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# Editor's Column

It gives us immense pleasure in bringing out the second edition of our newsletter this year.

For past couple of months months we were all busy managing dengue patients which is probably the worse epidemic in recent years. Also this year we faced varied clinical presentation of this disease from mild febrile illness to severe dengue with shock, seizures, hypokalemic paralysis, severe hepatitis and MODS.

Indian Medical Association has released guidelines regarding management of dengue patients which we have included in this edition. As we all know that EDPA is organising it's Annual CME "EDPACON 2015" on 13th December at Hotel Le Meridien, our next release would be newsletter cum souvenir which will also contain abstracts of the talks delivered during the CME apart from routine case reports.

We again request all of you to make active contributions towards our newsletter in the form of interesting articles, case reports, abstract from the journals etc. Our efforts will make the difference.

Signing off with an interesting quote by Paul J Meyer :

"Whatever you vividly imagine, ardently desire, sincerely believe and enthusiastically act upon will come to pass "

**Dr. Pankaj Nand Choudhry**
Editor

**Dr. Naresh Agarwal**
Co-editor

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# Presidential Address

Dear Friends,

This is the second NEWS & VIEWS Bulletin of this year and I must congratulate our Editorial Team especially Dr. Pankaj Nand Choudhary. It's because of his sincere & persistent efforts this second bulletin is on time. I request all the members to contribute articles, case reports etc. for News & Views Bulletin.

We are preparing for our Mega Conference EDPACON 2015 on 13 December, 2015 at Le Meridian. I request all the members to put forth their suggestions & topics to make it successful. Kindly block your date too as it's your conference.

I once again congratulate the Editorial team for bringing out such an informative bulletin.

GOD BLESS AND LONG LIVE EDPA!

**Dr. Rajeev Bansal**
President, EDPA

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# Secretary's Notes

Greetings from a junior to all his seniors!!

The first year of my tenure as the youngest secretary of EDPA has been a roller coaster ride, full of highs and lows.

The new executive had promised to make EDPA a socio-cultural and sports body. And we have succeeded!!!

The EDPA sports committee has been organising monthly cricket matches for our members. Although two cricket matches got rained out, the enthusiasm of our members for sports was heartening.

In addition, three of our members, Dr. Lalit, Dr. Anil Sharma and Dr. S.K. Sharma brought laurels to out association by winning medals at the recently held DMA swimming competition.

We are planning to hold table tennis and badminton matches for our members as well. We are also planning to have the first ever EDPA sports meet soon.

We are now busy in planning for our upcoming EDPACON 2105. The chairman for the conference, Dr. Paras Gangwal is leaving no stone unturned in order to make this EDPACON the best ever.

On the academic front, under the guidance of Dr. Vimal Nakra, Dr. Pankaj Choudhary and Dr. Nitin Sinha, the quality of cases and discussions at our monthly clinical meets has been steadily improving.

Dr. Pankaj Choudhary has also been the guiding force behind the Whatsapp group 'EDPA academics wing'. It is a group where cases and doubts are put up and solved on a daily basis. It is a unique initiative, giving an opportunity to 100 physicians to interact simultaneously, and a big help to all the members in solving difficult cases.

Its been a great year, and, I promise, the next year will be even better!!!

**Dr. Anirudh Lochan**
Secretary, EDPA

# Chairman's Message

Dear Friends,

It gives me immense pleasure on behalf of organising committee of East Delhi Physician's Association to announce our much awaited Annual Scientific Feast EDPACON 2015 which has been scheduled on 13th December, Sunday at Le Meridien Hotel, New Delhi.

We extend our warm welcome, to attend this academic feast themed on practical tips in various aspects of day to day practice, based on zist of consensus guidelines, controversies and dilemmas & expert recommendations based on their own experiences. Also we will have insight into path breaking landmark trials, new drug releases and future of internal medicine.

You will witness luminaries from national scientific forums as well as international speakers.

We are extending our invitation to all physicians practicing internal medicine, for registration, across all parts of Delhi and NCR, from all Govt and Private Institutions, as well as from various scientific affiliate bodies and associations.

We are expecting a gathering of around 400 delegates.

We invite all our medical practitioner colleagues to be part of this scientific fiesta and also invite our corporate and Pharma Colleagues to use this opportunity to showcase their calibre n excellence & products in the field of medicine to the august learned gathering of professionals of high repute and potential.

See you at 'EDPACON 2015".

**Dr. Paras Gangwal**
Chairman
EDPACON 2015

# TAKAYASU'S ARTERITIS PRESENTING WITH STROKE A CASE REPORT

**Dr. Arushi Nautiyal, Dr. N.P. Singh**

Takayasu's arteritis is a chronic, idiopathic inflammatory condition involving the aorta and its major branches. This disorder has been most commonly reported in India, Japan and China. Usual age of presentation is less than 40 years. It is characterized by stenosis of vessles or with more acute inflammation, aneurysm formation. It thus presents most commonly with absent or reduced pulses, bruits, hypertension (1). Most common neurological symptoms are dizziness and visual disturbance (2). Ischemic stroke occurs in <10% patients (1)

Here we describe a case of a young female who presented with stroke and pulselessness

## Case

A 38 years old female presented with complaints of sudden onset weakness of left side of body and slurring of speech since two hours. These symptoms were first experienced upon waking up in the morning. Developed slipping of slippers on left side, was unable to grip objects from left hand. No history of headache, seizures, or fever.

Six months earlier patient had developed palpitations for which she had consulted a local practioner, patient was diagnosed with an aortic regurgitation and started on beta blockers.

On examination, patient was conscious, oriented. Her upper limb pulses were impalpable, blood pressure of 120/80 was measured from lower limb.

There was deviation of angle of mouth towards the right side, power on left side upper and lower limbs was 3/5. She had an early diastolic murmur discernible on auscultation.

On investigation, Patient had a anemia, hemoglobin- 8.2, TLC-10.6, raised ESR- 60.

**NCCT head** showed right MCA thrombus with evidence of infarct in right MCA territory. T assess the intracranial vessels and extent of infarct, **MRI brain with MR Angiography of neck and intracranial vessels was done which revealed** a large area of restricted diffusion in perisylvian aspect of right frontal lobe- s/o acute infarct, complete non visualization of bilateral common carotids and most of cervical extent of bilateral internal carotid likely representing arteritis with unremarkable bilateral vertebrals supplying anterior circulation via circle of willis

Lipid profile was within normal limits, serum homocysteine levels normal. ANA, dsDNA, ACLA, APLA- negative.

Echocardiography showed concentric LVH and mild to moderate AR, global LVEF: 45-50%.

CT aortogram was done to assess the aorta and its branches, it revealed significant diffuse mural thickening in acending, arch and descending thoracic and abdominal aorta and extending into various branches of aorta. Features suggestive of aortoarteritis likely Takayasu's disease.

Patient was started on antiplatelets, steroids and oral methotrexate. Patient has been on regular follow up, weakness has improved. Still complains of difficulty with fine movements of left hand.

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## Discussion

Takayasu's arteritis is a rare, chronic inflammatory disease involving the aorta and its branches.

First described by a Japanese ophthalmologist, who discovered characteristic AV anastamoses in the fundus of a young female (3).

Its precise etiology has not been determined as yet. Data gathered so far describes the possibility of the disorder developing in a genetically predisposed indivual exposed to an unknown antigen which triggers an autoimmune process resulting in the inflammatory state. Studies conducted by Mehra et al in Indian patients suggest a strong association of the disease with HLA-B5 as well as its two serological subtypes, B51 and B52 (4)

It is characterized by stenotic as well as aneursymal lesions. According to Moriwaki et al. in Japanese patients, mainly the aortic arch and its branches are affected, whereas in Indians the abdominal aorta and renal arteries are mainly involved (5)

It most characteristically presents with absent pulses, bruits, retinopathy, hypertension (secondary to renal artery stenosis), aortic regurgitation, congestive cardiac failure (hypertension, AR), neurological manifestations- usually dizziness, seizures. Stroke may occur in 10 % patients(1)

Our patient was a young female who presented with sudden onset weakness of left side of body and slurring of speech. On examination she had left sided hemiparesis along with impalpable upper limb pulses which suggested the daignosis. Investigations revealed a right MCA infarct, mural thickening involving aorta and its branches, along with aortic regurgitation. These features were suggestive of Takayasu's arteritis grade 2B, based on Ishikawa's clinical classification (1).

The disorder often presents with non specific symptoms such as fever, fatigue, arthralgia before symptoms of organ ischemia develop. Thus a delay in diagnosis may occur. If our patient had been identified at the earlier presentation of palpitations and started on immunosuppressive therapy, the event of stroke could have been prevented.

The mainstay of treatment is immunosuppresion with steroids, and the addition of methotrexate in cases of steroid dependence, frequent relapses. Surgery and percutaneous angioplasty maybe considered in cases of vessel narrowing.

Determining disease activity is a difficult task, histopathological evidence of vascular inflammation is the gold standard but difficult to obtain. On a follow up basis usually constitutional symptoms and raised acute phase reactants- ESR, CRP are considered as markers of disease activity, however they have not always correlated with histopathological evidence of activity according to several studies and are therefore not reliable for assessment. Studies have shown a correlation with IL-6 and imaging techniques such as contrast enhencde MRI, ultrasound of extracranial vessels, intravascular ultrasound of the aorta(4).

## Conclusion

Takayasu's arteritis is a chronic condition which can present with constitutional symptoms of fever, malaise or even with catastrophic events such as stroke, congestive cardiac failure. Therefore it must be identified at the earliest and immunosuppressive therapy should be started to prevent such events.

