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Pharma Tab

Department of Pharmacy Practice

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Alzheimer's disease

Alzheimer's disease is a progressive brain disorder that damages and eventually destroys brain cells, leading to memory loss and changes in thinking and other brain functions. It usually develops slowly and gradually gets worse as brain function declines and brain cells eventually wither and die. Ultimately, Alzheimer's is fatal, and currently, there is no cure.

Hallmark changes of Alzheimer's

- Plaques, microscopic clumps of a protein fragment called beta-amyloid
- Tangles, twisted microscopic strands of the protein tau (rhymes with "wow")
- Loss of connections among brain cells responsible for memory, learning and communication. These connections, or synapses, transmit information from cell to cell.
- Inflammation, triggered by the body's immune system
- Eventual death of brain cells and severe tissue shrinkage

Symptoms of Alzheimer's

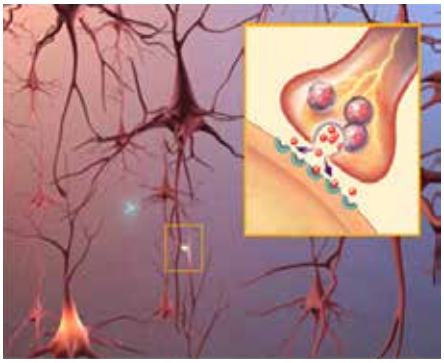
The most common early symptom is difficulty remembering newly learned information because Alzheimer's changes typically begin in the part of the brain that affects learning. As Alzheimer's advances through the brain it leads to disorientation; mood and behavior changes; deepening confusion about events, time and place; unfounded suspicions about family, friends and professional caregivers; serious memory loss and behavior changes; and difficulty speaking, swallowing and walking.

FDA-approved drugs

Drug name	Brand name	Approved For	FDA Approved
donepezil	Aricept	All stages	1996
galantamine	Razadyne	Mild to moderate	2001
memantine	Namenda	Moderate to severe	2003
rivastigmine	Exelon	All stages	2000
tacrine	Cognex	Mild to moderate	1993

The U.S. Food and Drug Administration (FDA) has approved five medications (listed below) to treat the symptoms of Alzheimer's disease.

How Alzheimer's drugs work



Neurons are the chief cells destroyed by Alzheimer's disease.In the brain, neurons connect and communicate at synapses, where tiny bursts of chemicals called scary information from one cell to another.

Current FDA-approved Alzheimer's drugs support this communication process through two different mechanisms:

1) Cholinesterase inhibitors work by slowing down the process that breaks down a key neurotransmitter. Donepezil,galantamine,rivastigmine andtacrine are cholinesterase inhibitors.

2) Memantine, the fifth Alzheimer's drug, is an NMDA (N-methyl-D-aspartate) receptor antagonist, which works by regulating the activity of glutamate, an important neurotransmitter in the brain involved in learning and memory.

Future treatment breakthroughs

Researchers are looking for new ways to treat Alzheimer's. Current drugs help mask the symptoms of Alzheimer's, but do not treat the underlying disease or delay its progression. There are several promising drugs in development and testing, but we need more volunteers to complete clinical trials of those drugs and increased federal funding of research to ensure that fresh ideas continue to fill the pipeline.1

10 WARNING SIGNS OF ALZHEIMER'S

1. Memory loss that disrupts daily life. One of the most common signs of Alzheimer's, especially in the early stages, is forgetting recently learned information.
2. Challenges in planning or solving problems. Some people may experience changes in their ability to develop and follow a plan or work with numbers.
3. Difficulty completing familiar tasks at home, at work or at leisure.People with Alzheimer's often find it hard to complete daily tasks.
4. Confusion with time or place.People

with Alzheimer's can lose track of dates, seasons and the passage of time.

5. Trouble understanding visual images and spatial relationships. For some people, having vision problems is a sign of Alzheimer's. They may have difficulty reading, judging distance and determining color or contrast.
6. New problems with words in speaking or writing.People with Alzheimer's may have trouble following or joining a conversation. They may stop in the middle of a conversation and have no idea how to continue or they may repeat themselves.
7. Misplacing things and losing the ability to retrace steps.A person with Alzheimer's disease may put things in unusual places.
8. Decreased or poor judgment.People with Alzheimer's may experience changes in judgment or decision making.
9. Withdrawal from work or social activities.A person with Alzheimer's may start to remove themselves from hobbies, social activities, work projects or sports.
10. Changes in mood and personality. The mood and personalities of people with Alzheimer's can change. They can become confused, suspicious, depressed, fearful or anxious.2

Reference

1. Alzheimer'sAssociation.2014. Available from: http://www.alz.org/research/science/alzheimers_research.asp
2. http://www.alz.org/national/documents/checklist_10signs.pdf

Neuroendocrine Tumors (NETS)

A neuroendocrine tumor is a tumor of the neuroendocrine system. The neuroendocrine system makes chemical messengers called hormones, which regulate the workings of different organs in the body. Neuroendocrine cells are spread throughout the body in organs such as the stomach, bowels and lungs.

