A Dissertation on

"A STUDY OF SUDDEN DEATH CASES WITH NO KNOWN HISTORY OF PRE-EXISTING DISEASES"

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APRIL 2017

CERTIFICATE

This is to certify that this dissertation titled "**A STUDY OF SUDDEN DEATH CASES WITH NO KNOWN HISTORY OF PRE-EXISTING DISEASES**" is the bonafide original work done by **Dr. N.KARTHIKEYAN,** Post Graduate in **Forensic Medicine** under overall supervision and guidance in the **Department of Forensic Medicine**, Government Kilpauk Medical College & Hospital, Chennai in partial fulfillment of the regulations of **The Tamil Nadu Dr. M.G.R. Medical University, Chennai**, for the award of **M.D. Degree in FORENSIC MEDICINE Branch –XIV.**

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DECLARATION

I, **Dr.N.KARTHIKEYAN** solemnly declare that the dissertation on **"A STUDY OF SUDDEN DEATH CASES WITH NO KNOWN HISTORY OF PRE-EXISTING DISEASES"** is a bona- fide work done by me during the period of September 2015 to August 2016 at Government Kilpauk Medical College and Hospital, under the expert Supervision of **Dr. R. SELVAKUMAR, M.D,** Professor and Head of Department of Forensic Medicine, Government Kilpauk Medical College, Chennai. This Dissertation is submitted to The Tamil Nadu Dr.M.G.R. Medical University towards partial fulfillment of the rules and regulations for the M.D. degree examinations in Forensic Medicine to be held in April 2017.

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CERTIFICATE

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INTRODUCTION

Sudden death can be defined as the sudden or unexpected death of a apparently normal healthy people from natural diseases. **Emphasis placed on the unexpected character rather than the suddenness of the death.** Suspicion usually arises when an individual is found dead and nobody witnessed it. An autopsy is desirable in persons covered by an insurance policy. **(2)**

As per WHO-Death is said to be sudden and unexpected when a person not known to have been suffering from any dangerous disease, injury or poisoning is found dead or dies within 24 hours after the onset of terminal illness. But some say sudden death as those occurring instantaneously or within one hour of onset of signs and symptoms. Simply sudden death is defined as sudden or unexpected death a apparently healthy individual from natural cause. It should be remembered that natural death means that the death was caused entirely by the nature of the disease, and trauma or poison did not play any part in bringing it about.**(2)**

In many occasions the individual may not die immediately but fight for few hours after the onset of terminal symptoms. Sometimes sudden deaths occur in older individual and generally under circumstances which arouse no suspicion. But such deaths in younger individuals are likely to cause suspicion. A Medical Officer should not issue death certificate in cases of sudden death without autopsy and so far its suddenness is expected, the doctor should report such case to the legal authorities for necessary investigation. **(3)**

Sudden death often results from:

- Diseases which gradually affect a vital organ without arising any noticeable symptoms until death like Coronary artery disease.
- Fatal bleeding from an Aortic aneurysm or aneurysm of cerebral arteries following a sudden rupture of a diseased blood vessel causing sudden death.
- Fulminating infectious disease without any recognizable symptoms like Lobar pneumonia, bacterial endocarditis etc.
- In cases of Volvulus, strangulated hernia, twisted ovarian cysts etc due to a shift in the position of viscus. **(4)**

The incidence of Sudden death is approximately 10 percent of all deaths. Where diseases of Cardiovascular system account for about 45-50%, diseases of Respiratory system 15-23%, diseases of Nervous system 10-18%, diseases of Alimentary system 6-8%, diseases of Genito-urinary system 3-5% and 5-10% are of miscellaneous causes. **(4)**

It is very arduous to make a comparison of the incidence of sudden death in various parts of the world because it widely varies in the prevalence of various diseases in different countries, environmental, socio economic and genetic factors. Apart from this, the range observed in the various reported incidence of sudden death in different studies may be due to various definitions of sudden death, inclusion criteria, exclusion criteria and age groups selected for the respective study. **(4)**

Establishing the cause of death in Sudden death cases is really a matter of challenge for an Autopsy Surgeon because the very purpose of Medico legal Autopsy in sudden death cases is to determine whether violence or poisoning has been in anyway responsible for the death. It has to be remembered that absence of external signs of injury does not rule out death from violence.

The age of the deceased, reliable information regarding past health, illness and above all the presence of witness at the onset of terminal symptoms is very much needed in deciding the real necessity for an autopsy.

In most of the cases due to celerity of events necessary emergency investigations like ECG, chest X-ray, etc. had not been performed. And above all due to lack of reliable witness or close relatives, in many cases, detailed clinical history could not be retrieved. **(4)**

AIMS AND OBJECTIVES

1. To know about the socio-demographic profile of the deceased of sudden death cases.

2. To know about the most predominant system of involvement (Cardio Vascular, Respiratory, Central Nervous, Genito Urinary) from the Post-Mortem Report.

