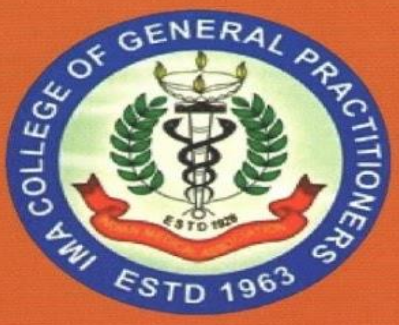

the FAMILY DOCTOR

THE OFFICIAL JOURNAL OF IMA COLLEGE OF GENERAL PRACTITIONERS

JANUARY 2022

Content

Indian Medical Association HQs
(New Delhi)

National President Message

Greetings from Indian Medical Association HQs!

It is my great pleasure to know that IMA College of General Practitioners has decided to publish its Monthly magazine for General Family Practitioners.Its first issue will come out on 31st January 2022 through virtual mode.

The I.M.A COLLEGE OF G.P has set up an uniform pattern of refresher activities for our General Practitioners working all over the country and is deriving full cooperation from the medical colleges and other postgraduate institutions. Assistance is also available sometimes from the Union and State Governments on individual programme basis.

I wish that this monthly magazine of IMA CGP will be a grand success. .

Long Live IMA!!

Dr. Sahajanand Prasad Singh
National President,
IMA HQs

Indian Medical Association HQs
(New Delhi)

Honorary Secretary General Message

Greetings from Indian Medical Association HQs!

It gives me immense pleasure to greet the IMACGP HQs for planning to publish the first issue of FAMILY DOCTOR Journal on 31st January 2022.

I strongly feel that this Journal contains all the information needed for the modern-day medical practitioners and this Journal will be a ready reckoner for the practitioners.

I am confident that the same will be a document of study and reference for our members.

Once again congratulating you and your team for the wonderful efforts and wishing you all the best in your endeavor.

Long Live IMA!!

Dr. Jayesh M. Lele
Honorary Secretary General,
IMA HQs

Message from - Dean, IMA CGP

I feel proud and delighted on the occasion of releasing the first issue of **'FAMILY PHYSICIAN'** the journal of IMA College of General Practitioners', for the year 202-22. The family practice is the foundation of the medical service given by all the doctors. IMA CGP has always strived to make this foundation a sturdy, multifaceted and updated.

This year we will be releasing this journal every month and to keep up with the ongoing trend of World Wide Web revolution, the magazine will be presented as an e-edition and will be circulated to all the members all over India.

I take this opportunity to invite all the great scholars of IMA CGP from all over the nation, all the state faculties of IMA CGP to contribute your own pearls of wisdom and make this mission of knowledge a thriving one. I am sure it will make this publication a true representation of IMA pan India.

I am grateful to the National President **Dr. Sahajanand Prasad Singh**, Hon. Secretary General **Dr. Jayesh Lele** for giving us this opportunity for dispersal of these capsulated insights for all the members of IMA CGP. I must congratulate **Dr. Ravisankar T.N**. and **Dr. Yasodha** for accepting and carrying out the challenge to publish this periodical in an innovative and brilliant way. I am indebted for the rock solid support given in all the activities by **Dr. C. Anbarasu**, the Secretary IMA CGP, **Dr. S. Rekha** and **Dr R.Anburajan**, the Joint Secretaries.

I wish a great success to the Team IMA CGP for this new confront.

Dr. Avinash Bhondwe
Dean, IMA CGP

CGP Secretary Message

Dear, Family Physicians,

I take pleasure to present the first issue of THE FAMILY DOCTOR journal for this IMA year. A journal is the communication with the members and we have envisaged an index journal for the Family Physician of our country. An Editorial team with members from all over the country is formed but we still need some more who can contribute for the growth of the journal. We have proposed to publish in print for once in three months and online on all the months. CGP activities at local branch and at State level will also be published, hence request all to send the details to the CGP mail for publication. Lets join together to bring back the reputation and recognition for the FAMILY PHYSICIAN with knowledge and skill. A good and knowledgeable Family Physician can provide affordable and accessible QUALITY health care for our citizens.

Dr.C.Anbarasu

Hon.Secretary, IMA CGP

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Hyperprolactinemia and Women's Health

Dr. Annith Kumar V.M,
MS(OG), MRCOG (UK), DRM (Germany), FRM.

Physiologic role of prolactin (PRL) hormone

The most important of which is to stimulate the mammary glands to produce milk (lactation). Increased serum concentrations of PRL during pregnancy cause enlargement of the mammary glands of the breasts and increases the production of milk. The hormone represses the effect of dopamine, which is responsible for sexual arousal, thus causing the sexual refractory period. The amount of PRL can be an indicator for the amount of sexual satisfaction and relaxation. On the other hand, high amounts are suspected to be responsible for impotence and loss of libido. Gregg, et al; 2007 PRL has been found to stimulate proliferation of oligodendrocyte precursor cells. These cells differentiate into oligodendrocytes, the cells responsible for the formation of myelin coating on axons in the CNS (Snyder and Dekowski, 1992)

It is postulated that PRL acts on hypothalamo-pituitary–ovarian axis. PRL inhibits pulsatile secretion of GnRH and therefore gonadotrophin secretion and has a direct effect on the ovary itself which is supposed to be responsible for the menstrual disturbances that are seen with HP. The amenorrhoea associated with elevated PRL is due to an inhibition of the pulsatile secretion of GnRH. The pituitary gland in these patients responds normally to GnRH or in augmented fashion (perhaps because of increased stores of gonadotrophins), thus indicating that this mechanism of amenorrhoea is a decrease in GnRH (Sauder, et al; 1984). Short-term administration of an opioid antagonist suggests that inhibition is mediated by increased opioid activity (Cook, et al; 1991).

Nevertheless, treatment that lowers PRL restores ovarian responsiveness and menstrual function. This is true whether the treatment consists of removal of PRL-secreting tumor or suppression of PRL secretion.

The increase in PRL levels observed in pathological HP results in effects equivalent to those observed during the postpartum period, namely inhibition of the release of GnRH from the hypothalamus and subsequent inhibition of LH and FSH, suppressed gonadal function and promotion of milk formation; this explains why HP is one of the most frequent causes of anovulation.