NETs can be non-cancerous (benign) or cancerous (malignant). This information is about malignant tumors and is mainly about NETs affecting the digestive system.

NETs are classified according to where the cancer started (where the primary tumor is) in the body.

For example:

- small bowel NETs
- large bowel NETs
- appendiceal NETs
- pancreatic NETs
- gastric NETs
- lung NETs.

Rarely, NETs are found in other areas, including the liver, gallbladder, bile ducts, kidneys, ovaries or testicles. You may hear some NETs referred to as carcinoid tumors.

NETs often grow slowly, and it may be several years before symptoms appear and the tumor is diagnosed. However, some NETs may be fast-growing and more likely to spread to surrounding tissues, and to other parts of the body.

CAUSES AND RISK FACTORS

Neuroendocrine tumors are uncommon. As with many other forms of cancer, the exact cause is unknown.

These tumors are most likely to affect people over the age of 60. People who have a rare condition called Multiple Endocrine Neoplasia have an increased risk of developing a NET.

SIGNS AND SYMPTOMS

Symptoms depend on the location of NET in the body.

If in the digestive system, it may cause pain or discomfort in the abdomen, nausea, vomiting, change in the bowel habits.

If in the lung, it may cause chest infections and shortness of breath, cough, cough with blood.

NETs that cause symptoms of carcinoid syndrome

Some NETs (more commonly NETs of the small bowel, large bowel or appendix) may overproduce serotonin leading to carcinoid syndrome.

Carcinoid syndrome:

- diarrhoea
- flushing of the skin
- wheezing (similar to asthma)
- loss of appetite
- weight loss.

People with carcinoid syndrome may be advised to avoid things that may trigger flushing, such as drinking alcohol or eating spicy food.

Other NETs that cause symptoms

There are a number of other NETs that can cause specific symptoms due to the overproduction of hormones. These include insulinomas, gastrinomas, glucagonomas, VIPomas and somatostatinomoas.

Insulinomas

These can occur in any part of the pancreas. In people with an insulinoma, the tumor produces an abnormally high level of insulin, which causes low blood-sugar levels (hypoglycaemia).

A low blood-sugar level may cause symptoms such as:

- headaches
- confusion
- trembling and palpitations
- anxiety
- eyesight changes
- fits (seizures)
- feeling weak.

A low blood-sugar level is most likely to occur first thing in the morning, when exercising or after missing a meal. You can often raise your blood-sugar level again by eating or by having a sugary drink.

Gastrinomas

Gastrinomas usually start in the pancreas or duodenum. They may produce too much gastrin. Gastrin is a hormone that causes gastric acid to be made. High levels of gastric acid can lead to ulcers in the stomach, oesophagus and the small bowel. There may be several ulcers, which often don't respond well to the usual ulcer medicines. This is often called Zollinger-Ellison syndrome.

Common symptoms include:

- bleeding into the stomach
- a hole (perforation) in the wall of the stomach or small bowel
- tummy cramps or feeling bloated (due to narrowing of part of the bowel)
- diarrhoea

- pale, greasy and offensive-smelling stools (steatorrhoea)
- soreness and tightness in the oesophagitis.

Glucagonomas

These tumors occur most often in the pancreas. They usually produce too much glucagon, which is a hormone that helps control blood-sugar levels.

Common symptoms of a glucagonoma include:

- anaemia (a low level of red blood cells)
- weight loss
- high blood-sugar (diabetes)
- a skin rash
- blood clots.

Vipomas

These usually occur in the pancreas. They may produce too much of a substance called vasoactive intestinal peptide.

Common symptoms include:

- watery diarrhoea
- low levels of potassium as a result of the diarrhoea
- feeling weak and tired
- feeling sick (nauseated) and being sick (vomiting).

somatostatinomas

Somatostatinomas are extremely rare tumors that usually occur in the pancreas or parts of the small bowel (the duodenum or jejunum). They produce extra somatostatin.