3. To know about history of any substance abuse by the deceased, based on Inquest report and available hospital documents brought by the police.

4. To establish the cause of death based on Post Mortem examination (based on findings and cause of death mentioned in the post mortem certificate).

LITERATURE

In many countries where deaths have to be officially certified, the responsibility for certification falls either to the treating doctor who attended the patient during life or one who can reasonably be assumed to know sufficient of the clinical history to give a reasonable assessment of the cause of death. This is an ‘honest opinion, given fairly, but many studies have shown that there is a large error rate in death certificates and that in 25–60 per cent of deaths there are significant differences between the clinician’s opinion to arrive at the cause of death and the lesions or diseases actually displayed at the autopsy. **(5)**

Unfortunately, it seems that there has been very little or no improvement in the problems of certification of death over the years and as a result the minimal and raw epidemiological data gathered by national statistical bureaux must be treated with some caution. All doctors should take the task of certifying the cause of death very seriously. Sudden death can be defined as a death occurring suddenly are unexpectedly and not due to apparent trauma or intoxication or poisoning. **(5)**

The World Health Organization (WHO) definition of a sudden death is said to be occurs within 24 hours of the onset of symptoms, but in practice most of sudden deaths occur within minutes or even within seconds of the

onset of signs and symptoms. Indeed, it is very likely that a death that is delayed by hours will not be referred to the medical examiner or other medico-legal authority, as a diagnosis and a cause of death may well have been made, and a death certificate can be completed by the treating doctors.

It is important to remember that a case of sudden death is not necessarily unexpected and case of unexpected death is not necessarily to be sudden, but these two facets are often combined together. **(4)**

When an apparently healthy person dies under circumstances, when his death is not expected and dies suddenly, it is called **sudden death.** Ordinarily the terminal event is < 24 hours before death or the person is not seriously ill during the 24 hours before death. Because the death occurs all of a sudden, there is a possibility of suspicion of foul play. If the last attending physician is satisfied with the terminal event he may issue the death certificate. If he has any doubt or suspicion, then it becomes necessary for him to get an inquest made and proceed in the direction of performing an autopsy. **(4)**

CAUSES FOR SUDDEN NATURAL DEATH:

When a natural death occurs very rapidly, the cause is almost inevitably towards the cardiovascular system. Indeed, if a person fell down and collapses immediately and was clinically dead before bystanders can assist him, this can be only due to a cardiovascular event resulting in a cardiac arrest. Virtually no other mode of death occurs so quickly. Extra-cardiac causes, even that elsewhere in the cardiovascular system, are rarely s fatal. Of course, in all such discussions of this nature, ‘death’ must be defined and, for our present purposes, irreversible cardiac arrest is taken as the criterion of death. **(4)**

VARIOUS CAUSES FOR SUDDEN DEATH:

- Cardiac Causes.
- Respiratory Causes.
- Neural Causes.
- Alimentary Causes.
- Miscellaneous. **(4)**

Cardiac Causes:

- Coronary Arterial Disease: Acute lesions in the coronary arteries are plaque, fissures, platelet clumps, organizing thrombus, (coronary artery spasm).

- Left ventricular hyper-trophy, acute infarct, healing infarct, healed infarct, ventricular dilation.
- Infectious Myocarditis (viral).
- Hypertrophic cardiomyopathy.
- Aortic Stenosis.
- Pulmonary Hypertension etc. sometimes an apparently normal heart is seen. More than 75% of sudden involving two or more vessels.
- In some cases old scars of earlier infarction are seen. **(4)**

Respiratory Causes:

- Pulmonary embolism.
- Acute oedema of Glottis.
- Bronchial asthma.
- Pneumonia.
- Massive Haemoptysis.
- Spontaneous pneumothorax. **(4)**

Neural Causes:

- Vagal inhibition.
- Epilepsy. (Idiopathic or post-traumatic).
- Non-traumatic subarachnoid haemorrhage (Berry aneurysm rupture).
- Intracerebral (hypertension).
- Cerebral Infarction (ischaemic stroke).
- Primary Undiagonosed Brain Tumour.