# AN INTERESTING CASE OF PERIPHERAL NEUROPATHY

**Dr. Abhinav Mohabey, Dr. Pankaj Nand Choudhary**

A 34 year old male, resident of Tripura (North-East), businessman by occupation, consulted in OPD with complaints of progressive weakness of both lower and upper limbs since 3 months, tingling sensation in both lower and upper limbs since 3 months, intermittent abdominal pain & loose stools for 2 weeks. Weakness was gradual in onset ,bilaterally symmetrical, initially involve distal muscle of both lower limbs. In 15 days weakness progress to involve proximal muscle of both lower and upper limbs. Weakness slowly progress to involve distal muscle of both upper limbs, in a month. There was no history of truncal, neck muscle weakness, no history of stiffness, losening of limbs & flexor spasm. Along with that he had tingling sensation in distal part of both lower followed by upper limbs, which slowly progress proximally in 3 months. He felt plaster cast like sensation in both lower limbs suggestive of posterior column involvement. He denies history of burning sensation. No bowel & bladder involvement (prior to two weeks), no history of vaccination, URI prior to onset of weakness, no history of waxing & waning of weakness. There was weight loss of around 10-12 kg in 4-6 months. Abdominal pain was diffuse, non colicky associated with 4-5 loose stools, watery in nature, not associated with nausea or vomiting. Patient took few courses of antibiotics. He attributed this to frequent travel. Patient was chronic alcoholic, use to take 280 gm/day since 5 years. There was no history of similar illness in past & no co-morbidities. On examination patient was concious, oriented, afebrile, hadtachycardia, face & palm were hyper pigmented. On systemic examination, per abdomen was soft, mild diffuse tenderness with no organomegaly. On nervous system examination, HMF was normal, all cranial nerves were intact, fasciculations were seen in biceps & thigh muscles, there was wasting of the nar/hypothenar muscles, thigh muscles, both UL & LL were hypotonic, power in proximal muscles of UL & LL were +3, distal muscles of UL & LL were + 4, all reflexes were diminished. On sensory examination deep sensation i.e. vibration & joint sense were impaired. Diagnosis of CIDP was made with HIV as probable etiology. He was investigated to rule out other etiologies of CIDP.

Haemogram, LFT, KFT, RBS, HbA1c were within normal limit. Viral markers i.e. Hbsag, HCV, HIV were non reactive. Ultrasound whole abdomen was normal, urine for BJ protein & porphobilinogen were negative. Serum for protein electrophoresis doesn't showed M spike.MRI brain with screening of whole spine showed mild disc protrusion at the level of C3/C4,NCV showed abnormal study suggestive of mixed, motor-sensory peripheral polyneuropathy involving upper and lower limbs. Idiopathic CIDP was considered after investigation. In view of weight loss, tachycardia, hyperpigmentation, fasciculation thyroid profile was send which showed **FT4-5.88 ng/dl** (0.8–2.0), **FT3-14.32 pg/ml** (1.4–4.2)**, TSH-0.06micIU/ml** (0.39–6.16), so final diagnosis of thyrotoxic neuropathy was made .

## Discussion

Thyroid hormone plays an important role in stimulatingthe development and differentiation of the neuromuscular junction and brain. Patients of thyrotoxicosis experience a wide range of muscular disorders: proximal myopathy, exophthalmic ophthalmoplegia, thyrotoxic periodic paralysis, myasthenia gravis, rhabdomyolysis. The neurological manifestations are: neuropsychiatric syndrome (anxiety, emotional lability, psychosis, depression, delirium,

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seizures, encephalopathy); movement disorder (fine tremor, choreoathetosis); thyrotoxic neuropathy. Thyrotoxic myopathy usually appears after 1 - 3 months of thyrotoxicosis. It is more common in men, and elderly patients; and can be the presenting feature in this group of patients. The common presenting symptoms are: muscle weakness, wasting of pelvic girdle and shoulder muscles, fatigue. Muscle pain is uncommon. In a study of thyrotoxic patients, 67% had weakness, and 81% had decreased strength in at least one group of muscles4Sphincter functions are often not affected. Although less common, bulbar, respiratory, and even oesophageal muscles may be involved presenting as dysphagia, dysphonia, breathlessness, and aspiration. Bulbar involvement is found in only 21% of patients with thyrotoxic myopathy5 . The extent and severity of weakness is related to the duration of hyperthyroidism, and not to the biochemical severity. Muscular atrophy is of a lesser severity than the weakness. Deep tendon reflexes can be normal or brisk. Hyperthyroidisminduced myopathy is secondary to: negative nitrogenbalance secondary to catabolic effect of thyroidhormone; increased mitochondrial respiration, accelerated protein degradation and lipid oxidation; and thyroid hormone directly affecting transcription of many genes related to myocytes. Typically, creatine kinase is normal, and muscle biopsy and EMG show non-specific changes. The prognosis of thyrotoxic myopathy is good; weakness resolves rapidly on achieving euthyroid state 5,6. Neuropathy is an uncommon manifestation of thyrotoxicosis. Acute neuropathy associated with paraplegia has been reported, and is known as Basedow's paraplegia 7 . However, studies have shown that peripheral neuropathy due to thyrotoxicosis is not so rare. In studies done on hyperthyroid subjects by Duyff et al4 , Ludin et al8 and Sozay et al9 , neuropathy was seen on electrophysiological testing in 19%, 65.5%, and 35.5% of patients respectively. Most of the patients with neuropathy were asymptomatic. Sozay et al and Ludin et al concluded that electrophysiological studies are useful in diagnosing asymptomatic polyneuropathy in these groups of patients. Possible pathogenic mechanisms are: direct effect of thyroid hormone, immune mediated; hypermetabolic state depleting the nerves of essential substances. Motoneuron soma is the site of the primary lesion ultimately affecting peripheral nerve, dorsal root ganglion, and anterior horn cell10. Electrophysiological study reveals a mixed axonal and demyelinating sensorimotor neuropathy. Our patient had sub-clinical entrapment neuropathy, both in upper and lower limbs. Carpal tunnel syndrome (CTS) has been reported in the past in thyrotoxic patients. Beard et al reported three cases of CTS associated with Graves disease, and all reverted on achieving euthyroid state11 . They proposed a causal relation between the two. Roqueret al in their prospective study, found CTS in 5% of hyperthyroid patients in the initial survey12. They followedthe patients for two years, during which four more patients developed CTS. The condition remitted on treatment, and they concluded that CTS should be considered another peripheral neurological manifestation associated with hyperthyroidism. Mononeuropathy, mononeuritismultiplex, and bilateral peroneal nerve palsy has also been reported13, 14, 15.

## Conclusion

1. The patients of hyperthyroidism may present with neuropathy even in mild cases of neuropathy
2. Thyroid function tests can be helpful in the diagnosis of this treatable neuropathy and should be included in the routine work up

# A CASE OF POST-TRAUMATIC HYPONATREMIA

**Dr. Anando Sengupta, Dr. Neeru P. Aggarwal**

## Introduction

Hyponatremia, defined as a serum sodium concentration <135 mmol/l, is the most common disorder of body fluid and electrolyte balance. It can lead to a wide spectrum of clinical symptoms, from subtle to severe or even life threatening. In a patient of head injury presenting with hyponatremia, the causes can range from extrarenal fluid losses to cerebral salt wasting (CSW) syndrome or SIADH (syndrome of inappropriate ADH secretion). Here we present a case of a middle aged male who presented with symptomatic hyponatremia due to cerebral salt wasting syndrome secondary to head injury.

## Case

A 35 year old male with no co-morbidities, presented to the emergency department with complaints of drowsiness, recurrent vomiting and hiccups for past 1 day. Attendants gave history of Road traffic accident with head injury (with right ear bleed) 1 week back for which patient was admitted in outside hospital and NCCT head done which was normal. There was no history of headache, fever, seizure like activity or any use of medication. On examination patient was drowsy, confused and dehydrated but was hemodynamically stable with unremarkable examination of other systems. On investigation patient was found to have sodium level of 118 mEq/l with CBC, kidney function tests and LFT within normal limits. Urine spot sodium was 138.2 mEq/l, urine osmolarity- 808 mosm/kg(normal 300-900), Serum osmolarity- 258 mosm/kg(normal 275–295). Patient was started on hypertonic saline, iv fluids and supportive treatment. Patient continued to be symptomatic and had persistent hyponatremia for next two days inspite of treatment. Diagnosis of cerebral salt wasting syndrome was made in view of hypovolemic hyponatremia, history of head injury and renal sodium loss. Patient was started on oral Fludrocortisone (0.1 mg OD). Patient's sodium levels recovered within two days and he was discharged.

## Discussion

The causes of hyponatremia with hypovolemia are many. Increased renal sodium losses(urinary sodium >20 mmol/l) can be seen in case of diuretic excess, mineralocorticoid deficiency, salt losing nephritis, cerebral salt wasting or osmotic dieresis. Extrarenal losses (urinary sodium <20 mmol/l) can be seen in vomiting, diarrhea, third space sequestration or burns. In a patient with head injury if there is renal sodium loss the differential diagnosis include CSW syndrome and SIADH which can be differentiated on the basis of the hydration status (CSW patient will be hypovolemic and SIADH will be euvolemic). Cerebral salt wasting syndrome is the development of extracellular volume depletion due to a renal sodium transport abnormality in patients with intracranial disease and normal adrenal and thyroid function.