Common symptoms of a somatostatinoma include:

- pale, greasy and offensive-smelling stools (steatorrhoea)
- weight loss
- anaemia (a low level of red blood cells)
- pain in the affected area
- diarrhoea
- high blood-sugar (diabetes).

NETs that don't cause symptoms

Some tumors don't overproduce hormones and may not cause symptoms. These are known as non-functioning NETs. They may be discovered by chance during an operation or a test being carried out for other reasons.

DIAGNOSIS

Urine tests

Some NETs cause an increase in serotonin. Serotonin is broken down by the liver into 5-Hydroxyindoleacetic acid (5HIAA) and then passed out of the body in the urine.

This test looks for raised levels of 5HIAA in the urine in a 24-hour period.

Blood tests

The levels of serotonin and chromograninA (CgA), may be raised in NET. Full blood count or FBC is evaluated.

Biopsy, Ultrasound , CT, MRI, Radioactive, PET/CT scan is used for diagnostic purpose.

STAGING AND GRADING

Grading

Grading refers to the appearance of tumor cells under a microscope.

Low-grade (grade 1) means that the cells look very much like normal cells. They are usually slow-growing.

Moderate-grade tumors (grade 2) fall somewhere in between low-grade and high-grade tumors in their appearance and behaviour.

High-grade tumors (grade 3) have cells that look very abnormal (sometimes called anaplastic or poorly differentiated NET). They are likely to grow more quickly and are more likely to spread.

Staging

The stage of a cancer is a term used to describe its size and whether it has spread beyond its original site.

Localised The cancer is contained inside the organ where it started.

Regional spread The cancer cell has grown through the wall of the organ into nearby tissues. It may also have spread to nearby lymph nodes.

Distant spread (metastatic) spread to other parts of the body, such as the liver, bones or lungs.

TREATMENT

Surgery

Somatostatin analogues

Somatostatin analogues are drugs used to treat the symptoms of carcinoid syndrome and can help reduce flushing and diarrhoea. They controle the production of harmones and control the growth of NETs.

The most commonly used somatostatin analogues are octreotide (Sandostatin®, Sandostatin Lar®) and lanreotide (Somatuline® LA, Somatuline Autogel®).

Chemotherapy

Targeted therapies

sunitinib (Sutent®) and everolimus (Afinitor®). These drugs are taken as capsules or tablets.

Interferon alpha (IntronA®)

It stimulate the body's immune system to destroy cancer cells.

Radiotherapy

Radiotherapy treats cancer by using high-energy x-rays to destroy cancer cells. It may be given externally from a radiotherapy machine (called external beam radiotherapy), or internally by placing radioactive material close to the tumor (called brachytherapy).

Targeted radionuclide therapy

This may be used to relieve symptoms of carcinoid syndrome and control the growth of NETs. A radioactive substance is taken as a drink or injected into the bloodstream. Almost all of the radiation is absorbed by the tumor, and very little goes to normal healthy tissues. This is why it's called targeted radionuclide therapy.

Hepatic artery embolisation

This may be used to shrink NETs in the liver and relieve the symptoms of carcinoid syndrome. It works by reducing blood flow to the tumors by formation of emboli with the help of drugs.

Radiofrequency ablation

Use of laser or radio waves to destroy the cancer cells by heating them to high temperatures.

REFERENCES

This information has been compiled using information from a number of reliable sources, including:

- DeVita, et al. Cancer – Principles and Practice of Oncology. 8thedition. 2008. Lippincott Williams and Wilkins.
- Oberg K, et al. Neuroendocrine gastroenteropancreatic tumors: ESMO Clinical Practice Guidelines for diagnosis, treatment and follow-up. Annals of Oncology. 2010. 21:5 223–227.
- Ramage J, et al. Guidelines for the management of gastroenteropancreatic neuroendocrine (including carcinoid) tumors (NETs) including carcinoid tumors. Gut. 2012. 61:1 6-32.

Recent Clinical Trials on Alzheimer's Disease

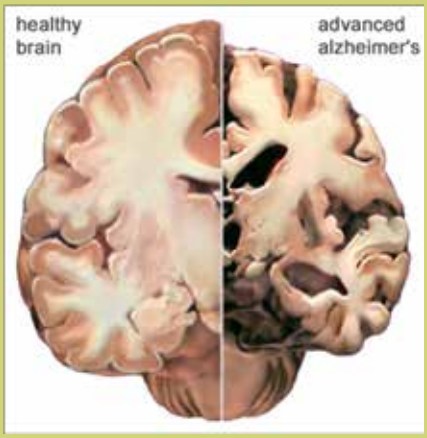
There are several clinical trials under progress regarding Alzheimer's Disease and associated neurological conditions (such as stroke, dementia).