Prolactin is encoded by a single gene on chromosome 6, producing a molecule that in its major form is maintained in three loops by disulfide bonds. Most, if not all, variants of prolactin are the result of posttranslational modifications. The predominant form, little prolactin, results from the proteolytic deletion of amino acids. Big prolactin has little biologic activity and does not cross-react with antibodies

to the predominant form of prolactin. The so-called big big variants of prolactin are due to separate molecules of prolactin binding to each other, either noncovalently or by interchain disulfide bonding. Some of the apparently larger forms of prolactin are prolactin molecules complexed to binding proteins. High levels of relatively inactive prolactin in the absence of a tumor can be due to the creation of macromolecules of prolactin by antiprolactin autoantibodies.

Overall, the presence of big prolactins accounts for somewhere between 10% and 25% of the hyperprolactinemia reported by commercial assays. Other variations of prolactin exist. Enzymatic cleavage of the prolactin molecule yields fragments that may be capable of biologic activity. Prolactin that has been glycosylated continues to exert activity; differences in the carbohydrate moieties can produce differences in biologic activity and immunoreactivity.

However, the nonglycosylated form of prolactin is the predominant form of prolactin secreted into the circulation. Modification of prolactin also includes phosphorylation, deamidation, and sulfation.

The prolactin receptor is encoded by a gene on chromosome 5 that is near the gene for the growth hormone receptor. However, there is evidence for more than one receptor, depending on the site of action (e.g., decidua and placenta). The prolactin receptor belongs to the receptor family that includes many cytokines and some growth factors, supporting a dual role for prolactin as a classic hormone and as a cytokine. The prolactin signal is mediated through a cytoplasmic tyrosine kinase pathway.

At any one point of time, the bioactivity (e.g., galactorrhea) and the immunoreactivity (circulating level by immunoassay) of prolactin represent the cumulative effect of the family of structural variants. Remember, immunoassays do not always reflect the biologic situation (e.g., a normal prolactin level in a woman with galactorrhea).

Galactorrhea

It refers to the mammary secretion of a milky fluid, which is non-physiologic in that being inappropriate (not immediately related to pregnancy or the needs of a child), persistent, and sometimes excessive. To elicit breast secretion, pressure should be applied to all sections of the breast beginning at the base of the breast and working up toward the nipple. Hormonally induced secretions usually come from multiple duct openings in contrast to pathologic discharge that usually comes from a single duct. A bloody discharge is more typical of cancer. The quantity of secretion is not an important criterion. Amenorrhea does not necessarily accompany galactorrhea, even in the most serious provocative disorders. Any galactorrhea demands evaluation in a nulliparous woman and if at least 12 months have elapsed since the last pregnancy or weaning in a parous woman. Galactorrhea can involve either breasts or just one breast. This recommendation has evolved empirically, knowing that many women have the persistence of galactorrhea for many months after breastfeeding, and therefore the rule is a soft one. The exact numbers have never been established by appropriate studies. Thus, there is room for clinical judgment with this clinical problem. Galactorrhea is present in about 30–80% women; this may reflect

the duration of gonadal dysfunction, because women with long- standing estrogen deficiency are less likely to have galactorrhea.

Galactorrhea with normal PRL level

Only one-third of women with high PRL levels have galactorrhea, probably because the low estrogen environment associated with the amenorrhea prevents a normal response to PRL. Another possible explanation again focuses on the heterogeneity of peptide hormones. PRL circulates in various forms with structural modifications, which are the result of glycosylation, phosphorylation, deletions, and additions.

Clinical presentations of hyperprolactinemia (HP)

PRL hormone may increase in some physiologic situations that should be considered firstly. They include pregnancy, breast stimulation, breastfeeding, sexual intercourse, stress, exercise, sleep and postictal state. Pathologic HP typically it may cause oligomenorrhea, amenorrhea, galactorrhea, or infertility (Jones, 1995). In hyperprolactinemic women, the incidence of galactorrhea is up to 80%, depending on the diligence with which galactorrhea is sought (Vance and Thorner, 1987). HP may be found in 30% of women with secondary amenorrhea, and in 75% of women with both amenorrhea and galactorrhea (Schlechte, et al; 1980).

Why some women develop menstrual irregularities up to amenorrhea?

In hyperprolactinemia, menstrual disorders ranging from irregular bleeding, insufficient luteal phase, spanio-amenorrhea, to anovulatory cycles and amenorrhea, are frequent. Multiple mechanisms are involved in these disorders: hyperprolactinemia could act at the hypothalamic level on LHRH secretion and directly on LH and sex steroids secretion. Hyperprolactinemia could also act by impairing fertilization or implantation at the endometrial level.

HP and polycystic ovaries

The increased production of PRL observed in patients with PCO is usually transient and does not require treatment (Milewicz, 1984). PCO may be associated in up to 40%. Laparoscopic ovarian drilling may lead to HP in one small sample sized study (Parsanezhad et al., 2005). It is seen in 40% of polycystic ovarian syndrome (PCOS) patients (Conner and Fried, 1998). PCOS and Prolactinoma may co-exist and may need to be treated independently (Bracero and Zacur, 2001).

Prolacin and Infertility

While hyperprolactinemia is a well-established cause of hypogonadotropic hypogonadism and infertility, the mechanisms of these effects are not well understood. Elevated prolactin may impact reproduction through inhibitory effects on hypothalamic gonadotropin-releasing hormone (GnRH) neurons and/or on the pituitary gland to reduce secretion of the gonadotropins luteinizing hormone (LH) and follicle-stimulating hormone (FSH), resulting in a reduction in both amplitude and frequency of LH pulses. Prolactin may act directly on GnRH neurons to suppress GnRH secretion, or the effects may be indirect, mediated through other afferent pathways, perhaps via other neurons influencing GnRH release. During lactation, additional metabolic factors induced by negative energy balance may also contribute to disruption of pulsatile GnRH and LH secretion.

IMA CGP Headquaters request all state and Sub faculty to send their activities report with photographs for publishing in this journal . It can be sent by mail addressed to The Editorila Team, The Family doctor , IMA CGP Hq. on cgpima@gmail .com

We will be glad if you can send in scientific articles form you branch or state which will be of intrest to the Family practationers. It could be orginal articles,clinical materials, Rare diaglosis, review articles etc.

Team CGP Hq.

Omicron Vs Delta: What we know and how both are different?

