NEO Summit

23 April 2016- Jehan Numa Retreat, Madhya Pradesh, Bhopal

'Neo Summit' was held at Hotel Jehan Numa Retreat, Dr. Saleem Ali Road, Near Van Vihar, Bhopal, Madhya Pradesh. It was organized by of Asian Society of Continuing Medical Education.

The sole objective of the CME was to update practicing doctors about Thyroid diseases, Hyperthyroidism and its associated spectrum of complications and thyroid management as the disease is now highly visible across all societies within India.

The session was open with an introductory session by Asian Society of continuing Medical Education and followed by the scientific agenda.

Date	**:**	23 April 2016
Venue	**:**	Jehan Numa Retreat, Dr. Saleem Ali Road, Near Van Vihar, Bhopal, Madhya Pradesh, India.
Total Participants	**:**	41

EXPERTS

Dr. Madhukar Mittal
MBBS, MD (MEDICINE), DM (ENDO)

Dr. Sushil Jindal
MBBS, MD (Medicine), DM (Endocrinology)

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Dr. Sandeep Julka
M.B.B.S,M.D, DM (Endocrinology)

Dr.SachinChittawar
MBBS,MD(Medicine),DM (Endocrinology)

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Dr.BiplabBandyopadhyay
MBBS,MD(Medicine),DM(Endocrinology)

TOPICS :

1. **Subclinical Thyroid disorders, Hyperthyroidism and Hypothyroidism**
2. **Compare and Contrast -Graves Disease & Thyroid Disorder**
3. **Discussion: Interpretation of TFT – Case Scenario**
4. **Anti-Thyroid Drugs - How Long and How Much?**
5. **Thyroid Emergencies**
6. **Crisp Corner – Thyroid and Heart / Thyroid and Brain**
7. **Open Forum**

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Dr. Sushil Jindal talked about Discussion: Interpretation of TFT – Case Scenario. The informative session was highlighted upon: -

- Why focus on thyroid function tests
- Signs and symptoms provide the best indication to request thyroid tests
- Screening patients at increased risk?
- Which test should be used?
- Thyroid Nodule
- Limitations of thyroid function tests
- Thyroid Function Tests in Special Patient Populations

Dr. Sachin Chittawar discussed about Anti Thyroid Drugs. The scientific talk was deliberated on: -

- Anti thyroid drugs
- Thionamides
- Thionamides Inhibit Peroxidase-catalyzing Reactions
- Two treatment strategies for using ATDs
- Hypothalamic-Pituitary Thyroid Axis
- Thionamides And Immune System
- Immunomodulation v/s Euthyroidism
- Incidence of Major Toxic Reactions to ATDs In Adults
- Agranulocytosis
- Beta Blockers
- Thyrotoxicosis
- Choice of Therapy

Dr.Biplab Bandyopadhyaya discussed about Thyroid and Bone Health and Thyroid and Heart The scientific session was based on: -

- Thyroid Gland
- Normal bone remodeling
- Hormonal regulation of normal bone homeostasis

- Endocrine glands exert their actions at remote locations
- They have local effects too
- They act through receptors- nuclear and cytoplasmic
- Thyroid Hormone & Cardiovascular Hemodynamics
- Heart Failure
- Types of Lipid Abnormalities in Patients With Hypothyroidism
- Molecular actions of thyroid hormones in bone tissue

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Dr. Madhukar Mittal Discussed about Subclinical Thyroid disorders Hyperthyroidism and Hypothyroidism .The Scientific Session Was Based On:-

- Introduction Of Thyroid gland
- What is Subclinical Thyroid Disease?
- What is Subclinical Hyperthyroidism?
- Epidemiology of Subclinical Hyperthyroidism
- Clinical features of Subclinical Hyperthyroidism
- Diagnosis of Subclinical Hyperthyroidism
- Cardiovascular Consequences of Subclinical Hyperthyroidism
- Subclinical Hyperthyroidism Management
- Risk factors for Subclinical Hypothyroidism