Clinical Trial ID	Study title	Groups/Cohorts	Study Type/design	Country
NCT02205424	Cognition And Neocortical Volume After Stroke (CANVAS)	IschaemicStroke Alzheimer's Disease Vascular Dementia	Observational/Case Control	Australia, Victoria
NCT01926691	Predictors for Post-stroke Outcomes: Tel Aviv Brain Acute Stroke Cohort Acute Stroke Cohort (TABASCO)	Acute First-ever Stroke Patients over 50 years and without pre-stroke dementia, displaying an ischemic first-ever stroke or transient ischemic attack (TIA), onset within the last 72 hours	Observational Model: Cohort Time Perspective: Prospective	Israel
NCT01366027	PRISM Registry: Pseudobulbar Affect Registry Series	Alzheimer's Disease, Amyotrophic Lateral Sclerosis, Multiple Sclerosis, Parkinson's Disease, Stroke, and Traumatic Brain Injury.	Observational/ Probability sampling	United States, California
NCT00001235	Genetic Studies in Alzheimer's Disease	Alzheimer's Disease Nervous System Disease	Observational	United States, Maryland
NCT00088387	Effect of Lithium and Divalproex in Alzheimer's Disease	Condition: Alzheimer Disease Intervention: Divalproex, Lithium	Interventional/ Endpoint Classification: Safety/Efficacy Study Primary Purpose: Treatment Phase II	United States, Maryland
NCT02164643	Longitudinal Study of Brain Amyloid imaGing in MEMENTO (MEMENTOAmyGing)	Alzheimer's Disease (AD) and Related Disorders Flutemetamol, Florbetapir	Interventional/ Allocation: Non-Randomized Intervention Model: Parallel Assignment Masking: Open Label Primary Purpose: Diagnostic	France

Some of the following trials are currently ongoing in various countries.
Reported by: Dr.D.Krishna Kumar, Professor& Head, Department of Pharmacy Practice, CLBM COPS.

Compiled by **Dr.D.Krishna Kumar**

BRAIN CHANGES IN ALZHEIMER'S DISEASE



Massive cell loss changes the whole brain in advanced Alzheimer's disease. The image shows a crosswise "slice" through the middle of the brain between the ears.

In the Alzheimer's brain:

- The cortex shrivels up damaging areas involved in thinking, planning and remembering.
- Shrinkage is especially severe in the hippocampus, an area of the cortex that plays a key role in formation of new memories.

Ventricles (fluid-filled spaces within the brain) grow larger.

FDA APPROVED DRUGS FOR NEUROLOGY

DRUGS APPROVED IN 2014

S. No	Drug Name	Brand	Indication	Manufacturer	Approved Month
1.	Suvorexant	Belsomra	For the treatment of insomnia	Merck	Aug-14
2.	Tasimelteon	Hetlioz	For the treatment of non-24-hour sleep-wake disorder in the totally blind	Vanda Pharmaceuticals	Jan-14
3.	Alemtuzumab	Lemtrada	Genzyme	For the treatment of relapsing multiple sclerosis	Nov-14
4.	Naloxegol	Movantik	AstraZeneca	For the treatment of opiod-induced constipation in adults with chronic non-cancer pain	Sep-14
5.	Droxidopa	Northera	Chelsea Therapeutics	For the treatment of neurogenic orthostatic hypotension	Feb-14
6.	Peginterferon beta-1a	Plegridy	Biogen IDEC	For the treatment of relapsing multiple sclerosis	Aug-14
7.	Topiramate	Qudexy XR	Upsher-Smith Laboratories	For the treatment of partial onset and primary generalized tonic-clonic seizures and Lennox-Gastaut Syndrome	Mar-14
8.	Oxycodone hydrochloride + naloxone hydrochloride extended-release tablets	Targiniq ER	Purdue Pharma	For the management of severe chronic pain	Jul-14
9.	Indomethacin;	Tivorbex	Iroko Pharmaceuticals	For the treatment of acute pain	February of 2014
10.	Oxycodone hydrochloride and acetaminophen extended release;	Xartemis XR	Mallinckrodt Pharmaceuticals	For the management of acute pain	Mar-14

Reference: https://www.centerwatch.com/drug-information/fda-approved-drugs/therapeutic-area/10/neurology

Compiled by **M.Ashok Kumar**

Public Awareness Program on Drug use

The students conducted a Public Awareness Program on 25.09.2014 to mark International Pharmacist Day.