Dr S Jamuna Rani, Professor, Department of Pathology,
Tagore Medical College & Hospital, Vandalur, Chengalpet Dt

Background:

The World Health Organization has announced that the Omicron variant is a matter of grave concern which created panic across the world. Even though vaccination is the key to prevent complications and mortality, reinfections and break through infections are seen very commonly during this third wave. On one end, it looks promising that the hospitalizations and ICU admissions are seen very less in the third wave when compared to Delta. On the other end, more evidence on this new variant will be helpful in looking at long term complications and treatment modalities.

Omicron variant:

A new variant called "B.1.1.529" was notified by the South Africa on 24th November 2021. The spike protein of the Omicron variant is characterized by at least 30 amino acid substitutions, three small deletions, and one small insertion. Notably, 15 of the 30 amino acid substitutions are in the receptor binding domain (RBD).

Comparison between Omicron and Delta

Characteristics	Omicron	Delta
Variant name/Pango lineage	B.1.1.529	B.1.617.2
Country of first identification	South Africa	India
Mutations	The spike protein of the Omicron variant is characterized by at least 30 amino acid substitutions, three small deletions, and one small insertion.	The spike protein contained eight mutations, including four mutations in the NTD, two in the RBD, one mutation close to the furin-cleavage site and one in the S2 region.
Transmissibility	More transmissible than delta and original SARS-CoV strain	More transmissible(twice) than original SARS-CoV strain
R0	>8 (under study)	6.5 to 8

Disease severity	No unusual symptoms. Mostly mild or asymptomatic infections. Hospitalizations are shorter if needed. Mortality is very lesser when compared to delta.	
Impact on vaccine induced immunity	As Omicron variant contains more changes in the spike protein, significant reductions in neutralizing activity of sera from vaccinated or previously infected individuals, which may indicate reduced protection from infection, are anticipated. But real world data shows that even though break through infections occur, mortality and severity is less among vaccinated individuals.	For prior variants, lower amounts of viral genetic material were found in samples taken from fully vaccinated people who had breakthrough infections than from unvaccinated people with COVID-19. For people infected with the Delta variant, similar amounts of viral genetic material have been found among both unvaccinated and fully vaccinated people. (Ref: CDC) Protective efficacy varies between 30-40%
Impact on monoclonal antibody treatments	The Omicron VOC, which has numerous mutations in the spike protein, is predicted to have markedly reduced susceptibility to bamlanivimab plus etesevimab and casirivimab plus imdevimab. Because sotrovimab is the only available anti-SARS-CoV-2 mAb with activity against the Omicron VOC. Molnupiravir is expected to be active against the	Currently, 3 anti-SARS-CoV-2 mAb products have received Emergency Use Authorizations (EUAs) from the Food and Drug Administration (FDA) for the treatment of mild to moderate COVID-19 in nonhospitalized patients with laboratory-confirmed SARS-CoV-2 infection who are at high risk for progressing to severe disease and/or hospitalization. (Ref: NIH)

	Omicron VOC, although in vitro and in vivo data are currently limited.[12]	• Bamlanivimab plus etesevimab • Casirivimab plus imdevimab. • Sotrovimab
Impact on diagnostics	Most of the available assays detect the Omicron variant. But to distinguish S gene mutations – only few assays like TaqPath can be used. Genomic sequencing is the gold standard to confirm the variant.	All the assays were able to detect delta variant during the second wave.

Fortunately, the real time scenario of omicron is not scary with regards to severity and mortality. We have learnt that vaccination and infection control measures are the only ways to combat the pandemic. As top virologists said, we must learn to live with the virus as it keeps on mutating forever.

Anti -Viral drugs in COVID -19: A brief overview

Dr.RUCKMANI. A.
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The pharmacotherapy of COVID-19 has been evolving over a period of more than 2 years with more understanding of the SARS- CoV-2 viral multiplication and disease progression. Sudden occurrence of such a massive pandemic with an unknown virus left the clinicians with no choice but to repurpose some of the available drugs combining with symptomatic treatment with the hope to provide relief or save lives.

Many available drugs like Hydroxychloroquine, (HCQ), Azithromycin, Glucocorticoids, Remdesivir, Favipiravir, Lopinavir/Ritonavir, Tocilizumab, Sarilumab, anti-coagulants, COVID-19 convalescent plasma, Mesenchymal stem cells, Interferons, Ivermectin, and traditional medicines have been used presumptively either for their anti-viral activity or for the prevention of complications of COVID.

Among these drugs the anti-viral drugs Remdesivir and Favipiravir were given Emergency use approval by FDA, USA and CDSCO, India.

On May 1, 2020, the FDA issued an Emergency Use Authorization (EUA) for the use of Remdesivir for the treatment of hospitalized patients with severe COVID-19 with an SpO2 $\leq$94%.

On June 1, 2020, Remdesivir use was approved by the Drug Controller General of India (DCGI) for patients with severe COVID-19 infection who require supplemental oxygen therapy.

The next anti-viral drug, Favipiravir film-coated tablet 200mg was approved on June 19, 2020, and 400 mg on 22 July 2020, for the treatment of patients with mild to moderate Covid-19 by DCGI.

FDA issued further an EUA for another antiviral drugs combination, Paxlovid (Nirmatrelvir and Ritonavir tablets, co-packaged for oral use) for the treatment of mild-to-moderate COVID-19 in adults and paediatric patients, on Dec.22, 2021(1).

The fourth drug recently approved by FDA under EUA is Molnupiravir, for the treatment of mild-to-moderate coronavirus disease (COVID-19) in adults on Dec 23, 2021(2).

The DCGI, based on the clinical data of Molnupiravir has approved Molnupiravir for treatment of adult patients with Covid-19, with SpO2 > 93% and those with high risk of progression of the disease including hospitalisation or death on Dec 28, 2021(3).