- Meningitis. Meningococcemia (rapid **fulminant** death within 12 hours of onset of symptoms).
- Hydrocephalus. **(4)**

Alimentary Causes:

- Massive hematemesis (Oesophageal varices) gastro – intestinal haemorrhage (gastric/duodenal ulcer, erosion in artery).
- Strangulated Hernia.
- Perforation of Gastric/Duodenal ulcer.
- Fulminant haemorrhagic pancreatitis.
- Diabetic coma.
- Hepato-cellular failure and hepatic coma,
- Adrenal crisis (phaeochromocytoma).
- Rupture of spleen (Malaria). **(4)**

Vascular causes:

- Kawasaki Disease.
- Pulmonary Hypertension. **(4)**

Haematological:

- Acute leukaemia.
- Bleeding Diathesis.
- Sickle Cell Haemoglobinopathy. **(4)**

Gastro Intestinal:

- Gastro enteritis. **(4)**

Genito Urinary :

- Glomerulonephritis.
- Pyelonephritis. **(4)**

Metabolic Disorders:

- Diabetes Mellitus.
- Hypoglycaemia.
- Hyperthyroidism. **(4)**

Miscellaneous:

- Sickle cell disease (haemoglobinopathy).
- Haemochromatosis.
- Ruptured tubal pregnancy. **(4)**

Vagal Inhibition:

It is also called reflex death. In vagal inhibition, death occurs all of a sudden as a result of reflex vagal stimulation leading to cardiac inhibition. **(4)**

The causes of vagal inhibition are:

- Sudden fear / emotion.
- Injury to trigger areas.
- Carotid body and carotid sinus in the neck.
- Solar plexus (pit of the stomach).
- Precordium.
- Tympanic membrane.
- Other serous membranes.
- Cervix uteri.
- Testes.
- Urethra. **(4)**

Stimulation of the trigger area brings about cardiac inhibition. Even a trifle injury or **micro trauma** to the receptor nerve endings in these trigger areas leads to death. The vaso vagal reflex is may be some pallor and death due to sudden cardiac asystole. At autopsy, no organic disease is discovered. The autopsy findings are negative. Vagal inhibition is an example of functional death. The circumstances of death can be application of pressure on the neck, foreign bodies in the air passages (e.g. sudden impaction of food in the glottis), puncture of pleural cavity, passing urethral sound, dis-tension of hollow organs (e.g. attempt at criminal abortion through passing instruments into cervix, fluids introduced into uterus), acute myocardial infarction causing ischaemic denervation of SA node. An opinion of vagal inhibition as to the cause of death should be given after excluding all the other possible causes of death. **(4)**

Sudden death due to Sympathetico-Adrenal Stimulation (SASA)

Reflex death due to vagal inhibition is well known. However, sudden fatal circulatory failure due to sympathetico-adrenal stimulation is less stressed. In sympathetic adrenal stimulation, there is an increased myocardial and ventricular fibrillation. Acute ventricular fibrillation gives rise to rapid death. This sympathetic death takes several minutes to develop in comparison to vagal inhibition which occurs instantaneously. Before death patient shows features of hyper acute cardiovascular failure e.g. dyspnea, pulmonary oedema etc. are seen.

At autopsy, coronary artery sclerosis, fatty changes in myocardium, chronic valvular disease may be seen, marked pulmonary oedema is seen. Pleural petechial haemorrhages are seen. **(4)**

Difference between Sympathetico-Adrenal Stimulation and Vagal Inhibition.

Sympathetico-Adrenal Stimulation	Vagal Inhibition
Cardiac disease may be present. Myocardial irritability occurs (Ventricular fibrillation). Pulmonary oedema. Pulmonary congestion. Evidence of hyper acute congestive cardiac failure. Cyanosis Intravenous injection of adrenaline.	Apparently healthy. Reflex death. Instantaneous. Negative autopsy. Functional death. Micro trauma or no evidence of trauma. Stimulation of urethra, cervix, precordium, other synovial membrane, testes etc. **(4)**

DISEASE OF THE HEART:

Most sudden unexpected deaths (SUDs) are caused by disease of the cardiovascular system. Although there is a huge geographical variation, due to the remarkable differences in the recorded incidence of atherosclerosis, the

prime causes of SUD usually lies in the heart itself. The following lesions are the most obvious. **(4)**

CORONARY ARTERY DISEASE:

Coronary stenosis from narrowing of the lumen by atheromatous plaque may lead to chronic ischaemia of the muscle which is supplied by that coronary vessel. If the myocardium becomes ischaemic, it may also vulnarable to electro-rhythmical abnormalities and liable to develop cardiac arrhythmias. The oxygen requirement of the myocardium is dependent upon the heart rate and so anything that increases the heart rate (exercise, a large meal or a sudden adrenaline response to fear or to anger, stress or other emotions) will lead to a sudden increase in the requirement of oxygen. **(3)**

If these cannot be met due to the restriction of the blood flow through the occluded and stenotic vessel, the myocardium distal to the stenosis will become ischaemic. There is no need for this ischaemia to produce a myocardial infarct. It just has to be sufficiently severe to initiate serious cardiac arrhythmias and, if the region becomes ischaemic includes one of the pace-making nodes or a major branch of the conducting system, the risk of electrical rhythm abnormalities will develop is greatly increased.