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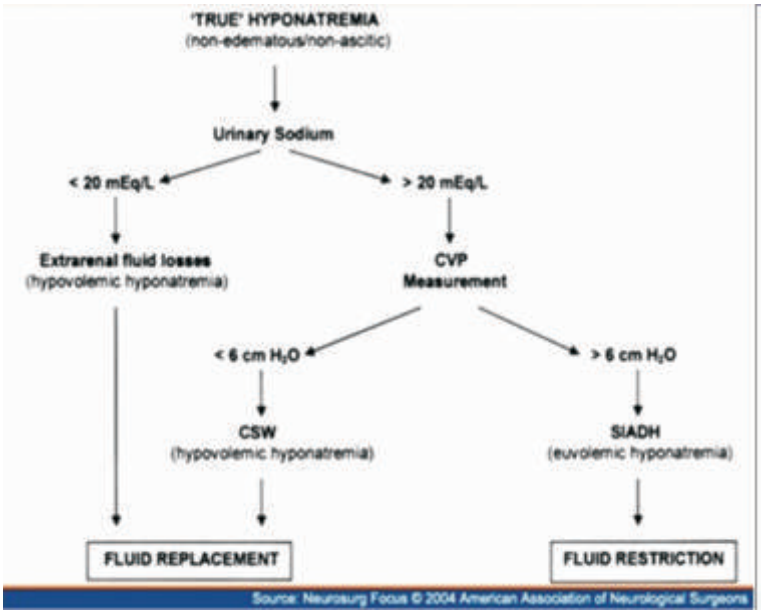
## Approach to a patient with head injury and hyponatremia




The pathogenesis of CSW syndrome includes increase in natriuretic peptides and decreased sympathetic outflow leading to hypovolemic hyponatremia. It occurs in the setting of acute CNS disease - Head injury, brain tumor, intracranial surgery, stroke, intracerebral hemorrhage, tuberculous meningitis, craniosynostosis repair etc. It develops in the first week following brain insult and spontaneously resolves in 2-4 weeks.

| Biochemical marker | SIADH | CSWS |
|---|---|---|
| Intravascular volume status | Normal to high | Low |
| Serum sodium | Low | Low |
| Urinary sodium level | High | Very high |
| Vasopressin level | High | Low |
| Urine output | Normal or low | High |
| Serum uric acid level | Low | Low |
| Initial fractional excretion of urate | High | High |
| Fractional excretion of urate after correction of hyponatremia | Normal | High |
| Urinary osmolality | High | High |
| Serum osmolality | Low | Low |
| Blood urea nitrogen/creatinine level | Low to normal | High |
| Serum potassium level | Normal | Normal to high |
| Central venous pressure | Normal to high | Low |
| Pulmonary capillary wedge pressure | Normal to high | Low |
| Brain natriuretic peptide level | Normal | High |
| Treatment | Water restriction | Fluids and/or mineralocorticoid |

CSWS = Cerebral salt wasting syndrome; SIADH = Syndrome of inappropriate antidiuretic hormone

Treatment includes correction of intravascular volume depletion and hyponatremia, as well as replacement of ongoing urinary sodium loss including hypertonic saline solutions. There is a favourable response to Fludrocortisone (0.05-0.1 mg/day PO in single daily dose or divided q12hr) in combination with sodium chloride supplementation.

# DENGUE AND MALARIA COINFECTION

**Dr. Danish Kathuria, Dr. Pankaj Nand Choudhary**

## Introduction

Even though both malaria and dengue share many epidemiological features, very few cases of coinfection have been reported. Here we report a case of middle aged female with concurrent malaria and dengue.

## Case description

A 42 year old housewife presented in the ER with complains of fever since 5 days and altered sensorium from last 6 hours. Her fever was high grade, associated with chills and rigors, was relieved only with medication, there was no diurnal variation and was accompanied by severe body aches. A few hours prior to her admission, she started talking irrelevantly, became drowsy on the way and by the time she reached hospital, her level of consciousness further deteriorated. She did not have any localizing symptom suggestive of respiratory tract infection, UTI or GI infection. There was no history of headache, vomiting, neck stiffness or any motor weakness or numbness. There was no history of any rash, joint pains, ill-contacts or recent travel. There was no history of any head trauma. Her past medical and surgical histories were unremarkable. There was no history of any substance abuse. On general physical examination, she was stuporous with a low GCS score of 7/15(E1 V2 M 4). Icterus and pallor were present. She had tachycardia (Pulse- 104/min), febrile (Temp: 100 F) and BP was 100/60 mm Hg. She was moving her all four limbs on painful stimuli and Kernig's sign was equivocal. Rest of the CNS and systemic examination was noncontributory. We admitted her in ICU and treated her on the lines of meningitis. As her outside reports showed positive test for P. vivax on peripheral smear, cerebral malaria was also considered and antimalarials were also added to the treatment regimen. Her initial investigations on day 1 are showed leukocytosis (TLC- 22.18, predominantly neutrophilic), thrombocytopenia (50,000/uL), grossly deranged renal and liver function tests (S. Urea-152, S. Creatinine: 4.1, S. Bilirubin:3.1, AST:13374 IU/L, ALT:5022 IU/L. Dengue serology (both IgG and IgM) and NS1 antigen returned positive. Patient was admitted in ICU and started on IV antibiotics (Ceftriaxone), IV antimalarials (Artesunate), oral doxycycline, N-Acetyl Cysteine infusion, ursodeoxycholic acid, oxygen supplementation and supportive care. CT Head showed features s/o cerebral edema. So IV steroids were added to the treatment. CSF analysis was unremarkable. MRI Brain showed essentially a normal study. After nephrology consultation patient was initiated on hemodialysis. Additional tests (serum ammonia, thyroid profile) done to rule out other causes of encephalopathy were WNL. CPK levels were high (1699). Abdominal ultrasound showed hepatosplenomegaly and polyserositis. Rest of the work up for enteric fever, viral hepatitis, leptospirosis and rickettsial diseases was negative. Gradually

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patient's fever subsided, kidney function and urine output improved, S. Bilirubin and hepatic enzymes started down trending and patient's sensorium improved and eventually she became fully conscious and oriented. Our final impression was Vivax malaria and Dengue coinfection with hepatorenal dysfunction with a possibility of accompanying myositis and rhabdomyolysis (leading to AKI).

## Discussion

Dengue fever and malaria are the most common arthropod-borne diseases in humans. They represent major public health problems. Reports of malaria and dengue dual infection are scarce. First case reported in 2005. There are various hypotheses explaining why this coinfection is rare. Firstly, the mosquito vector for each infection has different habitat. Secondly, if one turns out to be positive, physician may not order the tests for the other one. Moreover, co-infection has to be by chance, so the mathematical probability of acquiring both is quite less. Jaundice (in dengue patients) and spontaneous bleeding (in malaria patients) should raise the suspicion of co-infection. According to a retrospective matched pair study conducted at French Guiana, dengue and malaria coinfection tends to be more severe than single infections notably for hematologic abnormalities, such as thrombocytopenia and anemia. There is increased severity in terms of higher risk of bleeding and hepatic complication.

Since symptomology of dengue and malaria are very similar, all clinicians treating patients in or returning from endemic areas should systematically order investigations for both diagnoses, even if one or the other is positive. Failure to recognize malaria or dengue coinfection would delay the initiation of proper therapy and result in increased morbidity and even mortality.

**Medicine really matured me as a person because, as a physician,
you're obviously dealing with life and death issues,
issues much more serious than what we're talking about in entertainment.
You can't get more serious than life and death. And if you can
handle that, you can handle anything**

**- Ken Jeong**

# UNILATERAL PULMONARY HYPOPLASIA – A CASE REPORT

**Dr. K.K. Pandey**

## Abstract

A case of right sided pulmonary hypoplasia is described. A 20 years male was admitted in our department with complaints of recurrent chest infections since childhood. Clinical examination revealed smaller right hemithorax. Serial chest Xrays showed nonprogressive lesions over several years. Bronchoscopy and CT scan thorax confi rmed the diagnosis of right sided pulmonary hypoplasia. It was not associated with any other developmental anomaly

Lung India 2007; 24 : 69-71

## Key words

Hypoplasia,3 Bronchoscopy, CT scan thorax.

Hypoplasia of lung is a rare congenital anomaly in which gross morphology of the lung is essentially unremarkable but in which there is decrease in number or size of airways, vessels and alveoli. Under development of alveolar tissue results in small fi brotic and non functioning lung. Bronchiectatic changes have also been reported in the hypoplastic lung1 . This condition is frequently associated with other congenital anomalies. Common developmental anomalies associated with pulmonary hypoplasia involve urinary system2 , diaphragm3 , cardiovascular system (Tetralogy of Fallot)4 , central nervous system (anencephaly and hydroencephaly)5 , as also musculoskeletal anomalies of thoracic cage, Klippel Feil syndrome and Down syndrome6 .

## Case Report

A 20 years male was admitted in Department of Pulmonary Medicine, King George's Medical University, Lucknow with chief complaints of cough and expectoration, off and on fever, breathlessness increased on exertion since childhood. Patient also complained of hemoptysis several episodes, varying in amount from streaking of blood in sputum to 200-300 ml/episode since six years. There was history of dull aching pain over right side of chest for four years and loss of appetite for two months. General physical examination was non-contributory. Examination of respiratory system revealed smaller right hemithorax. Trachea and heart were shifted to right side. Movements were diminished and percussion note was dull over the right hemithorax. On auscultation, air entry was very poor on right side with diffuse crepitations. Rest of systems were within normal limits.

Routine haematological investigations (Bloodcounts, Liver function tests, renal function tests) were within normal limits. Sputum smear for Acid Fast Bacilli was negative on direct smear examination and later on culture. Chest roentgenogram showed a right sided massive homogeneous opacity, shifting of the mediastinum, and chest retraction. The intercostal spaces on right side were narrowed. Left lung was overinfl ated (Photograph-I). The lesions were non progressive on serial chest X-rays.

Bronchoscopy revealed the under development of right bronchial tree. Right upper lobe bronchus was seen arising just near the carina and only two small openings were seen. Middle and lower lobe bronchus were under developed. However, normal development of tracheobronchial tree of left side was seen. CT Scan Thorax revealed marked asymmetry in thorax. The left lung showed herniation through anterior recess with evidence of oligaemia suggesting compensatory over infl ation. The right sided lung showed severe decrease in

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volume with only minimal residual lung tissue. Bronchiectatic changes were seen in this lung tissue. The mediastinum was seen to be shifted to right side (Photograph-II). Patient is still under our follow up and treatment. Patient develops recurrent chest infections which respond to broad spectrum antibiotics and symptomatic treatment.