Thus 3 individual drugs and one combination of drugs have been approved for use in COVID-19. The following table shows the different comparative aspects of the drugs under discussion:

Remdesivir	**Favipiravir** (4,5)	**Paxlovid** (6)	**Molnupiravir** (7)
How do these drugs inhibit viral multiplication?			
Inhibits viral RNA dependent RNA polymerase enzyme (RdRp), preventing viral transcription and replication	Similar to Remdesivir, it also inhibits viral RNA dependent RNA polymerase (RdRp), preventing viral transcription and replication	Inhibits viral main protease (Mpro) hence, viral replication. Ritonavir slows Nirmatrelvir's metabolism so that it remains active in the body for longer periods of time at higher concentrations	Targets RdRp enzyme in the virus and causes mutagenesis
Dosage			
A single loading dose - 200 mg on Day 1 – followed by once-daily maintenance dose - 100 mg from Day 2 infused over 30 to 120 minutes diluted in 250 mL of 0.9% sodium chloride for 5-10 days depending upon severity and renal function.	1800 mg twice a day on day 1, followed by 800 mg twice a day maximum up to 14 days in	3 tablets (two tablets of 150 mg Nirmatrelvir and one tablet of 100mg ritonavir) taken together orally twice daily for five days, for a total of 30 tablets. Not to be used for longer than 5 consecutive days.	800 mg 12 th hourly for 5 days for adult patients.
Route of administration			
IV	Oral	Oral	Oral
Indication			

Hospitalized patients with severe COVID	Mild to moderate COVID-19 patients.	Mild-to-moderate COVID-19	Mild-to-moderate COVID-19 with high risk of progression of the disease including hospitalisation or death
Side Effects			
Allergic reactions and increase in liver enzymes. Chloroquine or hydroxychloroquine may decrease the antiviral activity of remdesivir; hence coadministration of these drugs is not recommended	Mild to moderate diarrhoea, increase of blood uric acid and transaminases, and decrease in the neutrophil counts.	Impaired taste, diarrhoea, high blood pressure and muscle aches. Ritonavir may cause liver damage.	Diarrhoea, nausea, dizziness.
Approval for use in children			
Hospitalized pediatric patients between 3.5 and 40 kg, as well as those under 12 years of age who weigh at least 3.5 kg, with suspected or laboratory-confirmed COVID-19.	For those who are 12 years or older who weigh more than 40 kg (88 lbs).	Not approved	For those who are 12 years or older who weigh more than 40 kg (88 lbs).
Use in pregnancy			
Data insufficient to assess the risk (8)	Contraindicated (9)	Risk for the combination unknown (10)	Not recommended (11)
Availability in India			
Yes	Yes	Not yet available	Recently launched

All the above four formulations, in general, are found to be safe. Remdesivir is indicated for hospitalised patients with severe COVID infection and it is recommended for all pediatric age groups. But it can be given only parenterally. The other 3 formulations are indicated for patients with mild to moderate COVID -19 and can be given orally. These drugs prevent the progression of the disease to severity. However, dosage adjustment has to be considered in patients suffering or at risk for hepatic and or renal dysfunction for all the drugs. Caution is needed when patients are on hepatic drug metabolizing enzyme inducers or inhibitors.

The safety of these formulations in pregnancy has not been established due to the non-availability of human data.

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CERVICAL CANCER AWARENESS AND SCREENING SCENARIO IN INDIA (Tamil Nadu)

Dr.S.Rekha Hon. Joint Secretary, IMA CGP HQs

Introduction

Cancer of uterine cervix is a major health problems faced by Indian Women and accounts for around 16% of the annual deaths due to it.

Its Prevalence rate continues to be high in rural India.

The Indian Journal of Medical Research Wolters Kluwer- Medknow Publications .

RATIONALE FOR SCREENING

The usual 10-20 years of natural history of progression from mild dysplasia to cancer makes this type of cancer a relatively early of preventable disease and provides the rationale for screening.

The Indian Journal of Medical Research Wolters Kluwer- Medknow Publications .

REASONS FOR INCREASING INCIDENCE OF CANCER CERVIX IN INDIA

- Early marriage
- Early age at first childbirth.
- Multiparity
- Poor Menstrual hygiene.
- Reproductive tract infections especially(HPV)
- Illiteracy
- Psychological like fear of pain
- Low accessibility to health care service.

URBAN FACTORS FOR INCREASING INCIDENCE

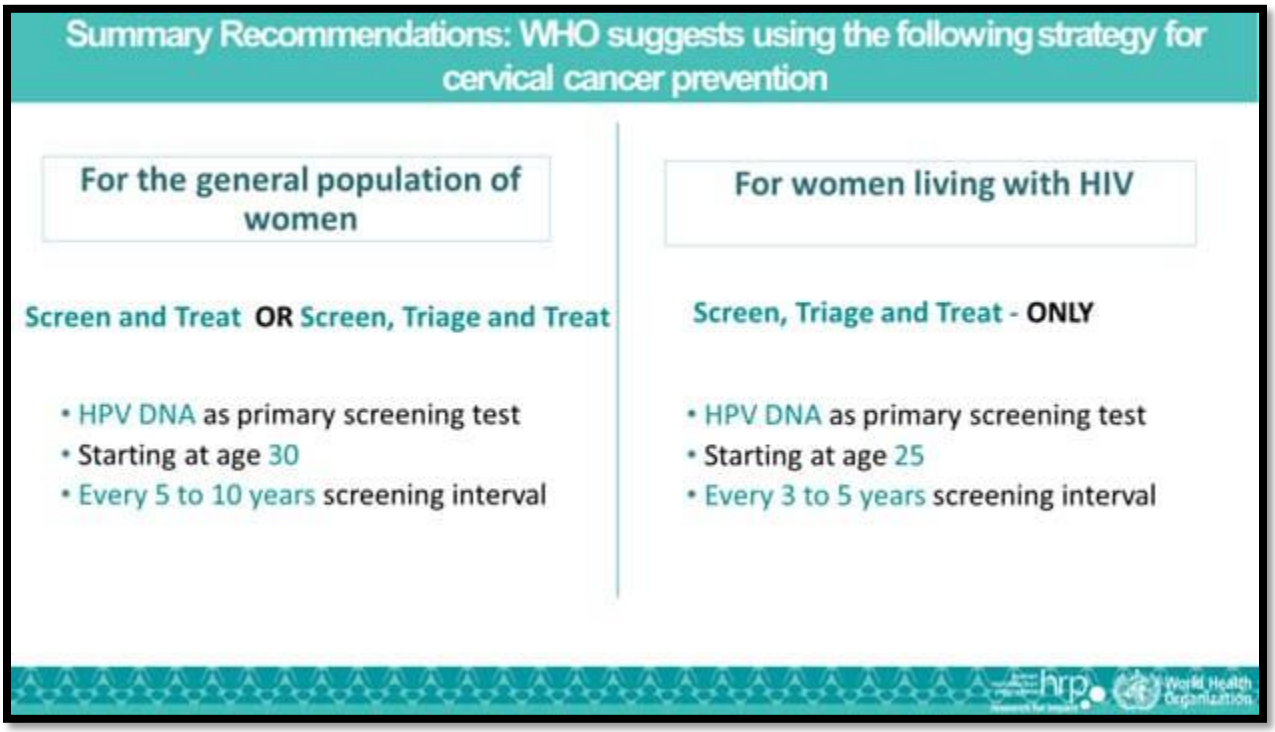
- Consumption of junk food, fatty food, alcohol etc.
- Stress
- Lack of Exercise
- Smoking
- Pollution
- Long term use of contraceptives

Steps taken to control the incidence at Rural Level

- Cancer Registries
- Camp Approach
- Applying Diagnostic methods suitable to low resource settings of rural India- VIA, VIL.
- The Tamil Nadu Health Services project for cervical cancer screening trains Village Health nurses in Screening for Non-Communicable diseases in their screening programmes. The project shows that using health care workers is feasible and effective.