Dr. Sushil Jindal Discussed about Compare and contrast - Graves Disease & Thyroiditis. The Scientific Session Was Based On :-

- Both Graves' disease and chronic thyroiditis (Hashimoto's thyroiditis) are autoimmune diseases of thyroid gland. Graves' disease is caused by stimulation of TSH receptor located on the thyroid gland by an antibody, which is known as TSH receptor antibody (TRAb)

- Furthermore, this may lead to hyperplasia and hyperfunction of the thyroid gland. On the contrary, the cause of Hashimoto's thyroiditis is thought due to a TSH stimulation-blocking antibody (TSBAb) which blocks the action of TSH hormone and subsequently brings damage and atrophy to thyroid gland. Approximately 15-20% of patients with Graves' disease had been reported to have spontaneous hypothyroidism resulting from the chronic thyroiditis (Hashimoto's disease).

- Pathogenesis for chronic thyroiditis following anti-thyroid drug treatment in patients with Graves' disease remains unclear. It has been estimated that chronic thyroiditis or Hashimoto's disease, which occurs following the Graves' disease episode is due to extended immune response in Graves' disease.

- It includes the immune response to endogenous thyroid antigens, i.e. thyroid peroxidase and thyroglobulin, which may enhance lymphocyte infiltration and finally causes Hashimoto's thyroiditis. We report four cases of chronic thyroiditis (Hashimoto's disease) in patients who have been previously diagnosed with Graves' hyperthyroidism. In three cases, Hashimoto's thyroiditis occurs in 7 to 25 years after the treatment of Grave's disease; while the other case has it only after few months of Grave's disease treatment.

- The diagnosis of Hashimoto's disease (chronic thyroiditis) was based on clinical manifestation, high TSHs level, positive thyroid peroxidase antibody and thyroglobulin antibody, and supported by positive results of fine needle aspiration

biopsy. Moreover, the result of histopathological test has also confirmed the diagnosis in two cases. All cases have been successfully treated by levothyroxine treatment.

Dr. Sandeep Julka Discussed About Thyroid Emergencies, The Scientific Session Was Based On:-

- Thyroid storm is a rapid decompensation of severe hyperthyroidism which can best be described by the three criteria of hyperthermia, tachycardia and altered mental state with severe agitation. There has to be a precipitating factor such as infection, iodine contamination, surgery or even I-131 treatment. Severe hyperthyroidism not fulfilling the criteria of thyroid storm can also be an indication for emergency treatment, particularly in the elderly with heart disease. Suppressed serum TSH and elevated free T4 levels are essential to confirm the diagnosis. When rapidly available, radioiodine uptake of the thyroid can be useful. Therapy aims at rapidly reducing the active circulating hormone pool, hypermetabolic state, tachycardia, and finally hormone synthesis. Thyroid secretion can be blocked by ioipanoic acid or ipodate while hypermetabolic state can be reduced with beta-blockers or calcium channel-blockers. Treatment of hyperthyroidism in patients with iodine contamination is a real therapeutic challenge. Myxoedema coma, a complication of severe hypothyroidism, is defined by hypothermia (rectal temperature less than 36 degrees C), bradycardia, slow mentation, precipitating factor such as infection or drug overdose, and increased serum creatine phosphokinase levels. Diagnosis of severe hypothyroidism should be confirmed by serum measurements of TSH and free T4. Treatment consists of general supporting measures including rewarming, correction of serum electrolyte disturbances, and adequate alimentation. Thyroid hormone treatment should initially be aggressive using either 300-400 micrograms of T4 or 20-40 micrograms of T3 intravenously. Cortisone therapy may be added. Patients should be under close monitoring as arrhythmias and myocardial infarction are frequent complications of myxoedema coma and/or its treatment with thyroid hormones.

Post completion of all the scientific talks of the faculties. Dr. Sushil Jindal went through the closing remarks. After an interesting and interactive open forum vote of thanks were conveyed by Asian society of Continuing Medical education.

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Antithyroid drugs- How long and How much?
Dr Sachin Chitnavar