Department of Pulmonary Medicine, King George's Medical University, Lucknow Correspondence : Dr Surya Kant, Associate Professor, Department of Pulmonary Medicine, King George's Medical University, Lucknow (U.P.), INDIA Received : Jan. 2006 Accepted : Jan. 2006

## Discussion

Development of the bronchial tree takes place at about 26th to 31st day of intrauterine life. Monaldi divided the mal-development of lung in four groups. Group I: No bifurcation of trachea; Group II: Only rudimentary main bronchus; Group III: Incomplete development after division of main bronchus; and Group IV: Incomplete development of subsegmental bronchi and small segment of the corresponding lobe. The present case belongs to the fourth group of Monaldi classifi cation.

According to Boyden7 there are three degrees of mal-development: (i) agenesis, in which there is complete absence of lung tissue, (ii) aplasia, in which rudimentary bronchus is present but no lung tissue is present, and (iii) hypoplasia, in which all the normal pulmonary tissues are present but are under-developed.

Hypoplasia of the lung may be regarded as primary (idiopathic) or secondary (when it occurs in association with environmental factors or other congenital anamolies that may be implicated in its pathogenesis). The incidence of secondary form is diffi cult to determine; however, because of its association with a variety of other abnormalities and the diffi culty of pathologic diagnosis in some cases, it is likely to be more common than generally recognized. The incidence of primary hypoplasia has been estimated to be 1 to 2 per 12,000 . Several mechanisms have been implicated in secondary pulmonary hypoplasia including decreased hemithoracic volume, decreased pulmonary vascular perfusion, decreased fetal respiratory movement and decreased lung fluid.

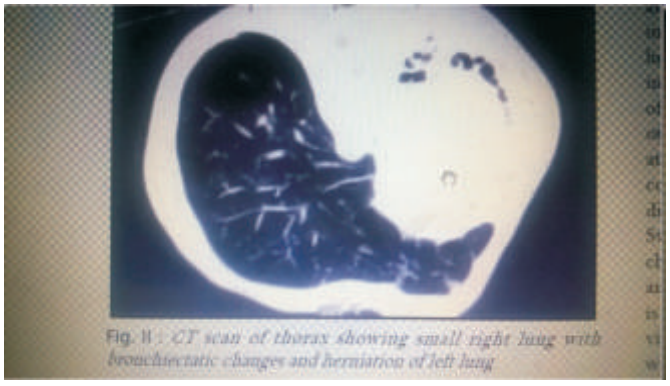

Fig. II : CT scan of thorax showing small right lung with bronchiectatic changes and herniation of left lung

Although the pathologic diagnosis of pulmonary hypoplasia can be made on formalin-infl ated, routinely processed lungs on the basis of a combination of fresh lung weight, fi xed lung volume, radial alveolar count and estimates of tissue maturity, precise characterization of the morphologic changes is best performed by morphometric measurement after inflation of the lungs to a known transpulmonary pressure.

Hypoplastic lungs are typically smaller and weigh less than normally expected for their age. Although there is variation in the severity and type of changes between different cases, the

most consistent fi nding is a decrease in number of airway generation, ranging form about 50% to 75%, of normal. In addition, then is frequently a decrease in the number of alveoli, estimated by one group of investigators to be about one-third normal. This is often associated with a decrease in alveolar size. Some investigators have shown normal airway and alveolar maturation for gestational age; others have found an immature appearance. Abnormalities of pulmonary arterial system have also been identifi ed consisting of decreased elastic tissue in the larger arteries, increased muscle in normally muscular arteries, and extension of muscle into normally non-muscular arteries

The basis of variation in morphologic findings may be related to severity and cause of hypoplasia as well as to the timing of the etiologic events that led to anomaly15.

Radiographic findings in cases of hypoplasia are similar and characterised principally by almost total absence of aerated lung in one hemithorax. The markedly reduced volume is indicated by approximation of ribs, elevation of ipsilateral diaphagram, and shift of the mediastinum. In most cases the contralateral lung is greatly over inflated and displaced along with anterior mediastinum into the involved hemithorax; this displacement of air containing lung to the side of involved lung may lead to some confusion in diagnosis. CT scan may be required to establish the degree of under development and to differentiate hypoplasia from other conditions that may closely mimic it radiographically: atelectasis from other causes, severe bronchiectasis with collapse and advanced fibrothorax. Main differential diagnosis of hypoplastic lung is Swyer James syndrome. Swyer James syndrome is an uncommon abnormality characterized radiologically by hyperlucent lobe or lung and functionally by air trapping during expiration. There is substantial evidence that the syndrome is initiated by viral bronchiolitis. Although both conditions are associated with unilateral very low volume, patients with Swyer James syndrome characteristically demonstrate an air trapping on radiographs or HRCT scans performed at the end of maximal expiration16.

Clinical fi ndings depend on degree of pulmonary abnormality and presence of other congenital malformations. Usually, however the patient is symptomatic. Physical examination characteristically reveals asymmetry of two sides of thorax, reduction in respiratory movements and absence of air entry in the affected side. This may be diagnosed incidentally during childhood when complicated by pulmonary infection19-21. Diagnosis may be established with help of chest x-ray, CT thorax17-18, fiber optic bronchoscopy, and if possible pulmonary angiography and bronchography. Still however there are no clear clinical diagnostic criteria to facilitate the identification and management of lung hypoplasia. Hence current research is based on identification of such clinical diagnostic criteria22.

Dialysis may be required for support of renal function.

Surfactant administration at 4 ml/kg improves survival rate. Surgical care consists of intrauterine vesicoamniotic shunts and endoscopic ablation of valves and PLUG (plug the lung until it grows) by fetoscopic tracheal occlusion with a clip. Post-delivery surgery can be done to correct diaphragmatic hernia, cystic adenomatoid malformations and decompresses pleural effusions

Treatment in adults consists of control of recurrent infections, symptomatic treatment in form of expectorants and bronchodilators and management of other complications.

Prophylaxis for respiratory syncytial virus, pneumococcus, infl uenza infections are recommended.

# NEWER ANTI-LIPIDS

**Dr. Nitin Sinha**

A new group of drugs has come up as anti-lipids. These drugs are monoclonal antibodies that inhibit Protein Convertase Subtilisin Kinase-9 (PCSK-9) in liver. Inhibition of PCSK-9 causes less degradation of Low Density Lipoprotein Receptor (LDL Receptor) and thus, causes increase uptake of LDL in the liver thereby decreasing the blood values of LDL cholesterol. Alirocumab (one of the member of this new group of anti lipid) has gained FDA approval in July 2015 to be used as an anti-lipid. It is to be given as a subcutaneous injection once every 2 weeks. Dose ranges from 75 mg to 150 mg. This drug, however, as per the studies has been used only in conjunction with other anti lipid agents. This drug is to be used in patients of Familial Hypercholesterolemia, established Coronary Artery disease patients and in patients having CHD risk equivalents that have not received desired LDL goal (<70 mg/dL of LDL) on maximum dose or maximum tolerable dose of statins or other anti lipid drugs. There is no data as of now to support alirocumab use as a solo therapy for hyperlipidemia. ALirocumab significantly reduces LDL cholesterol and also reduces non-LDL cholesterol, Triglycerides, apolipoprotein a and total cholesterol and raises HDL cholesterol modestly. Maximum duration of alirocumab use alongwith maximum tolerable dose or maximum dose of statins or other antilipids has been nearly one and a half years and the side effect profile when compared to placebo is almost the same. Adverse events seen with this drug are neurocognitive defects, opthlmological defects, injection site reactions and myalgias. However, there is no rise in CPK or aminotransferases.

Alirocumab promises to reduce LDL to desired levels alongwith statins/other antilipids but its utility as monotherapy still needs to be seen. Also, whether it leads to plaque stabilization needs to be assessed. Cost definitely is a deterring factor. Furthermore, neurocognitive and opthalmological assessment needs to be done prior to starting this drug.

**He who studies medicine without books sails an uncharted sea,**
**but he who studies medicine without patients**
**does not go to sea at all**

**- William Osler**

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# PROPOSED SCIENTIFIC PROGRAM OF EDPACON 2015

| | | |
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| 9:00 - 9:30 am | : | Newer antimicrobials and MDR Infections in Clinical Setting, IPD and OPD |
| 9:30 -10:00 am | : | Recent advances in seizure management & overview of newer antiepileptics |
| 10:00-10:30 am | : | Medical Management of BPH |
| 10:30-11:00 am | : | Newer oral anticoagulants - Which is better and where |
| 11:00-11:30 am | : | Approach to patient with GI bleed |
| 11:30-12:00 pm | : | Hepatitis C - Current management guidelines in physicians perspective |
| 12:00-1:00 pm | : | Late Mrs. Bela Devi Oration & Life Time Achievement Award Ceremony |
| 2:00 -2:30 pm | : | Recent advances in the management of COPD and Bronchial Asthma |
| 2:30 - 5.00 pm | : | Diabetes symposium - Proposed topics Diabetic dyslipidemia - Indian scenario, Gliptins - present status & future promise, Weekend therapy in type 2 Diabetes Mellitus (few more interesting topics are in in pipeline) |
| 5:00-5:30 pm | : | Panel discussion on Step wise management of Chronic Kidney Disease |
| Invited Faculty | : | Dr. Naresh Trehan, Dr. S.K. Sarin, Dr. Sheshadri, Dr. Radhakrishnan, Dr. Randeep Guleria, Dr. A.H. Zerger, Dr. K.K. Sethi |

# MANAGEMENT OF ALCOHOL RELATED CHRONIC LIVER DISEASE - A CASE SCENARIO

**Dr. Kunal Khaneja, Dr. Rohit Goyal, Dr. Deepak Lahoti**

**Liver diseases have many causes including Hepatitis B , Hepatitis C , alcoholism and steatohepatitis associated with diabetes and obesity. Patients can present in various ways Including massive upper gi bleeding as a first manifestation of underlying cirrhosis . In the case described below it seems that alcoholism and steatohepatitis associated with diabetes both contributed to cirrhosis ultimately leading to liver transplantation .The case also reflects on various therapeutic interventions that are possible in case of chronic liver disease.**

Mr A, 55 year male, presented in emergency at our hospital with history of large amount of blood with vomitus about 2 hours prior, associated with giddiness and black stools. He never had similar symptoms in past. He denied any recent history of ingestion of NSAIDs. He was a diabetic for last 12 years with fair glycemic control with his antidiabetic medications. There was no past or family history of liver disease. He used to consume alcohol regularly (about 200 ml of whisky daily for last 10 years).