Complications of atheromatous plaques may worsen the coronary stenosis and produces subsequent myocardial ischaemia. Bleeding may occur into atheromatous plaque and this can be seen as sub intimal haemorrhage at autopsy. **(3)**

Sudden increase in the size of the plaque may lead to rupture, which may also occur if the plaque get ulcerated. When a atheromatous plaque ruptures, the extruded fat, cholesterol and fibrous debris will be pushed downstream in the coronary artery and lodged distally, often causing multiple mini-infarcts. The endothelial cap of a ruptured atheromatous plaque may act as a flap valve within the vessel and cause a total obstruction of the vessel. An atheromatous plaque is a site for the development of mural thrombus, which will further narrow the lumen of the vessel lumen without necessarily fully occluding the vessel. Coronary thrombosis is commonly over-diagnosed by physicians as a cause of sudden cardiac death, and less than one-third of sudden cardiac deaths reveal macroscopic or microscopic changes of coronary thrombus at autopsy. **(3)**

The simple stenosis and the complications of atheroma are both sufficient to cause death and much more common. However, coronary thrombosis is still a frequent finding at autopsy and it will be associated with an area of myocardial infarct, providing there has been a sufficiently long period of survival for the macroscopic changes of infarction to develop. Myocardial infarction occurs when there is a complete occlusion or severe stenosis of a coronary artery so that the blood supply to that area is insufficient to maintain the oxygen perfusion of the myocardium. However, if there is a sufficient collateral circulation, blood can still reach the myocardium through the collateral vessels. The serious effects of a myocardial infarction may appear at

any time after the muscle has become ischaemic due to lack of blood supply. **(3)**

The area of muscle damaged by a myocardial infarction is again weakened by the process of cellular death and the inflammatory response to these ischemic cells. The area of the myocardial infarct is weakest between 3 days and7 days after the clinical onset of the infarction and it is at this time that the weakened area of myocardium may yield to rupture and cause sudden death from a haemo pericardium and cardiac tamponade. The myocardial rupture occasionally occurs through the inter ventricular septum, resulting in a left–right shunt. If a papillary muscle is infarcted, it may rupture, which will allow part of the cardiac valve to prolapse, which may be associated with sudden death or may present as a sudden onset of valvular insufficiency. An infarct heals by the process of fibrosis, and fibrotic plaques in the wall of the ventricles or septum may interfere with the physical (ejection) or electro-rhythmical function of the heart. **(3)**

Cardiac aneurysms may develop at sites of infarction; they may calcify and they get easily ruptured. Physical lesions in the cardiac conducting system have been studied elaboratively in recent times, especially in relation to sudden cardiac death. Many different abnormalities have been found, varying from extensive fibrosis to haemorrhage, tumours and infective lesions. It may be very difficult to ascertain if such lesions are the cause of the fatal cardiac arrhythmias or merely an incidental finding, but in the absence of any other

abnormality, it is reasonable to arrive that they were a significant factor in causing the death. **(2)**

HYPERTENSIVE HEART DISEASE

Hypertensive heart diseases causes sudden cardiac death from left ventricular hypertrophy. The upper limit of normal heart weight is about 400 g (although this depends greatly on the body size and weight) and this may increase to 600 g or more, reflecting the increased thickness of the left ventricle. However, blood can only flow through the coronary arterioles during diastole because they are compressed during systole. At rest, when diastole is relatively long, the whole of the myocardium can be adequately perfused, but if the heart rate increases, diastolic time is reduced and the perfusion of the sub endocardial cells is reduced. These cells become unstable and irritable and may produce arrhythmias and fibrillation. Atheroma is often associated with hypertension so that the enlarged heart may also be deprived of a normal blood flow in the major coronary vessels by the presence of atheromatous plaques and their complications. **(3)**

AORTIC STENOSIS

Aortic stenosis is a disease that affects males over the age of 60 years with tricuspid aortic valves, but which may also be seen in younger people who have a congenital bicuspid aortic valve. The myocardial hypertrophy produced by aortic stenosis is similar to hypertension and it leads to left ventricular hypertrophy, which may, in some cases, produces the enlargement of heart weights of over 700 g. In aortic stenosis, the perfusion problem is worsened by

the stenosed valve, which results in a lower pressure at the coronary orifices and hence in the coronary arteries. Sudden death is common in these types of patients. **(2)**

PRIMARY MYOCARDIAL DISEASE

These are much less common than the degenerative disease conditions described above and they commonly affect a significantly younger age group. Myocarditis occurs in many infective diseases, such as diphtheria and virus infections, including influenza, but the clinical manifestations, complications and sudden death associated with these infections may occur some days or even weeks after the onset of main clinical symptoms. Care must be taken in interpreting the histological appearances of small foci of myocarditis because isolated collections of lymphocytes may be identified in the myocardium of young adults who have died suddenly from trauma, suggesting that these foci are simply incidental findings. These foci used to be known as ‘isolated Fiedler’s myocarditis’. Other infective and inflammatory processes can also involve the myocardium, including disseminated sarcoidosis. **(3)**

