fluid in the inner ear can lead to attacks of vertigo with ringing in the ears and hearing loss

Otosclerosis : a middle ear bone problem that causes hearing loss and tinnitus

Acoustic neuroma : a benign growth on the vestibular nerve that traverses between the inner ear to the brain

Side effects of medication or drug toxicity

Transient ischemic attack or **cerebro vascular stroke**
Transient ischemic attack or cerebro vascular stroke

Diagnosis:

Symptoms : such as hearing loss, tinnitus, nausea, vomiting or fullness in the ear, how often your symptoms occur and how long they last for, whether anything triggers your symptoms or makes them worse, such as moving your head in a particular direction

Hearing tests : an audiometric test for assessing the hearing loss, tinnitus and to assess the inner ear function by vestibular evoked Myo-potential (VEMP) and Oto-Accoustic Emission test

If the doctor observes specific eye movements along with experiences of giddiness, those of nystagmus, this can indicate that the patient has vertigo. This is achieved through a number of tests, including

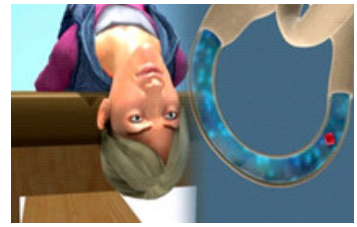
Electronystagmography (ENG): This can electronically record the nystagmus. The patient wears a headset that places electrodes around the eyes. The device measures eye movements

Videonystagmography (VNG): This is a newer technology can provide a video recording of the nystagmus. The person with vertigo puts on a pair of special glasses that contain video cameras, these record horizontal, vertical and torsional eye movements using infrared light. Computer processing can analyze the data collected.

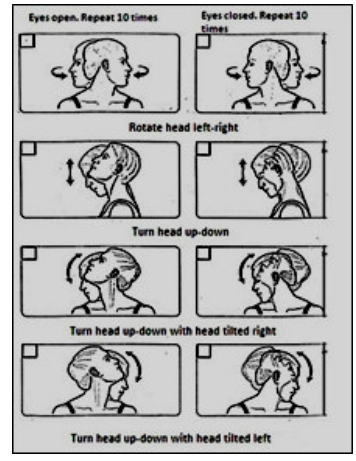
Radio- imaging of MRI/ CT brain may be used to look for the cause of vertigo, such as a TIA, acoustic neuroma (a non-cancerous brain tumour) and cerebellar lesions.

Treatment:
Some cases of vertigo improve over time, without treatment.

However, some people have repeated episodes for many months, or even years, such as those with Ménière's disease.There are specific treatments for some causes of vertigo.A series of simple head movements (known as the Epley manoeuvre) is used to treat BPPV



Medicines, such as prochlor perazine and some antihis tamines can help in the early stages or most cases of vertigo. Many people with vertigo also benefit from vestibular rehabilitation training (VRT), which is a series of exercises for people with dizziness and balance problems.



Pressure pulse treatment, in which a device fitted to the outer ear delivers air pressure pulses to the middle ear, reducing vertigo

Self Care:
Depending on what's causing your vertigo, there may be things you can do yourself to help relieve your symptoms.
Reduce Stress by doing a physical therapy Yoga, can be increasing flexibility and balance.
Eat healthy diet and stay hydrated- simply drink plenty of water (3-4 liters/day).
Do simple vestibular exercises to correct your symptoms
Sleep with prober bed and soft pillows
Get up slowly when getting out of bed and sit on the edge of the bed for a minute or so before standing, Move or Turn your head carefully and slowly during daily activities
Avoid suddenly bending forward to pick up items
Avoid extending your neck – for example, while reaching up to a high shelf
Avoid high salted diet like pickle and salted chips
Avoiding caffeine, chocolate, alcohol, and smoking tobacco

Key Events

Kauvery Hospital
Joint Managing Director Dr. S.Manivannan received the award ,
as excellent service in Tamil Nadu for The Hindu Doyens 2018
from Governor Banwarilal Purohit

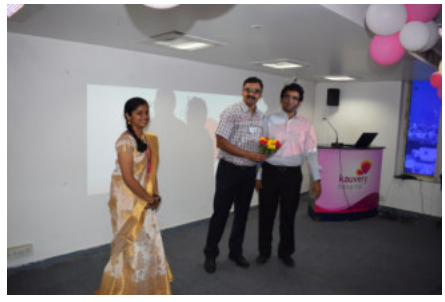
Pride of Tamilnadu Awards 2018

Key Events

Women's Wellness Carnival. @ Kauvery

Back to Basics
State Level Nursing conference @ Savera Hotel

World Kidney Day Celebration @ kauvery Hospital



Self Defence Workshop @ Nageshwara Rao Park.

Kauvery Hospital, Chennai

Welcomes - Our new-family Members

Dr Booma
MBBS., DNB (Med), DNB (Cardiology)
Senior Resident, Department of Cardiology

Dr.Pushkala M.S
MBBS., MD.,PGDID
Consultant, Department of Paediatric

Dr.Manikandan
MBBS., DNB., MCH
Junior Consultant, Department of CTS

Dr.Anantha Subramanian
MBBS., MD (Pul Med)
Senior Resident , Department of Pulmonology

Dr.K.Rajan
MBBS., DMRD., MD (Radio Diagnosis)
Senior Consultant , Department of Radiology

Dr.Priya Kalaimani
MBBS., PGDMCH., PGDHSc
Resident , Department of Geriatrics