At admission, his pulse rate was 108/minute, his blood pressure was 86/60 mm Hg and he had severe pallor. He also had mild jaundice. He was resuscitated, his baseline blood investigations were sent and gastric lavage was done. Active bleed was noted on gastric lavage. Therefore, emergency endoscopy was planned and intravenous vasopressors were started to control bleeding. Blood and FFP were arranged.

His endoscopy showed active bleeding from his esophageal varices, for which endoscopic banding was done. His lab reports showed significant anemia, low platelets, deranged liver functions and hyperglycemia. His glycosylated Hb was 9% and ultrasound of abdomen showed cirrhotic liver. He was stabilised with blood transfusions and was sent home on hematinics and beta blockers were added to his previous medications. Glycemic control was optimised. He was also advised to abstain completely from alcohol and take his medicine regularly to maintain good glycemic control. He was also advised to undergo endoscopy after 2 weeks to eradicate the residual esophageal varices.

The patient went home and did well initially. His endoscopy was done after 2 weeks and repeat endoscopy with banding of varices was done. His jaundice improved gradually. Afterwards, he was lost to followup. He started consuming alcohol after 4 months of discharge and stopped all medications.

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He again presented in emergency after 8 months, with massive hematemesis and altered behaviour. This time also, was hypotensive at admission. In addition, he had icterus and moderate abdominal distension. He had poor respiration and required ventilatory support. His endoscopy showed stigmata of recent bleeding and banding of his varices was done. His sensorium improved gradually. His ultrasound confirmed presence of ascites. There was no evidence of spontaneous bacterial peritonitis or hepatocellular carcinoma. Other supportive treatment was given and patient showed gradual recovery.

His liver functions, after initial stabilization, again showed worsening and his ascites continued to persist. He also had intermittent episodes of encephalopathy despite abstinence from alcohol. He was counselled regarding need for liver transplant and strict abstinence from alcohol.

**Liver transplant** team was involved in management of patient, medical therapy was optimized. However, in view of gradual worsening of liver functions despite optimal medical therapy and about six months of complete abstinence from alcohol, decision was taken for liver transplant which was done successfully. Patient has been doing well post transplant and has been living a normal life at present.

## Discussion

Alcohol is one of the commonest causes of liver diseases in India. The other common causes are diabetes, hepatitis B and C viruses and obesity. A combination of more than one factor, as in our case, often leads to rapid liver damage and sometimes liver failure.

Liver is an organ which has multiple functions including synthesis of proteins, carbohydrate and fat metabolism and detoxification of drugs and metabolites. It has enormous capacity to regenerate and repair itself. In fact, liver failure does not manifest itself till more than 90% of liver is damaged.

Blood tests and ultrasound are common tools to assess liver damage in our day to day practice. Unfortunately, none of them has a high sensitivity or specificity in predicting liver diseases. Hence, patients at risk should undergo further evaluation to detect liver damage at early stage so that further steps can be taken to treat them before irreversible damage to liver occurs. Many tools are now available for the same, fibroscan, fibrosure and liver biopsy being the most notable.

We generally recommend and use fibroscan in patients at risk for liver damage. On fibroscan, normal liver stiffness is about 4-6 KPa. A stiffness of more than 12-14 KPa has been associated with liver cirrhosis, while liver stiffness of more than 20 KPa is usually associated with clinically significant portal hypertension, presence of varices and risk of GI bleeding. The risk factors for liver cirrhosis include alcohol intake, long standing diabetes (generally more than 5 years), age more than 40 and high BMI (>35). In addition, presence of iron overload, hepatitis viruses or autoimmune disease puts the patient at risk for progressive liver damage. Presence of two or more risk factors is a strong indication for assessment of liver fibrosis.

Early interventions can reverse or halt the progression of liver disease at least in a proportion of patients at risk. Liver damage can be reversed if underlying factor for liver damage can be eliminated. Treating viral hepatitis and stopping alcohol are strategies which are usually helpful in reversing early cirrhosis. Recent studies have shown reversal of cirrhosis with long term antiviral therapies in patients of hepatitis B and C. However, beyond a certain stage, liver damage becomes irreversible and medical treatment usually fails to restore liver functions at this stage. decompensated liver disease is usually manifested by onset of ascites, coagulopathy, encephalopathy or jaundice. Presence of liver decompensation generally indicates that the patient is going to follow a rapidly downhill course and consideration for liver transplant should be discussed with all patients with hepatic decompensation.

Endoscopy with band ligation is treatment of choice for bleeding due to varices (secondary prophylaxis for variceal bleed). In addition, beta blockers are generally used to further reduce the variceal pressure. the most commonly used beta blocker is propanolol. however, there is growing trend towards carvedilol, which has become drug of choice at many centres for both primary and secondary prevention of variceal bleed. Refractory bleed requires special endoscopic techniques like glue injection, esophageal stenting (Danis stent), transjugular intrahepatic portosystemic shunt (TIPSS) placement and the technical success rates for control of bleed approach 100% with availability of these techniques.

New onset ascites is best managed by spironolactone with or without loop diuretics. however, as cirrhosis progresses, ascites tends to become refractory. Patients with refractory ascites have a poor prognosis with mean survival of only 6 months. Refractory ascites also tends to progress to hepatorenal syndrome and usually is managed by repeated paracenteses, TIPSS or liver transplant.

In addition to liver related problems, once cirrhosis occurs, there is a constant risk of hepatocellular cancer and all patients with liver cirrhosis should be regularly screened for the same. Incidence of HCC in patients with liver cirrhosis is about 2-8% per year. Early stage cancer, if detected, can be offered curative management. Treatment options at this stage include liver resection,tumor ablation and liver transplant depending on the stage of disease and liver function, as well as presence of portal hypertension and functional status of patient. Presence of more than 3 liver lesions, large tumor and portal vein thrombosis usually are associated with lower chances of cure and require palliation. Treatment options in these cases inclu.de transarterial chemoembolisation, radioembolisation and oral chemotherapy.

Liver transplant is the only treatment which offers 'cure' in patients with liver cirrhosis. The transplant process involves several crucial steps. First of all, it should be made sure that the patient is unlikely to do well without transplant and deserves listing for liver transplant. Secondly, patient should not have and major illness which is likely to interfere with transplant outcomes. For example, patients with associated kidney or heart failure or advanced cancers are unlikely to be benefited with liver transplant and are not generally offered the same. In

alcoholic liver disease, strict abstinence should be ensured to prevent recidivism after transplant, which portends poor post tansplant outcomes.

Once a patient is planned for transplant, availability of liver is the next major issue. Liver can be donated by one of the family members of the patient (live donor related transplant) or can be harvested from a dead person after liver donation.

Unfortunately, in India, cadaveric organ availability is meager. However, with increasing awareness, encouragement and support from medical fraternity and paramedical staff, many people are now taking the initiative for organ donation, making it one of the emerging sources of availability of organs for transplant.

Liver transplant, in most centers, has a success rate of more than 90%. Many people are able to lead a normal life after transplant. As discussed earlier, it is the only 'cure' for patients with liver cirrhosis as of today.

**In summary,** liver cirrhosis is common disease especially in people with risk factor like alcohol intake, diabetes and hepatitis viruses. It is usually detected late due to subtle symptoms. Presentation, even for the first time, can be in form of life threatening symptoms as occurred in our case. High index of suspicion and use of more sensitive tests like fibroscan has allowed disease detection at early stage in a significant proportion of patients.

Endoscopy is the treatment of choice in patients presenting with Gi bleed and helps in diagnosing and treating most of these cases.

Close monitoring and strict adherence to therapy is likely to benefit majority of patients with early and intermediate stages of disease. In advanced liver cirrhosis, many patients will require liver transplant. Early detection of complications and timely referral for transplant is crucial for post transplant success.

**There are, in truth, no specialties in medicine, since to know fully many of the most important diseases a man must be familiar with their manifestations in many organs**

**- William Osler**

# TETANUS

**Dr. Nitin Sinha**

Tetanus has been derived from a greek word "Tetanos", which means "to stretch". CDC defines tetanus as "the acute onset of hypertonia or painful muscular contractions (usually of the muscles of the jaw and neck) and generalized muscle spasms without any other apparent medical cause." Neonatal tetanus is defined as "ability of an infant to suck and cry normally for first two days with the ability being lost between day 3 and 28 with the infant becoming rigid and having spasms".

*Clostridium tetani*, the causative organism is a gram positive, spore forming, anaerobic, motile bacilli. The terminal spore gives it a drumstick appearance. C. tetani is found worldwide in soil, in inanimate environment, in animal faeces. Spores of C. tetani can survive in harsh environmental conditions and can resist boiling. C. tetani produces tetanospasmin, which is a highly potent neurotoxin (minimum lethal dose being 2.5 ng/kg). Tetanospasmin binds to presynaptic nerve terminals and inhibits the release of certain inhibitory neurotransmitters (glycine and GABA) which in turn leads to the observed clinical manifestations.

Tetanus occurs worldwide but is more common in hot, damp climates with soil rich in organic matter and in developing countries.

Risk factors for tetanus are inadequate immunization, unhygienic delivery practices, maternal malaria & HIV, otitis media in children, heroin drug abuse etc. Spores or bacteria enter the body through abrasions/ wounds and in case of neonate via the umbilical stump. Tetanus prone wound is any wound which is > 6 hours before surgical toilet or wound of any duration but is of puncture type or shows substantial devitalised tissue or is contaminated with soil or manure or is septic. Incubation period is 3-21 days (median period being 7 days) with period being shorter if the injury/wound is closer to CNS. Once toxin gets attached to the nerve terminals, it cannot be removed and the recovery from tetanus requires generation of new nerve terminals. Recovery from tetanus generally takes 4-6 weeks.