Pamphlets were prepared on Proper Use of Medicines in both English and Tamil.

More than 200 students formed a human chain on the busy OMR Road.

They held placards depicting role of Pharmacists and Proper use of medicines.

Pamphlets in simple language which can be understood by general public were distributed to them.

Any queries put forth by the people were answered by the students.

Spinal Accessory Nerve Schwannomas Masquerading As A Fourth Ventricular Lesion

Dr. Shyam Sundar Krishnan

Shyam Sundar Krishnan, Sivaram Bojja, Madabhushi Chakravarthy Vasudevan.

Department of Neurosurgery, Achantha Lakshmipathy Neurosurgical Centre, Voluntary Health Services, Chennai, Tamil Nadu, India.

Dr. Vasudevan

Schwannomas are benign lesions that arise from the nerve sheath of cranial nerves. The most common schwannomas arise from the 8th cranial nerve (the vestibulo-cochlear nerve) followed by trigeminal and facial nerves and then from glossopharyngeal, vagus, and spinal accessory nerves. Schwannomas involving the oculomotor, trochlear, abducens and hypoglossal nerves are very rare. We report a very unusual spinal accessory nerve schwannoma which occupied the fourth ventricle and extended inferiorly to the upper cervical canal.

A 22-year-old man presented to us with raised intracranial pressure type of headache with obscuration of vision for about 6 months in duration. The patient complained of gradual loss of vision in the left eye since 3 months prior to presentation. Also, over the last month he has had severe gait ataxia and hence was unable to walk without support. His examination revealed florid papilledema in the right eye with concentric field cuts and in the left eye secondary optic atrophy with no perception of light.

MRI of the brain revealed a large contrast enhancing heterogeneous lesion that was hypo intense on T1 weighted image and hyper intense on T2 weighted image with areas of necrosis and calcification. The lesion was expanding the fourth ventricle causing obstructive hydrocephalus. It was found extending inferiorly through the foramen magnum into the upper cervical canal till C2 vertebra level displacing the medulla and cervical spinal cord anteriorly and to the right side.

The patient underwent a midline sub-occipital craniotomy, with removal of posterior arch of C1. The lesion was found arising from the trunk of the left spinal accessory nerve at the level of the foramen magnum. The tumor was variable in consistency, mostly firm and vascular with some areas of cystic degeneration. The lesion was extending from C2 and into the fourth ventricle below the vermis, pushing the upper cervical cord and cervicomedullary junction to the right. The accessory nerve was thinned out and draped over the lesion and a portion of it was embedded within. The lesion was excised along with the embedded segment of the nerve, preserving the uninvolved portion which was draped over the tumor.

The patient recovered postoperatively with no obvious sternocleidomastoid or trapezius weakness ascribable to the left accessory nerve partial excision at the time of surgery. His post-operative contrast CT scan showed no residual lesion with resolving hydrocephalus. On last follow up after 5 months no neurological deficit was noted and he was doing well.

Pathology report revealed benign schwannoma with hyper cellular (Antoni Type A pattern) and hypo cellular (Antoni Type B pattern) areas.

Discussion

Lower cranial nerve schwannomas are rare and when present are located in the jugular foramen most commonly arise from the glossopharyngeal or vagal nerves with accessory nerve involvement being least common. The other locations in the accessory nerve course where schwannomas arise are extracranial beyond the jugular foramen in the cervical region and cisternal proximal to the jugular foramen. Both of these are very rare locations.

The spinal accessory nerve arises from the lateral funiculus of the medulla spinalis as low as the fifth cervical nerve and unites to form a single trunk. It passes between the denticulate ligament and the dorsal roots of the spinal cord through the foramen magnum passing to and entering the jugular foramen. Here it lies in the same dural sheath as the vagus but separate and receives one or two fibers from the cranial part, which arises from the nuclear ambiguous in the medulla. On exiting the jugular foramen, the spinal accessory supplies the sternocleidomastoid and trapezius, whereas the cranial portion contributes to the pharyngeal, superior laryngeal, recurrent nerve and the cardiac nerves.

Intracranial accessory nerve schwannomas were classified into two types as intrajugular schwannomas and intracisternal schwannomas. The intrajugular variant grows into the cisterna magna whereas the intracisternal variant grows into the jugular foramen. The jugular foramen variant presents with jugular foramen syndrome with the hypoglossal and glossopharyngeal nerve involvement.