A more definite group of intrinsic cardiac diseases is the ‘cardiomyopathies’. The initial descriptions referred to cardiomegaly with huge hearts of over 1000 g and asymmetric thickening of the ventricular walls in the hypertrophic, obstructive type of cardiomyopathy (HOCM) or dilatation of the chambers in congestive cardiomyopathy. Both types of the disease are usually associated with areas of disordered myocardial fibres. Extensive research has now shown that the cardiomyopathies are a much more complex group of

primary myocardial diseases, commonly with a genetic background, which often do not show the typical macroscopic appearances described above. Right ventricular cardiomyopathy is now described and is associated with fatty infiltration of the wall of the right ventricle. Other primary myocardial diseases or conduction defects, many of which are inheritable, are now described and include long QT syndrome or Brugada syndrome and catecholaminergic polymorphic ventricular tachycardia. The autopsy in these cases will be entirely negative but, as DNA techniques improve, diagnosis will be made in the laboratory rather than the mortuary. **(3)**

DISEASES OF THE ARTERIES

The most common problem of the arteries themselves that is associated with sudden death is the aneurysm. **(3)**

ATHEROMATOUS ANEURYSM OF THE AORTA

These aneurysms are most commonly found in young and elderly males in the abdominal aorta. They are formed when the elastic layer of the aortic blood vessel wall below an atheromatous plaque is damaged and the blood pressure is able to balloon the weakened vessel wall. The aneurysms may be saccular in shape (expanding to one side) or in fusiform shape (cylindrical). The wall of the aneurysm is commonly gets calcified and the lumen is commonly lined by old laminated thrombus. Many aneurysms remain silent and are found as an incidental finding at autopsy, but others becomes eventually rupture. Because the aorta lies in the retroperitoneal space, that is where the bleeding is found; it usually lies to one side and may envelope the

kidney. Rarely, the aneurysm itself, or the retroperitoneal haematoma, ruptures through the retroperitoneal tissues to cause a haemo peritoneum. **(3)**

DISSECTING ANEURYSM OF THE AORTA

The destructive damage caused by an atheromatous plaque can also result in the weakening of the aortic media, and a defect in the intima, usually also associated with the plaque, allows blood from the lumen to dissect and diverted into the weakened area of media. Once the dissection has started, the pressure of the flowing blood extends the dissection along the aortic wall. The commonest site of origin of a dissecting aneurysm is in the thoracic aorta and the dissection usually tracks distally towards in the abdominal region, sometimes reaching the iliac and even the femoral arteries. In serious cases, the track may rupture at any point, resulting in bleeding into the thorax or abdomen. Alternatively, it can dissect proximally around the aortic arch and into the pericardial sac, where it can produce a haemo pericardium or cardiac tamponade and causes sudden death. Dissecting aneurysms are also seen in association with diseases of the aortic media such as cystic medial degeneration. **(3)**

INTRACRANIAL VASCULAR LESIONS

Several types of intracranial vascular hemorrhages are important in the causes of sudden or unexpected death. **(3)**

RUPTURED BERRY ANEURYSM

A relatively most common cause of sudden collapse and rapid death of young to middle-aged men or women is a subarachnoid haemorrhage resulting from rupture of a berry aneurysm of the basal cerebral arteries either in the circle of Willis itself or in the arteries which supply it. Whether berry aneurysms can be described as ‘congenital’ depends on the interpretation of the word: strictly speaking, they are not present at birth, but the weakness in the medial layer of the vessel wall usually starts (usually at a bifurcation) from the birth. The aneurysms may be a in a of few millimetres in diameter or they may extend to a size of several centimetres in diameter, they may be single or multiple and they may be found on one or more than one arteries. **(3)**

The aneurysms may remain clinically silent or they may leak, producing a severe throbbing or thunder bolt type of headache, neck stiffness, unconsciousness and sometimes paralysis or other neurological symptoms. The rupture of a berry aneurysm in the arterial circle of Willis allows blood to flood over the base of the brain or, if the aneurysm is embedded in the brain, into the brain tissue itself. The speed of death can be such that the initial impression is of a cardiac event. It is thought that sudden exposure of the brainstem to blood under arterial pressure depresses the cardiorespiratory centres in the brainstem. The role of direct trauma in the rupture of an aneurysm is in dispute. It seems reasonable to suspect that a large, fragile aneurysm on the circle of Willis might be damaged by a substantial head injury; such an aneurysm should be easy to identify at autopsy and so this should not present a diagnostic problem. **(3)**