Diagnosis of tetanus is made on clinical grounds. It is triad of muscle rigidity, spasms & autonomic dysfunction. Clinical features of generalised tetanus include trismus, opisthotonus, neck stiffness, laryngeal spasms, risus sardonicus etc. Other forms of tetanus are localised (confined to a particular area and has excellent prognosis), cephalic, otogenic, opthalmoplegic, maternal (during pregnancy and within six weeks of delivery), neonatal and idiopathic.

Differential diagnosis of tetanus include drug induced dystonias, strychnine poisoning, stroke, neurolept malignant syndrome, seizures, encephalitis, stiff man syndrome, rabies.

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Treatment requires ICU care in a dark, silent room. Thorough wound toileting is required to remove devitalized tissue/foreign body. Post exposure prophylaxis by Tetanus Toxoid and anti tetanus immunoglobulin is also required as per the guidelines mentioned in Table 1. Complete course of Tetanus Toxoid to category D patients comprises of administering TT two doses 6-8 weeks apart with third dose being administered at the end of one year and the booster being given after 5 years of the first dose. TT should never be administered in the gluteal reigon. Human anti tetanus immunoglobulin is preferred as it has fewer chances to cause adverse events when compared to its equine derived counterpart. Sedative-hypnotic agents (diazepam, chloroxazone, baclofen etc.) and antibiotics (metronidazole) are mainstays of treatment. Penicillin G was drug of choice but has gone into disrepute following reports of it causing potentiation of toxin mediated action. Muscle relaxation (vecuronium, atracurium) is indicated where sedation alone is inadequate. Pancuronium is not used as it can lead to worsening of autonomic instability. Magnesium sulphate is an effective adjunct for relaxation, sedation and controlling autonomic disturbance.

**Table 1:- Guidelines for administration of Tetanus Toxoid and Anti-tetanus immunoglobulin after wound**

| | **Category A** (Pat had complete course of Toxoid or booster in last five years) | **Category B** (One who had a complete course of Toxoid or booster more than 5 years ago but less than 10 years) | **Category C** (One who had a complete course of Toxoid or booster more than 10 years ago) | **Category D** (One who has not received TT or his immunization status is unknown) |
|---|---|---|---|---|
| **Non tetanus prone wound** | Nothing required | Toxoid one dose | Toxoid one dose | Complete course of tetanus toxoid |
| **Tetanus prone wound** | Nothing required | Toxoid one dose | Toxoid one dose + Human anti tetanus Ig | Complete course of tetanus Toxoid + Human anti tetanus Ig |

Tetanus is the only vaccine preventable non contagious disease. Under our National Immunization Programme, vaccine for tetanus is administered along with diphtheria and pertussis (DPT) as indicated in the schedule to children with a recommendation of booster dose of TT at 10 years and then at 16 years of age. Under the RCH programme, pregnant ladies are given TT injections to prevent maternal and neonatal tetanus (details not mentioned). India has successfully eliminated maternal and child tetanus in 2015.

# ENTERAL NUTRITION

## (Gastrointestinal tract feeding)

**Dr. Naresh Agarwal**

As our insight into balance of nutrition and immunity increase, we have learnt that better nutrition is one of the key factors in better outcome of disease, particularly in critical illness. In intensive care settings, many times clinicians are bound to manage patients with parental nutrition. But as a standard dictum, these must be switched over to enteral nutrition as soon as possible. We are aware that enteral feeding, being natural way of calory intake, is way beyond more advantageous than parenteral feeding. This article pertains to enteral feeding for patients who can not, for one reason or another, take normal oral feeding. Distinct advantages of enteral feeding over parenteral feeding are:

1. It reduces chances of bacterial translation across gastrointestinal mucosal barrier
2. Being natural mode of feeding, it maintains gut functioning and prevents GI failure. It has been observed that compared to parenteral nutrition, patients given enteral feed have better villi-crypt ratio.
3. It provides Glutamine in lumen of GI tract for use of enterocytes and help them maintain their normal homeostasis, as we know glutamine is primary amino acid used for enterocytes nutrition.
4. Less costlier than parenteral nutrition
5. Improves morbidity and mortality

Enteral feeding is considered when Oral intake is likely to be compromised for next 5-7 days, earlier if preexisting malnutrition is there (BMI <16 gm/m or >15% weight loss in last 12 weeks). Enteral feeding can be either pre-pyloric (substances delivered into stomach) or post-pyloric (into jejunum). The choice between the two depends on patency of pyloric channel. Obviously in cases of gastric outlet obstruction (pyloric or duodenal obstruction), nutritional substances have to be delivered beyond obstruction (post-pyloric). However, as a basic thumb rule, pre-pyloric feeding should be the first choice because it reduced problems related to osmolality, and therefore hypertonic, bolus feed at higher feeding rates is possible.

Usual basic and starter rate of calory intake should be 30-40 k.cal /Kg / day. In severe malnutrition (BMI <16 kg/$m^{2)}$, starting dose should not be more than 10-15 Kcal/day, since a higher rate can can cause severe metabolic disturbances.

The calory intake can be increased over next 3-4 days in cases with severe malnutrition or those on ventilator. Even when patient is getting parenteral nutrition, enteral feed at a rate of 10 ml/h should be continued with TPN to maintain intestinal villi and gut flora. It is easy to

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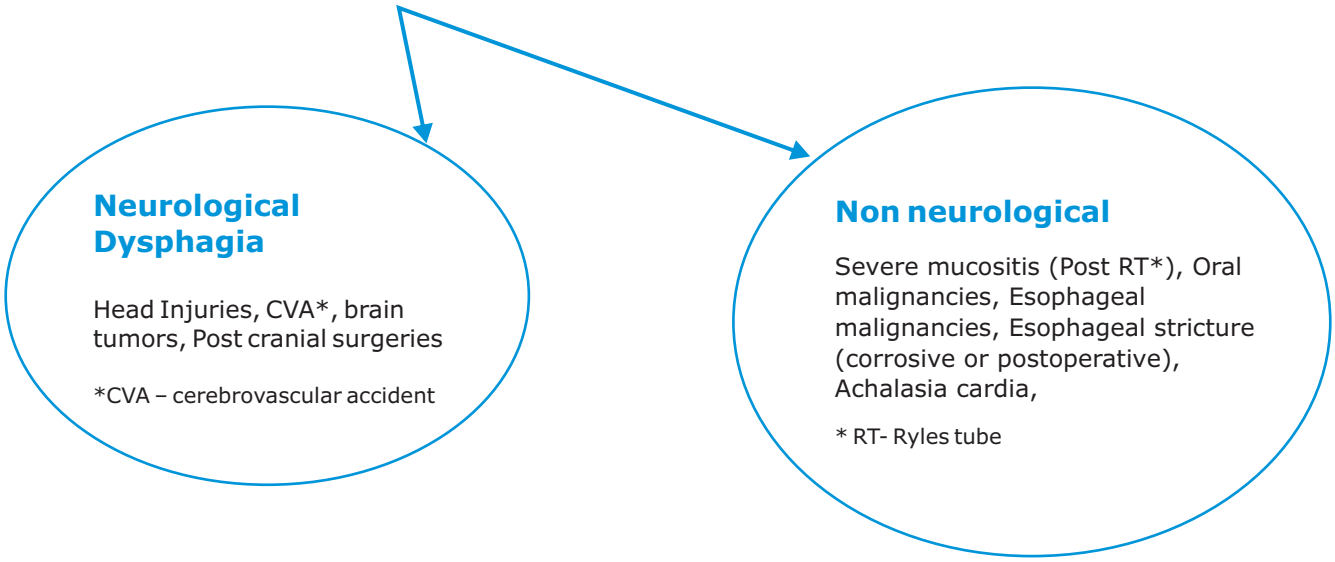
count calory of a particular food substance in an indoor patient since most of the feeds are prepared in hospital kitchen, with calory specification mentioned with them. At home however, it may be difficult to calculate calories. Still it can be calculated at http://www.caloriescount.com. Table 1 depicts few prominent indications for enteral nutrition.

**Table 1**. Indications of enteral nutrition

| |
|---|
| 1. Neurological disorders |
| 2. Mechanical dysphagia (upper GI obstruction, pharyngeal, oral disorders, swallowing disorder) |
| 3. Liver disease with severe anorexia |
| 4. Partial intestinal failure (Postoperative ileus) |
| 5. Inflammatory bowel disease, short bowel syndrome |
| 6. Cystic fibrosis |
| 7. Renal disease |
| 8. Psychological problems : Severe depression / Anorexia nervosa |
| 9. Mechanical dysphagia (upper GI obstruction, pharyngeal, oral disorders, swallowing disorder) |

**Dysphagia** is one of the prominent indications of enteral feedings. Most of these disorders mentioned in table above have dysphagia as common denominator. Dysphagia can be of two main verities..




## Modes of enteral feeding

### Oral feeding

In cases of UGI tract stricture, irrespective of cause, facilitating and enabling patients to take

oral feedings is a rational approach. Common causes of these strictures are corrosive exposure, malignancies, tuberculosis or post operative strictures. Endoscopic dilatation and stent placement are two main modalities in such cases to allow patients to regain oral feeding.

### Assisted enteral

In some cases, oral feeding can not be resumed, and therefore enteral feeding has to be assisted. Various modalities of enteral feeding are (table 2)

- Nasogastric tube feeding
- Gastrostomy feeding
- Nasojejunal tube
- Feeding jejunostomy

**Table 2.** Modalities of enteral feeding

| Nasogastic tube | Nasojejunal tube | PEG * | FJ # |
|---|---|---|---|
| Easy placement<br><br>Non invasive | Good for gastric outlet obstruction | For prolonged feeding, low risk of aspiration, | When PEG or NJ or NG are technically difficult |

*PEG- Percutaneous endoscopic Gastrostomy, # FJ- Feeding jejunostomy

Various factors which influence choice of different modes as stated above are disease indications, longevity for which feeding is required, socioeconomic factors, intellect of the patient and anatomical factors. Table 3 indicated broad criteria for choice of such modalities.