Indian Journal of Gynecologic Oncology 19 Article Number 41 (2021)

Steps Taken to Control the Incidence at Urban Level



According to the latest WHO guidelines.

- For general population of women HPV DNA detection is recommended as the primary screening method starting at the age of 30 years with regular testing every 5-10 years.
- For women with HIV, HPV detection starts at the age of 25 with regular screening every 3-5 years.

Human reproduction programme hrp research for impact 6th july 2021 –UNDP-UNFPA-UNICEF-WHO-WORLD BANK

https://www.euro.who.int>nelos 11/9/2021

HPV DNA BASED TEST

- Detects high risk strains of HPV.
- Relatively simple test.
- Less prone to human errors
- Self sampling kits are available.
- Needs to be repeated only every 5 years
- Requires sophisticated infrastructure.

PRIMARY PREVENTION BY HPV VACCINATION

SL No	HPV VACCINE FORMULATION	AGE(Female)	DOSES	SCHEDULE
1	**Bivalent Vaccine**	19-14 15-26	2 3	Dose 1-0 Dose 2-6 months Dose 1-0 Dose2- 1-2 Months Dose 3-6 Months
2	Quadrivalent Vaccine	9-14 15-26	2 3	Dose1 -0 Dose 2-6 Months Dose 1-0

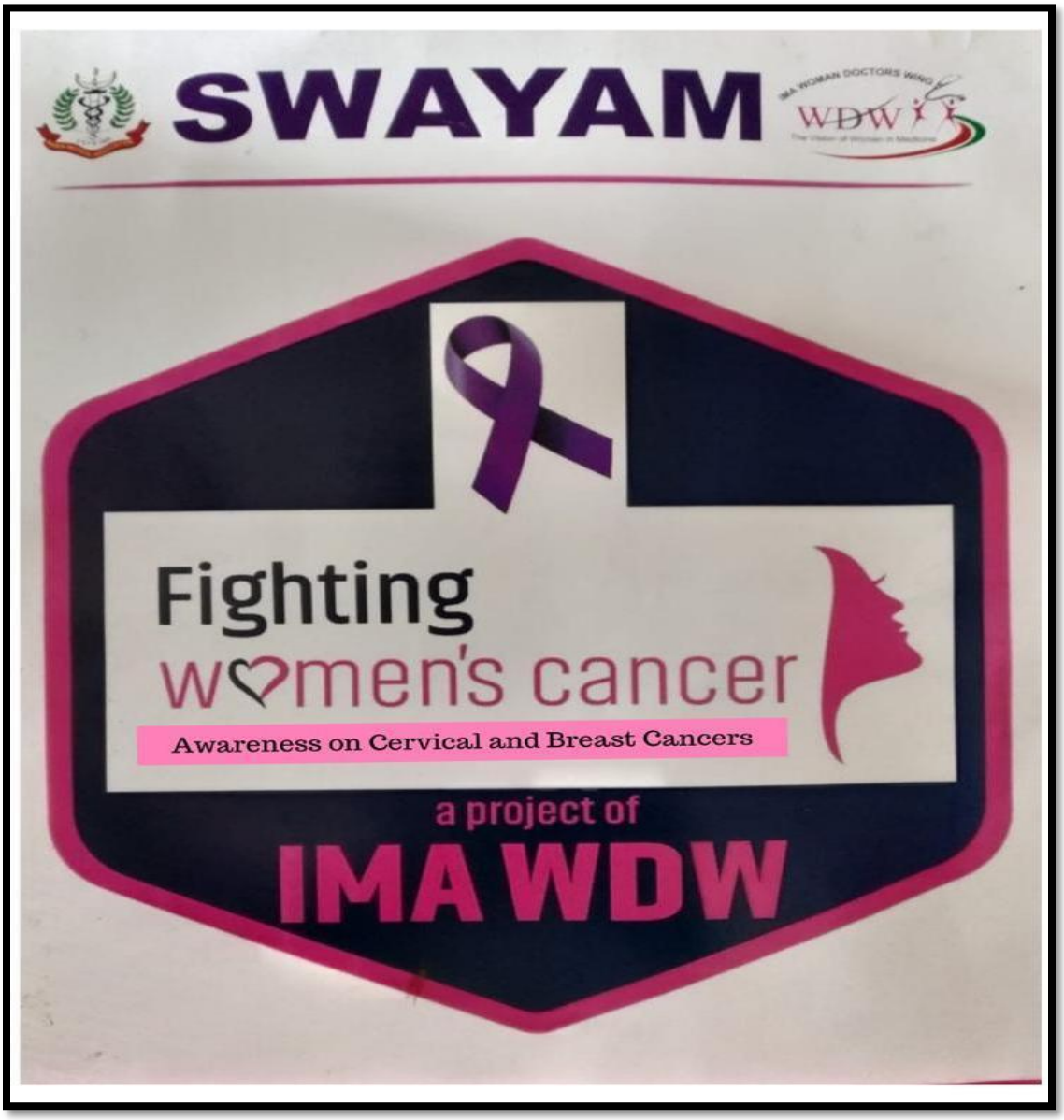
				Dose2-1 -2 Months Dose 3-6 Months
3	Nonavalent Vaccine	9-14 15-26	2 3	Dose1 -0 Dose 2-1-2 Months Dose 1-0 Dose2-1 -2 Months Dose 3-6 Months

CDC Weekly / December 16, 2016/65(49), 1405-1408

STEPS TAKEN BY PRIVATE SECTOR

The Indian Medical Association, the largest professional body with over 3.5 L doctors has launched a Women's cancer Awareness Programme through its

“Women Doctor’s Wing in 2021.