However, for the more common, small aneurysms situated deeply inside the skull, it seems unlikely that a blow that causes no other cranial or intracranial injury would cause the aneurysm to be selectively ruptured. There is another cause for subarachnoid haemorrhage that is well known to be associated with trauma to the head and neck following violence or an assault when there is forceful lateral flexion of the neck or rotation of the head. **(3)**

The anatomical course of the vertebral arteries is convoluted and, for much of their course in the neck, they are protected within the lateral foramina of the cervical vertebrae. However, they are more exposed to trauma above the first cervical vertebra, and it has been claimed that most traumatic subarachnoid haemorrhages are due to tearing of this extra-cranial region of a vertebral artery within or adjacent to the first cervical vertebra or between it and the base of the skull. Damage to the vertebral arteries at these sites requires the bleeding to dissect in the wall of the artery as far as the base of the skull and then rupture to cause the subarachnoid haemorrhage. This dissection in the arterial wall can often be identified more easily than the intimal damage that marks the origin of the haemorrhage. There may be bruising on the skin surface of the neck to indicate direct trauma or the bruising may be confined to the deep muscles of the neck, indicating indirect trauma to the neck. **(3)**

CEREBRAL HAEMORRHAGE

Sudden bleeding into the cerebral substance of is common, usually in older age and in those with significant high blood pressure and together with cerebral thrombosis and the resulting infarction, this is the commonest cause of

the recognized cluster of neurological signs colloquially termed a ‘stroke’. Spontaneous intra cerebral haemorrhage is most often found in the external capsule of one cerebral hemisphere and arises from rupture of a micro-aneurysm of the lenticulo-striate artery, which is a branch of middle cerebral artery and sometimes called a Charcot–Bouchard aneurysm. The sudden expansion of a haematoma which compresses the internal capsule and may produce ischemia to some of it, leading to a hemiplegia. Haemorrhage can also occur in the cerebellum and the mid-brain, possibly as a result of a ruptured aneurysm or other vascular abnormality, although the abnormality may be extremely difficult to identify at autopsy. Death is seldom instantaneous, although it can be extremely rapid following a haemorrhage in the brainstem. **(2)**

CEREBRAL THROMBOSIS AND INFARCTION

Cerebral thrombosis rarely causes sudden death, as the process of infarction is relatively slow, although the neurological symptoms and signs may have a very rapid onset and be severe.

The term ‘cerebrovascular accident’ (CVA) is a common term used as commonly for both as a clinical diagnosis and as a cause of death. Occasionally, it is misinterpreted by the common people, and sometimes also by legal officials, as indicating an unnatural cause of death because of the use of the word ‘accident’. To avoid this risk, it is much more satisfactory, if the exact cause is known, to use the specific term that describes the aetiology – ‘cerebral haemorrhage’ or ‘cerebral infarction’ – or, if the aetiology is not known, to use the generic term ‘cerebrovascular accident’. **(3)**

PULMONARY EMBOLISM

The major cause of sudden death within the respiratory system is again vascular component. Pulmonary embolism is very common and, in fact, it is the most clinically underdiagnosed cause of death. In almost every case, the source of the embolus is in the leg veins. Tissue damage, especially where it is associated with immobility of a person or bed rest, is a very common predisposing factor in the development of deep vein thrombosis. Most thrombosis remains clinically silent and causes no problems, but a proportion of it ebonizes and blocks pulmonary arteries of varying size. Some produce no lung lesions at all, whereas others produce infarcts that may or may not lead to clinical signs, and a minority (though an appreciable number) block a major vessel and cause death. **(3)**

About 80 per cent of pulmonary embolism deaths have a predisposing cause such as massive tissue damage, fractures, surgical operation, immobilisation by bed rest, forced immobility etc., but the remainder occur unexpectedly in normal, ambulant people who have reported no clinical symptoms. This makes establishing the relationship of death to an injurious event difficult. For the purposes of law (where ‘the balance of probabilities’ is enough), the embolism can often be linked to the trauma, but in a criminal trial in which the higher standard of proof – ‘beyond reasonable doubt’ – is required, it is much harder to relate fatal pulmonary emboli to the trauma. **(3)**

Other rare causes of sudden deaths in the respiratory system include a massive haemoptysis from pulmonary tuberculosis or from a malignant tumour.