**Table 3.** Choice of enteral feeding

| Factor | Nasogastric | Nasojejunal | PEG/PEJ |
|---|---|---|---|
| Short term (<10-15 days) | √ | | |
| Risk of aspiration | | | √ |
| Prolonged feeding | | | √ |
| Poor intellect | √ | | |
| Poor socioeconomic status | √ | | |
| Gastric outlet obstruction | | √ | √ |

## Nasogastric tubes:

Ryles tube or Freka single lumen (Fig 1) tubes

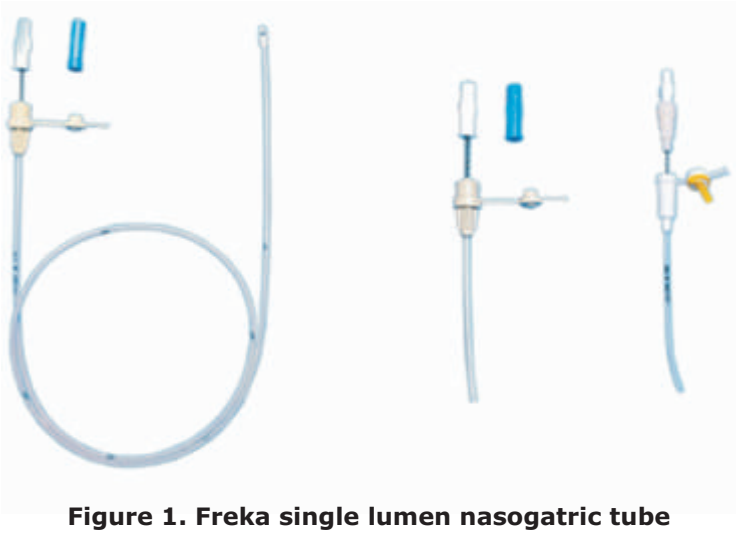


**Figure 1. Freka single lumen nasogatric tube**

Whereas initially Ryles tube was in common use but at present times, Freka tube because of its obvious advantages over Ryles tube are used for feeding purpose.

1. These tubes specifically allows bolus feeding, hypertonic feeding and higher feeding rates
2. Gastric aspiration is usually not required and 15 F tubes for adults and 8-10 F tubes for children are sufficient.
3. Polyurethane tubes (Freka) are preferred over Ryles tube because they have sifter material and are more comfortable for patients, lesser chances of getting cracked and easier to place with lesser trauma
4. Periodically position must be checked and best method for checking position check is skiagram.

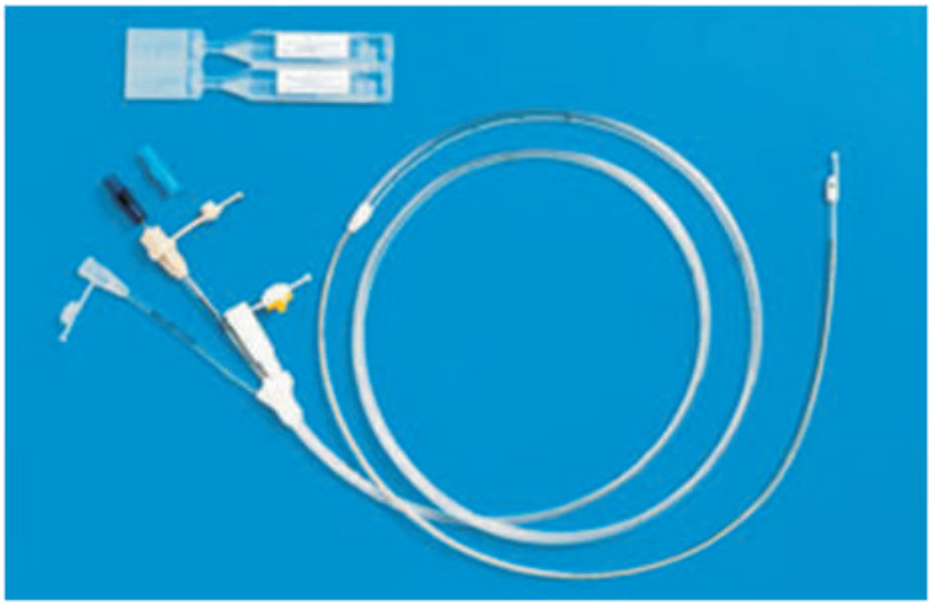


**Figure 2. Nasojejunal tube (triple lumen NJ tubes)**

1. Chosen over NG tube in patients who are nursed flat
2. Ideal for cases of partial gastric outlet obstruction. In complete pyloric obstruction, these tubes may be difficult to place.
3. Most of the NJ tubes are triple lumen Polyurethane tubes (Freka) and have diameter of 6-8 F (jejunal port). They are longer than NG tubes and need endoscopy or fluoroscopic guidance for placement.

## Per-cutaneous Endoscopic Gastrostomy (PEG)

This modality is most widely accepted method of enteral feeding when required for prolonged periods. PEG is primarily done for neurological and oncology patients. It gained popularity and acceptance because of its distinct advantages over any other feeding modality

1. It provides adequate calories even in uncooperative patients.
2. PEG placement is easy and carries very low rate of complications.
3. It is reversible and removal of PEG usually is non invasive, Moreover normal oral feed can be continued along with PEG.
4. Helps patients recover rapidly by preventing respiratory infections resulting from frank or micro-aspirations in these patients.

**Table 4**. Indications of PEG placement

| Mechanical causes | Neurological causes |
|---|---|
| • Mucositis because of radiotherapy (prophylactic or therapeutic)<br>• Commando surgery or other oro-facial surgery associated with dysphagia<br>• Esophageal and gastric malignancies | • Bulbar or pseudobulbar paralysis<br>• Recurrent aspiration related to head injury<br>• Cerebrovascular events (CVA)<br>• Parkinson's disease<br>• Multiple sclerosis<br>• Severe debility<br>• Amyotropic lateral sclerosis |

**Table 5**. Contraindications of PEG placement

| Absolute contraindications | Relative contraindications |
|---|---|
| • Uncorrected coagulopathy or thrombocytopenia<br>• Severe ascites<br>• Hemodynamic instability<br>• Sepsis | • Presence of oropharyngeal or esophageal malignancy (potential risk of seeding of the PEG tract)<br>• Hepatomegaly<br>• Splenomegaly |

| | |
|---|---|
| • Intra-abdominal perforation<br>• Active peritonitis<br>• Abdominal wall infection at the selected site of placement<br>• Gastric outlet obstruction (if PEG tube is being placed for feeding)<br>• Severe gastroparesis (if PEG tube is being placed for feeding)<br>• History of total gastrectomy<br>• Lack of informed consent for the procedure | • Portal hypertension with gastric varices<br>• History of prior abdominal surgeries (possible presence of adhesions and bowel interposition)<br>• Ventral hernia<br>• Peritoneal dialysis<br>• History of partial gastrectomy |

## Method of PEG placement

PEG placement is done by puncturing stomach and abdominal wall in endoscope vision and then threading PEG set with the help of a guide wire by pulling (pull technique - done in 90%) (Fig 3) or by pushing over guide wire (push technique- uncommon). Usually local anesthesia is infiltrated on skin. Intravascular sedation and analgesic are used for fully awake patients. Total procedure takes around 15-20 minutes.

PEG set is hooked inside stomach by a round boster (Fig 4) and on skin by a plastic lock. During Initial 3-4 weeks, daily or alternate day dressing should be done and after that PEG site may be kept open.

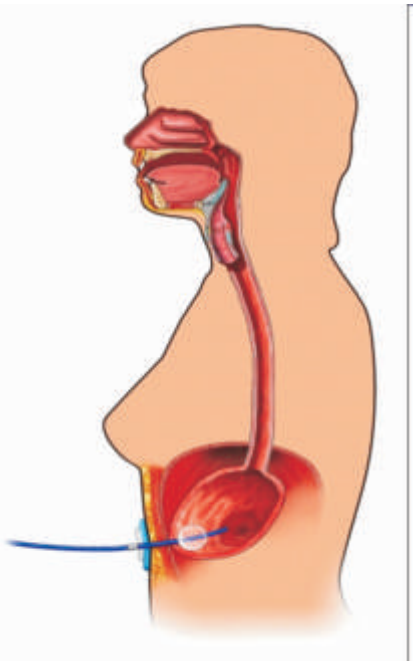


**Figure 3. Pull technique of PEG placement**

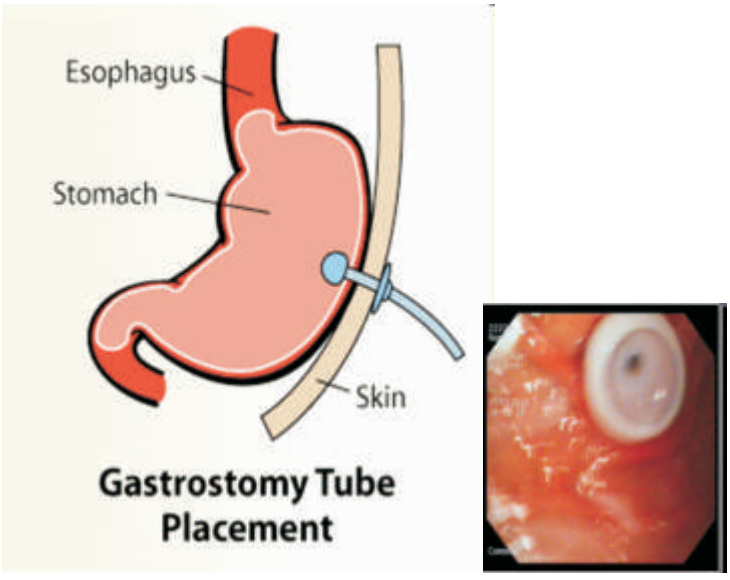



**Figure 4. Inner (Gastric) part of PEG with boster**

## Modes of PEG feeding

Feeding through a PEG tube may be in a bolus form 200-300 ml feeding over 60-90 minutes. This feeding module is difficult in cases of NJ or NG tubes as in those cases it can cause dumping syndromes. Else, a continuous infusion up to 40-50 ml. Hr may be adopted by infusion pump.