Dr. VEENA BALAMURUGAN, Physician
Dr. MALATHI, Ophthalmologist
Dr. G.P.THILAGAM, Physician
Dr. POORNIMA GOPI KRISHNA, Gynecologist
Dr. S.RAJESWARI, Consultant, Preventive Oncologist, Colposcopy Specialist
23-10-2021
சேதுபாஸ்கரா
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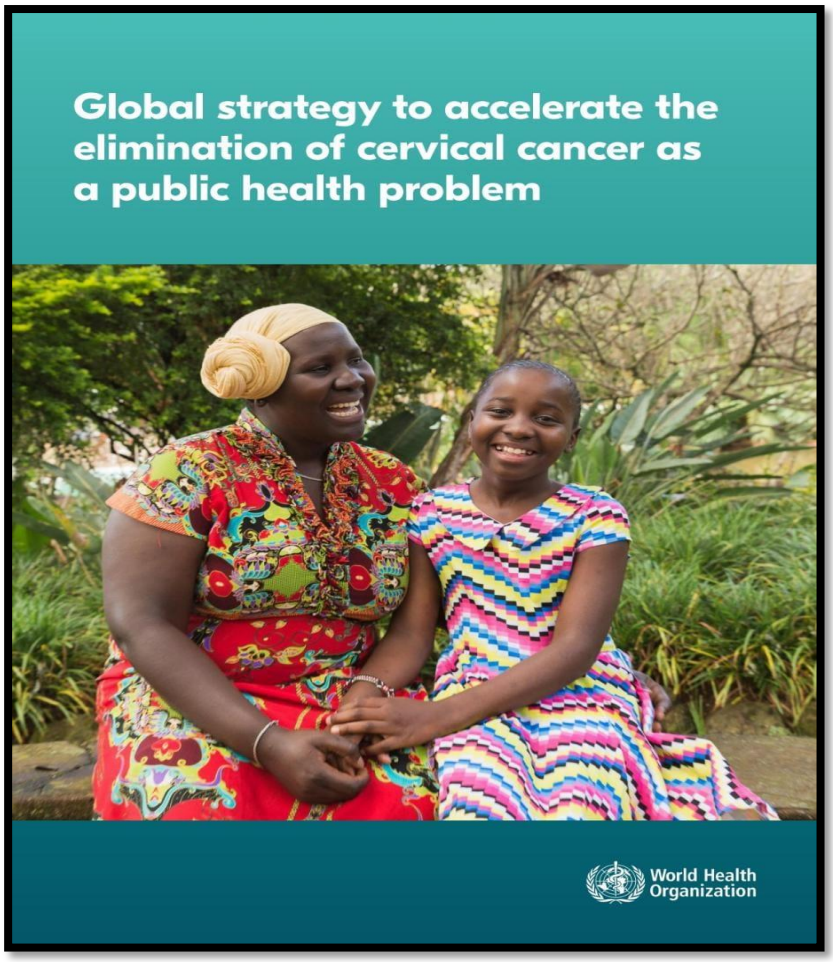
SOUTH INDIAN POST
Fighting
womens cancer
a project of
IMA WDW


SWAYAM
Fighting
IMA WDW



"Through cost-effective, evidence-based Interventions, including human papillomavirus Vaccination of girls, screening and treatment of Precancerous lesions and improving access to diagnosis and treatment of invasive cancers, we Can eliminate cervical cancer as a public health Problem and make it a disease of the past."

Dr Tedros Adhanom Ghebreyesus,
Director-General, World Health Organization

Hereditary Diffuse Gastric Cancer

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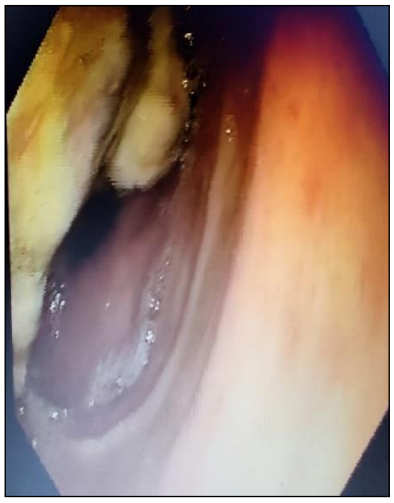


Hereditary diffuse gastric cancer (HDGC) is a rare inherited condition associated with an increased risk of **gastric (stomach) cancer**.

Mr. X. Bora. 42 years working at SSB, complains of pain abdomen with nausea. No H/O weight loss. No H/o melaena. His father died of gastric cancer at 55 yrs. of age. His uncle also died of esophageal cancer at an early date. On endoscopy big growth in the body of stomach seen. No active bleeding seen. Biopsy from the growth done shows- signet ring cell gastric carcinoma.
He is finally diagnosed as Hereditary Diffuse gastric carcinoma from the history and endoscopic finding. And referred to cancer institute for better treatment.

Diffuse gastric cancer is a specific type of stomach cancer, sometimes also called "signet ring cell gastric cancer" or "linitis plastic." The word "diffuse" is used because this cancer tends to affect much of the stomach, rather than staying in 1 area of the stomach. Approximately 20% of all stomach cancers are diffuse gastric cancers, and a small number of these are due to HDGC. The average age for someone with HDGC to be diagnosed with stomach cancer is 38, although it can be diagnosed much earlier or later than that. Gastric cancers not associated with this syndrome tend to occur in people older than 60. Women with HDGC also have an increased risk of lobular **breast cancer**. Some people with HDGC may also have had a cleft lip or cleft palate at birth, although the vast majority are unrelated to HDGC.

What causes HDGC?

HDGC is an inherited genetic condition that is rare. This means that the cancer risk and other features of HDGC can be passed from generation to generation in a family. The gene most commonly associated with HDGC is called CDH1. A mutation (alteration) in the CDH1 gene gives a person an increased risk of developing gastric cancer and other cancers associated with HDGC. Researchers believe that other genes, including CTNNA1, may be associated with HDGC. Studies are ongoing to learn more about all of these genes, and testing for inherited genetic mutations in these newer genes is currently not recommended except in clinical trials.

How is HDGC inherited?