Rapid (but not sudden) deaths can also occur from massive fulminating chest infections, especially virulent forms of influenza. Once again, the causes of sudden death have a vascular component in that very severe bleeding from a gastric or duodenal peptic ulcer can be fatal in a very short time, but, more commonly, the bleeding is less torrential and is therefore amenable to medical or surgical treatment. **(3)**

Mesenteric vein thrombosis and embolism, usually related to aortic or more generalized atheroma, may result in infarction of the bowel, but once again, a rapid but not sudden death is expected if the infarction remains undiagnosed. **(3)**

Gastro Intestinal Complications

Perforation of a peptic ulcer disease can be fatal in hours if not treated and intestinal infarction due to a strangulated hernia or due to torsion of the bowel around an area of peritoneal adhesions can also be fulminating and fatal conditions, as can peritonitis arising from diverticular disease or a perforated carcinoma. **(2)**

Many of these conditions as described above present as sudden death in elderly people because they cannot or will not seek medical assistance at the time of onset of the symptoms and are then unable to do so as their condition worsens. When a woman of childbearing age is found unexpectedly dead, a complication of pregnancy must be considered to be the most likely cause of her death until all such causes are excluded. Abortion remains one possibility anywhere in the globe, but especially in countries where illegal abortion is still very common **(2)**

A ruptured ectopic pregnancy, which is usually tubal in position, is another important and serious obstetric emergency that can end in death from intra peritoneal bleeding unless rapidly diagnosed and treated by surgical intervention. As a ‘rule of thumb’, the three commonest causes of death in a woman of reproductive age are: A natural complication of pregnancy such as ruptured ectopic gestation or an induced complication such as an abortion; pulmonary embolism from leg vein thrombosis; Ruptured cerebral aneurysm. **(3)**

In both conditions, death may occur during an attack and the autopsy will reveal specific features to enable a positive diagnosis to be made. However, both of these conditions are also associated with a few sudden and unexpected deaths each year where the specific features of an attack are absent and for which no obvious cause of death can be identified at autopsy. Even well-controlled epileptic patients may die rapidly and inexplicably; it was once thought that they must have been exhausted from status epilepticus, but this is now not thought to be so. There are some who doubt that these patients are even having a fit when they die because of the lack of pathological features, but many epileptics have fits that leave no pathological signs, rendering this theory unlikely. Epileptics are also at risk from the hazards of all types of accidents during a pre-fit aura (if they have one), while having a fit or immediately afterwards; these hazards include falls, drowning, suffocation and postural asphyxia. **(3)**

LOBAR PNEUMONIA

Lobar pneumonia, which is most commonly caused by Streptococcus Pneumoniae, is usually diagnosed clinically. In those who do not seek medical attention and proceed to die. Consolidated patches involving the lower lobes during autopsy may confirm Lobar Pneumonia. However Histopathologiocal Examination can prove that this can be a case of Lobar Pneumonia. **(3)**

PNEUMOCYSTIS CARINII PNEUMONIA

Forensic pathologists must be aware of the numerous infections that can arise secondary to human immunodeficiency virus (HIV) infection and attempt to confirm the diagnosis with post-mortem serology. Public health officers must be notified of either a possible or a con-firmed diagnosis of HIV infection so that follow-up of close contacts of the decedent can be undertaken. **(3)**

HANTAVIRUS INFECTION

Some pulmonary infections (particularly those due to viruses) can present as diffuse alveolar damage. Hantavirus is transmitted to humans when they contact infected deer mouse (Peromyscus maniculatus) feces or urine. As such, it is most commonly encountered in rural settings. As with many viral infections, the affected individual will initially have nonspecific symptoms of a flu-like illness, but will then develop rapid onset of respiratory symptoms and hypotension. Death can occur within hours to days of the onset of symptoms. The lungs have the typical gross and histologic appearance of diffuse alveolar damage, without features specific for hantavirus. However, the diagnosis can be made by post-mortem serology. Confirmatory serologic tests require non

formalin fixed lung tissue, so it is wise to freeze a small amount of fresh lung at autopsy if hantavirus pulmonary syndrome is suspected. **(3)**

PULMONARY ARTERY THROMBOEMBOLI

Traditionally thrombo emboli are discussed in the context of diseases of the respiratory system, even though they are vascular in origin and their underlying causes generally have nothing to do with the respiratory system. It is not sufficient to simply determine that an individual has died of a pulmonary artery thrombo embolus. Pulmonary thrombo emboli and deep venous thrombi are immediate and intermediate causes of death, respectively, but do not reflect the actual underlying cause of death. Although natural disease processes can cause pulmonary thrombo emboli, as in the case illustrated next, they can also be caused by injury (e.g., deep venous thrombosis and embolization complicating immobilization of a fractured ankle in a cast). **(3)**

When a pulmonary thrombo embolus is found, one should attempt to identify the source of the embolus. Lethal pulmonary thrombo emboli arise from large veins above the level of the knees, including the inferior vena cava, the renal veins, the common iliac veins, the external iliac veins, and the deep veins of the thighs. The latter can be examined by making an incision that extends from the medial third of the inguinal ligament down to the medial surface of the knee, reflecting the muscle, and opening the femoral and popliteal veins. The only difficulty with this procedure is that the entire thrombus may embolize, such that the primary site of thrombosis cannot be seen. Another common method for examination of deep leg veins is to incise

the midline of the calf, expose and reflect the gastrocnemius muscle and then cross section and squeeze it to see if thrombi can be expressed from the veins at this level. If thrombi are present in these smaller veins, the presumption is made that thrombi are also present in the large veins above the knee. **(3)**