Feeding formula may be either Polymeric containing whole protein, starches and whole fats. This is almost equal to routine diet in grinded form. Polymeric forms of feeds are used mostly for mobile unhospitalized patients. Oligomeric feeds, (commonly used for hospitalized and critical patients, contain short peptides or amino acids, partially digested carbohydrates and long or medium chain fatty acids.

## Complications

Largely, PEG placement is a safe procedure, but complications can occur. In best of centers, rate of minor complications are up to 5-7 % and major complications up to 2%. Life threatening complication are extremely rare. Table 6 mentions various complications and their management.

**Table 6**. Complications of PEG placement and their treatment

| Complication | Rate | Treatment |
|---|---|---|
| Pneumo-peritoneum | 50% | Self limiting |
| Colonic injury | 2% | Removal of PEG, Parenteral nutrition, surgery required rarely |
| Gastro-colo-cutaneous fistula | <0.5% | Often conservative TPN |
| Small bowel injury | Rare | |
| Liver injury | Rare | Conservative |
| Necrotizing fasciitis | < 0.5% | Surgical debridement |
| Buried bumper syndrome | 1.5-1.9% | Conservative (surgery if sepsis) |
| Peri-ostomal leakage | 2-3% | Repositioning |
| Accidental pull | Up to 5% | Conservative (surgery if sepsis sets in) |

Buried bumper syndrome (BBS) is a condition in which inner boster lies between gastric wall and skin and can further complicate into peritonitis. This usually happens if PEG tube is partially pulled up accidentally by staff or patients himself. In such scenario, feeding should be immediately stopped and medical attention sought. Treatment includes removal of PEG tube, intravenous replacement of calories, fluid and antibacterials. Most of the cases respond to conservative treatment

Leakage from the site f PEG tube insertion is not uncommon and can be easily treated by readjusting PEG tube. Peri-ostomal denudation of skin is also common and does not pose any threat to PEG functioning. It can be managed by adequate and proper dressing. Here it is pertinent that in fully developed countries, PEG ostoma are not dressed and are kept open. However in patients with poor socioeconomic background, disinfection methods, hand sterilization and hygiene are never assured and thus alternate day dressing and covered stoma may a better approach.

## Button PEG

Tube of the conventional PEG, lying outside abdominal wall sometimes is cumbersome to patients and is a source of accidental and recurrent pull in few cases. It can get cracked over time. Recently button PEG (technically known as low profile PEG set) (Fig. 5) has been designed by few US companies. They have

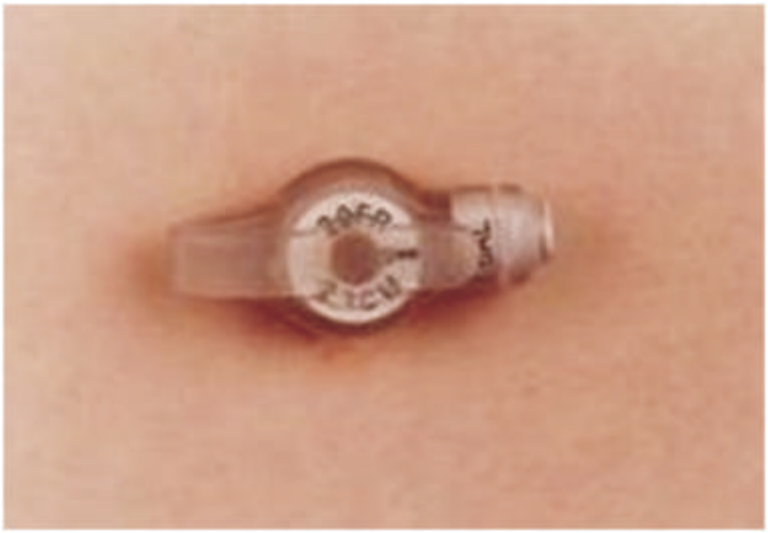


**Figure 5. Low profile PEG set in situ**

Detachable tubes and body of PEG set lies almost parallel to skin. It improves cosmetic appearance and provides least inconvenience to patients. More and more designs and better quality products are needed before button PEG becomes universally accepted.

## Summary

All efforts should be made to enable compromised patients to promote enteral feedings as long as possible. Approach and choice of modality and feeding modules should be individualized. Nasogastric / nasojejunal feeding for short term feeding, PEG for long term feeding, and polymeric formula are obvious preferences.

*Note*: Any queries related to PEG or enteral feeding can be forwarded to my email: "nareshagarwal.na@gmail.com". It will be my pleasure to share with you the best possible authentic replies.

# 2015 RECOMMENDATIONS FOR THE TREATMENT OF MALARIA BY WHO

## Diagnosis of Malaria

All cases of suspected malaria should have a parasitological test (microscopy or Rapid diagnostic test (RDT)) to confirm the diagnosis.

Treat children and adults with uncomplicated P. falciparum malaria (except pregnant women in their first trimester) with one of the following recommended artemisinin-based combination therapies (ACT):

- artemether + lumefantrine
- artesunate + mefloquine
- artesunate + sulfadoxine–pyrimethamine (SP)
- artesunate + amodiaquine
- dihydroartemisinin + piperaquine

## Duration of ACT treatment

ACT regimens should provide 3 days treatment with an artemisinin derivative.

Revised dose recommendation for dihydroartemisinin + piperaquine in young children

Children < 25kg treated with dihydroartemisinin + piperaquine should receive a minimum of 2.5 mg/kg body weight (bw) per day of dihydroartemisinin and 20 mg/kg bw per day of piperaquine daily for 3 days.

## Reducing the transmissibility of treated P. Falciparum infections

In low-transmission areas, give a single dose of 0.25 mg/kg bw primaquine with ACT to patients with P. falciparum malaria (except pregnant women, infants aged < 6 months and women breastfeeding infants aged < 6 months) to reduce transmission. Testing for glucose-6-phosphate dehydrogenase (G6PD) deficiency is not required.

## Treating uncomplicated P. falciparum malaria in special risk groups

### First trimester of pregnancy

Treat pregnant women with uncomplicated P. falciparum malaria during the first trimester with 7 days of quinine + clindamycin.

Treat infants weighing < 5 kg with uncomplicated P. falciparum malaria with ACT at the same mg/kg bw target dose as for children weighing 5 kg.

### Patients co-infected with HIV

In people who have HIV/AIDS and uncomplicated P. falciparum malaria, avoid artesunate + SP if they are being treated with co-trimoxazole, and avoid artesunate + amodiaquine if they are being treated with efavirenz or zidovudine.

Non-immune travellers

Treat travellers with uncomplicated P. falciparum malaria returning to non-endemic settings with ACT.

### Hyperparasitaemia

People with P. falciparum hyperparasitaemia are at increased risk for treatment failure, severe malaria and death and should be closely monitored, in addition to receiving ACT.

## Treating uncomplicated P. vivax, P. ovale, P. malariae or P. knowlesi malaria

Blood stage infection

If the malaria species is not known with certainty, treat as for uncomplicated P. falciparum malaria.

In areas with chloroquine-susceptible infections, treat adults and children with uncomplicated P. vivax, P. ovale, P. malariae or P. knowlesi malaria with either ACT (except pregnant women in their first trimester) or chloroquine.

In areas with chloroquine-resistant infections, treat adults and children with uncomplicated P. vivax, P. ovale, P. malariae or P. knowlesi malaria (except pregnant women in their first trimester) with ACT.

Treat pregnant women in their first trimester who have chloroquine-resistant P. vivax malaria with quinine.

Preventing relapse in P. vivax or P. ovale malariab

The G6PD status of patients should be used to guide administration of primaquine for preventing relapse.

To prevent relapse, treat P. vivax or P. ovale malaria in children and adults (except pregnant women, infants aged < 6 months, women breastfeeding infants aged < 6 months, women breastfeeding older infants unless they are known not to be G6PD deficient, and people with G6PD deficiency) with a 14-day course (0.25-0.5 mg/kg bw daily) of primaquine in all transmission settings.

In people with G6PD deficiency, consider preventing relapse by giving primaquine base at 0.75 mg/kg bw once a week for 8 weeks, with close medical supervision for potential primaquine-induced haemolysis.

When G6PD status is unknown and G6PD testing is not available, a decision to prescribe primaquine must be based on an assessment of the risks and benefits of adding primaquine.

## Pregnant and breastfeeding women

In women who are pregnant or breastfeeding, consider weekly chemoprophylaxis with chloroquine until delivery and breastfeeding are completed, then, on the basis of G6PD status, treat with primaquine to prevent future relapse.

## Treating severe malaria

Treat adults and children with severe malaria (including infants, pregnant women in all trimesters and lactating women) with intravenous or intramuscular artesunate for at least 24 h and until they can tolerate oral medication. Once a patient has received at least 24 h of parenteral therapy and can tolerate oral therapy, complete treatment with 3 days of ACT (add single dose primaquine in areas of low transmission).

## Revised dose recommendation for parenteral artesunate in young children

Children weighing < 20 kg should receive a higher dose of artesunate (3 mg/kg bw per dose) than larger children and adults (2.4 mg/kg bw per dose) to ensure equivalent exposure to the drug.

If artesunate is not available, use artemether in preference to quinine for treating children and adults with severe malaria.