Normally, every cell has 2 copies of each gene: 1 inherited from the mother and 1 inherited from the father. HDGC follows an autosomal dominant inheritance pattern, in which a mutation happens in only 1 copy of the gene. This is called a germline mutation. This means that a parent with a gene mutation may pass along a copy of their normal gene or a copy of the gene with the mutation. Therefore, a child who has a parent with a mutation has a 50% chance of inheriting that mutation. A brother, sister, or parent of a person who has a mutation also has a 50% chance of having the same mutation. However, if the parents test "negative" for the mutation (meaning each person's test results found no mutation), the risk to the siblings significantly decreases but their risk may still be higher than an average risk.

Options exist for people interested in having a child when a prospective parent carries a gene mutation that increases the risk for this hereditary cancer syndrome. Pre-implantation genetic diagnosis (PGD) is a medical procedure done in conjunction with in-vitro fertilization (IVF). It allows people who carry a specific known genetic mutation reduce the likelihood that the child will inherit the condition. A woman's eggs are removed and fertilized in a laboratory. When the embryos reach a certain size, 1 cell is removed and is tested for the hereditary condition in question. The parents can then choose to transfer embryos which do not have the mutation. PGD has been in use for over 2 decades and has been used for several hereditary cancer predisposition syndromes. However, this is a complex procedure with financial, physical, and emotional factors to consider before starting. For more information, talk with an assisted reproduction specialist at a fertility clinic.

How common is HDGC?

The specific number of families with HDGC is unknown. The overall incidence of gastric cancer varies in different parts of the world. In the United States, it is estimated that less than 1% of the population will develop stomach cancer of any kind; only a small fraction of these will be due to HDGC. The highest rates of gastric cancer in the world are in China, Japan, and other countries in Southeast Asia, as well as in Central and South America.

How is HDGC diagnosed?

Guidelines for the diagnosis of HDGC syndrome have been proposed, but may change over time as more is learned about this condition. Currently, the diagnosis of HDGC is suspected, and CDH1 genetic testing should be considered, if a person or family meets any of the criteria listed below:

- Families with 2 or more cases of stomach cancer, with at least 1 being diffuse gastric cancer
- A person diagnosed with diffuse gastric cancer before age 40
- Personal or family history of both diffuse gastric cancer and lobular breast cancer, if at least 1 person was diagnosed before age 50.
- Families with 2 or more cases of lobular breast cancer diagnosed before age 50
- A person diagnosed with multiple different lobular breast cancers before age 50
- A person with diffuse gastric cancer and a personal or family history of a cleft lip or cleft palate

Genetic testing for mutations in the CDH1 gene is available. However, only about 20% to 30% of families that appear to have HDGC will have a mutation found in the CDH1 gene. Therefore, both clinical and genetic aspects must be considered in counseling individuals about the potential for their family to have HDGC. Talking with a specialist who has training in genetic diseases and conditions, called a genetic counselor or geneticist, who is familiar with the syndrome is recommended.

What are the estimated cancer risks associated with HDGC?

Not everyone who inherits a gene mutation for HDGC will develop cancer. In people who have a mutation in the CDH1 gene, the lifetime risk for diffuse gastric cancer is estimated to be 67% to 70% for men and 56% to 83% for women by age 80. Women with a mutation in the CDH1 gene have about a 39% to 52% risk of developing lobular breast cancer by age 80. Although some earlier studies suggested a possible link to risks of colon and/or rectal cancer, the current findings suggest that there is likely no increased risk for these cancers in people with an inherited CDH1 mutation.

What are the options for reducing cancer risks associated with HDGC?

Given the increased risk of cancers associated with germline mutations in CDH1, it is recommended that people found to have this genetic mutation (called carriers) discuss the most appropriate strategies to reduce cancer risks with their health care team:

Stomach cancer: A baseline upper endoscopy exam (esophagogastroduodenoscopy or EGD) is recommended for carriers; previous studies have shown that screening endoscopy exams often miss early-stage diffuse gastric cancers.

Therefore, since endoscopic surveillance can be ineffective for preventing or detecting early-stage diffuse gastric cancers, individuals with a germline CDH1 mutation should consider having their stomach surgically removed, also known as prophylactic total gastrectomy, even if their endoscopy is normal. This type of surgery is the only proven effective way to prevent diffuse gastric cancer in individuals with HDGC. However, it is important to know that surgical removal of the stomach results in permanent changes to the digestive tract and can be associated with long-term side effects. It is very important for each patient to talk with their doctor about what tests and procedures would be appropriate for their individual care.
People who decide not to have surgery to remove the stomach may consider an intensive surveillance schedule with their doctor, with an annual EGD with multiple (more than 30) mucosal biopsies. Both the surgical and endoscopic management of people with known or suspected HDGC are best performed in centers with expertise in care of people with this syndrome.

- Additional screening for women: Women at risk for HDGC are at high risk for lobular breast cancer and should talk with their doctor about breast cancer screening options at the age of 30, or 10 years before the age of the youngest breast cancer diagnosis in the family. It is not yet clear what the best breast cancer screening strategy is for women with a CDH1 mutation. Screening options include:
- Monthly breast self-examinations
- Clinical breast examinations performed by a doctor or nurse every 6 months
- Regular breast imaging with mammograms, ultrasound, and/or a breast magnetic resonance imaging (MRI)

Since lobular breast cancer can be difficult to detect with a mammogram, breast MRI is recommended for breast cancer screening for women with a CDH1 mutation. Surgical removal of the breasts, called prophylactic mastectomy, is sometimes recommended to reduce a woman's risk of breast cancer. Women should discuss options for reducing their breast cancer risk with their doctors.

In conclusion, in this case if primary doctor who previously treated his father would screen the family members and advised to do the genetic testing then we could have probably saved the patient.

Ref- https://www.ncbi.nlm.nih.gov>pmc

THE EVOLUTION OF ECG MACHINE

Team CGP

It's a long history about ECG and its evolution from 1775 when a hen was brought alive with an electrical shock to its head by the Danish, Veterinarian and Physician to 2018 when smart phones recorded the electrical activities of the heart.

What was a primitive defibrillation to a hen, which gave the clue for the electrical activities of the heart, there were numerous, physiologist, physician (of course a separate cardiologist was not there at that time) were involved in designing the ECG machine and the waves of the present day. Every time the activities of the heart was demonstrated which was approved by peer group, leading to more innovation in the following years.