The lesion in our patient was located predominantly in the fourth ventricle but with a large extension unto C2 across the cervico-medullary junction. So far only one case has been reported in the fourth ventricular location. In spite of their origin from the accessory nerve, these tumors tend to be located in the midline and posteriorly, probably because the cisterna magna allows sufficient space for the tumor to expand. This makes it appear more as a midline lesion and occupy the cisterna magna or the fourth ventricle, as in our case, making it difficult to differentiate from other midline posterior fossa lesions.

Thus, the cisternal variant of accessory nerve schwannomas by virtue of their extension in to various cisternal spaces can be classified by location as: 1) Cisterna magna, 2) Cervical, 3) Cervicomedullary, 4) Fourth ventricular, 5) Craniovertebral junction. The intracisternal variant often present with neck stiffness, secondary to accessory nerve irritation.

Unlike other cranial nerve schwannomas, the accessory nerve may or may not present with nerve dysfunction. This is dependent on the extent of nerve involvement. Our patient presented predominantly with neck pain and no signs of accessory nerve dysfunction. Signs and symptoms of cerebellar involvement, due to extension into the vermis and associated obstructive hydrocephalus was the presentation in our patient.

The MRI appearances of these tumors usually have regular contours and a round or oval shape, lack edema, and enhance homogenously with contrast. Cystic degeneration is generally seen in large tumors and this may alter the otherwise uniform texture of the mass.

Total removal is the primary treatment of schwannomas arising from the spinal portion of the accessory nerve. Excision of an involved segment or rootlet along with the lesion sparing the rest of the nerve in continuity, rarely results in any neurological deficit. This is because the insidious tumor growth in a portion of the nerve causes functional compensation by the remaining intact uninvolved nerve segment. This was the case in our patient who had intact neurological function preoperatively and also postoperatively after removal of lesion leaving the remaining uninvolved nerve in continuity.

Conclusion

We have presented a rare case of an accessory nerve schwannoma in the fourth ventricle. The possibility of the same, as a differential diagnosis for midline posterior fossa and high cervical lesions is the highlight of our report.

Reference: Journal of neuroscience in rural practice, Volume 6 Issue 1 January-March 2014, page no: 112-115.

Free Patient Awareness Camp on Diabetes, Hypertension and Obesity

The department of Pharmacy Practice in association with The Voluntary Health Services organized Free Patient Awareness Camp on Diabetes, Hypertension and Obesity at Outpatient department of VHS Hospital, Tharamani, Chennai on December 8th, 2014.

Pharm.D students were actively involved in free screening of blood pressure, blood glucose level & BMI of patients and provided patient counseling along with their patient information leaflets. 110 persons were registered and screened during the camp.

ONE DAY SEMINAR ON

CURRENT PERSPECTIVES IN PHARMACOVIGILANCE AND DRUG SAFETY

Department of Pharmacy Practice
on 9th August, 2014 at Hotel Fortune Select Palms, Chennai.

The seminar started at 9.30 a.m and was inaugurated by our Vice-Chairman Mr.R.Srinivasan and Secretary & Correspondent Mr. LUday Metha.

The major topics covered under this seminar includes

1. The introduction to Pharmacovigilance: An extensive talk was given by Dr.Kishore Gnana Sam, Chief Clinical Pharmacist, College of Pharmacy, Gulf Medical University, He has given an insight to "Role of Clinical Pharmacist in ADR assessment".
2. Dr.C.Adithan, Senior Professor & Head, Department of Clinical Pharmacology, JIPMER, Pondicherry spoke on how to setup Pharmcovigilance Center in a clinical set up with the title "Pharmacovigilance in clinical practice and setting up of Pharmacovigilance centre"
3. Then Dr.Kalaiaselvan, Senior Scientific Officer, Indian Pharmacopeia Commission, Ministry of Health, New Delhi provided detailed information on Pharmacovigilance program of India- a step towards patient safety and their achievement in National Pharmacovigilance Program.
4. Dr. Deepa Raj, Executive Manager and her team from Accenture Ltd., Chennai provided the scientific information and the industrial perspectives of ADR monitoring, and 'Opportunities in Pharma outsourcing', 'Clinical Data Management' and 'Pharmacovigilance reporting system an overview'.

200 participants from various pharmacy colleges from Tamil Nadu, Pondicherry and Pharmacists working in pharmaceutical sectors, and staffs from Voluntary Health Service attended the seminar.

For details and feedback contact:
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