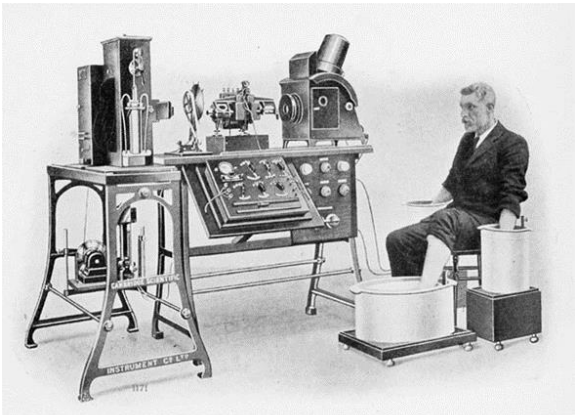
A Capillary electrometer was devised by Gabriel Lippmann which consisted of glass tube containing mercury (now mercury cannot be used as per the pollution norms) in inserted into sulphuric acid. The change in mercury levels during the contraction was recorded to find the electrical activities. This happened in 1873 and more cardiac electrical activities was demonstrated by Lungi Luciani, Wenckebach, Alfred Levis Galabin, in particular with Rheumatic heart diseases.

1887 saw the first tracing of the Electro Cardiogram of an intact human heart using the capillary electrometer which was done at St. Mary's Hospital in London in the presence of "Einthoven" by Augustus D Waller.

The postage stamp issued by the Netherlands in 1993 in honor of Einthoven. The ECG imprinted on his forehead was made with a galvanometer. It had become standard to label waves made by a galvanometer as PQRST. Note that Einthoven reverted to the use of PAB to label the waves in this particular ECG.

Willem Einthoven a Dutch Physiologist in credited to have invented the first practical ECG in 1895 went on to identify the cardiac activities with his sting galvanometer. He recordable the hypertrophy of Atria, Ventricles, the tachy and brady arrhythmias. He was also credited in introducing the 'U' wave in the ECG. Even at that time in 1905, he used cables to visualize the ECG at 1,500 meter away on March 22nd 1905.

1913 sir Thomas Levis demonstrated the atrial activities in association with the ventricles, enabling to identify atrial flutter which gave the importance to 'P' wave.



Photograph of a Complete Electrocardiograph, Showing the Manner in which the Electrodes are Attached to the Patient, In this Case the Hands and One Foot Being Immersed in Jars of Salt Solution

Holdemar Mobitz applied mathematical approach to analyse the arrhythmias. All these discoveries lead to Einthoven to be recognized with a Nobel Prize in Medicine in 1924. He is also credited naming PQRS waves, though it was accepted universally much later.

In 1932 Wolforth and Wood introduced the first precordial leads in Clinical Cardiology. It was Claude Sohaeffer back in 1947 successfully did the first Defibrillation to the Human heart.

With advancement of science and technology the present day ECG in Smart watches /phones has come into existence , 4 Lakes population was used as samples for getting the Apple Smart watch ECG monitor.

Internationally the top five producers are, GE health care, Philips Health Care, Nihon Kohden Corporation, Mindray medical international limited and Schiller Ag.

Biotechnology has become an indispensable part of Clinical medicine and ECG Machine has detected and saved human lifeS for the past four centuries. An incredible ECG Machine.

REPORT OF IMA CGP ASSAM STATE FACULTY

Total Membership strength as on date

Existing = 511 Nos.
New member enrolled = 3 Nos.
Grand Total = 514 Nos.

Total Sub-Faculty – 4 Nos.
Tezpur – Tinsukia – Hojai
North Lakhimpur is approved by the Governing Council meeting during the NATCON-21 held at 'PATNA' Bihar on 27th and 28th December'2021.

ACHIEVEMENT BY THE MEMBER OF THE IMA CGP ASSAM STATE FACULTY IN THE NATIONAL LEVEL.
Total 6(six) Nos. Life members of the State Faculty Awarded in different category by the National H.Qs during the award giving ceremony 'NATCON-2021' at Patna, Bihar, they are : -
1. Dr. Hemendra Kumar Borah, Director of Faculty- IMA CGP Dr. M.G. Bhide Memorial Award.
2. Dr. Satyajit Borah, President, IMA CGP State Faculty –
a) Best State President (National President's Appreciation Golden Star Award)
b) IMA National President's Appreciation Award for Cultural Activities.
c) Dr. B.R. Ramasubramanian Oration Award.
d) Young Academic Excellency Award on Teachers' Day
3. Dr. Sikha Sarma, Hony. State Secretary, IMA ASB cum Secretary State Faculty (Ex-officio)
a) Best State Secretary (National President's Appreciation Golden Star Award)
b) IMA Prof. Rajam Authilingam Award in Safe Motherhood Activities.
4. Dr. Upendra Nath Dutta of North Lakhimpur Branch – Best Local Branch President (National President's Appreciation Award)
5. Dr. Prerna Keshan of Tinsukia Branch – IMA Dr. Kanak Goel Award in Safe Motherhood Activity.
6. Dr. Hiranmoy Adhikari – Past CGP Dean. - Bangaigaon Branch – IMA National President's Appreciation Award for Life Long Service to IMA.

IMA ASSAM STATE BRANCH LIFE MEMBER -AWARD IN FOLLOWING CATEGORY

1. Dr. Surajit Giri – Sibsagar Branch Best Local Branch Secretary (National President Appreciation Award)
2. Dr. Kamal Narayan Kalita – Tezpur Branch IMA Dr. Ramachandra Moorthy Award in Psychiatry.
3. Dr. Chandan Chowdhury – Bongaigaon Branch – IMA Dr. Ketan Desai Yuba Leader Award
4. IMA Dr. D.S. Munagekar Award in Research – Dr. Gayatri Gogoi of Dibrugarh Branch

OTHER CATEGORY

1. IMA Dr. C.L. Jhaveri Safe Motherhood Activity Award for – Assam State Branch.
2. IMA Special Award for Organising Central Working Committee Meeting – Assam State Branch.
3. IMA Bulletin Award – IMA ASB Calling

ACTIVITIES

1. Privileged to attend as appointed speaker in the Awareness meeting of "World AIDS Day" on 01-12-2021 organised by IMA Tezpur Branch, District Health Society, Sonitpur and District Legal Cell, Sonitpur.
2. Attend National CGP hybrid Zoom meeting on 09-01-2022 and took part in discussion of the plan of action for the year 2022.
3. Attend Republic Day celebrations function organized by IMA Tezpur Branch and Tezpur Sub Faculty CGP.

Dr. Jagadis Basumatary
Hony Secretary, IMA-CGP
Assam State Faculty

Dr. Hemendra Kr. Borah
Director, IMA-CGP
Assam State Faculty

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