REACTIVE AIRWAY DISEASE

Bronchial asthma is also associated with rare unexplained sudden and unexpected deaths, even where there is no evidence of status asthmaticus or even of an unusually severe or prolonged asthmatic episode. Several decades ago, an increase in sudden deaths in asthmatics was traced to the over-use of adrenergic drugs – especially the self-administered dose given by inhaler – but awareness amongst doctors soon reduced this hazard. **(3)**

Extrinsic asthma is a relatively common disease, particularly in children, which both patient and parent often regard to be more of an inconvenience than a serious illness. The reality is that asthma can be lethal within minutes of symptom onset. The typical findings at autopsy include hyperinflation of the lungs with thick mucous plugging of the bronchi. The chronicity of the disease is reflected by protrusion of bronchi above the cut surfaces of the lungs, produced by para bronchial hyperplasia, and by thickening of basement membranes and para bronchial inflammation, which usually includes numerous eosinophils. Difficulty is sometimes encountered in cases where hyperinflation of the lungs and mucous plugging are not particularly pronounced. In these cases, determining the cause of death lies, as always, in providing the most reasonable opinion, taking into account the history, the circumstances of the

death and the pathologic findings. The forensic pathologist tends to see chronic obstructive pulmonary disease as an incidental finding in individuals dying of injuries or other natural diseases, rather than as the underlying cause of a sudden death. Emphysema and chronic bronchitis can be the underlying cause of cor pulmonale, with right ventricular hypertrophy and other clinical or autopsy findings of right-sided heart failure. These individuals may have terminal arrhythmias that would make their death seem “sudden.” They are also prone to developing pulmonary thrombo emboli, pneumothorax, and bronchopneumonia. This is usually caused by a malignant lung tumor, but can also be caused by tuberculosis or conceivably by any other necrotizing pulmonary disease. Perform histology, lung tissue or blood cultures, and post-mortem serology, and also freeze a small piece of fresh lung, in cases where there is gross evidence of an “atypical” pneumonia or diffuse alveolar damage. Remember that acute asthmatic exacerbations can rapidly progress to death in some cases. Search for the thrombotic source and the underlying cause of pulmonary thromboemboli. **(3)**

Don’t

Forget that the incidence of tuberculosis is on the rise and that it can be spread more easily to the unwary pathologist and autopsy personnel than some of the viruses that most people are more conscious and fearful of (e.g., HIV). **(3)**

GASTROINTESTINAL SYSTEM

Diseases of the gastrointestinal system tend not to present as sudden death, but rather with signs and symptoms that warrant medical attention. In some cases, however, avoidance of clinicians, ignoring of early symptoms, or vagueness of symptoms can create situations where there is at least an appearance that the death was sudden. Bleeding gastric or duodenal ulcers (usually benign, but occasionally malignant) are one of the most common gastrointestinal diseases that will present in this manner, and the scene can be as dramatic as that for massive hemoptysis. Coffee-ground emesis is a good indicator of hemorrhage arising from an ulcer, because the blood must pass through the acid of the stomach, but occasionally the rapidity of hemorrhage can produce the same bright red bleeding that one would see with hemoptysis or bleeding esophageal varices. In cases where the bleeding is slower, there may not be any hematemesis but altered blood will extend throughout the small bowel and colon. **(3)**

PERFORATION OF GASTRIC AND PEPTIC ULCERS

Another presentation of gastric and duodenal ulcers as a cause of "sudden death" is peritonitis arising from ulcer perforation. In fact, whenever peritonitis is encountered at autopsy it is wise to check the gastric antrum and the duodenum first as the most likely sites of gastrointestinal perforation. As with any perforated viscus, one will tend to see tense abdominal distention on external examination **(3)**

COMPLICATIONS OF APPENDICITIS

Although not regarded as sudden death, the forensic pathologist may come across rare cases of ruptured appendix with peritonitis, typically when there are concerns about the medical care that a patient received in an emergency room or walk-in medical clinic. Clinically differentiating acute appendicitis and other acute gastrointestinal inflammations (e.g., diverticulitis) from viral flu-like illnesses can be difficult, occasionally leading to the discharge of a patient with an incorrect diagnosis and significant risk of dying with peritonitis and/or sepsis. Finally, as a further comment on how some individuals can simply adjust to or put up with the symptoms of acute-on-chronic disease. The small bowel with in the hernia may be strangulated and that may be presented as perforated chemical peritonitis. **(3)**

SEPSIS

An overwhelming variety of bacterial and viral infectious diseases can present as sudden death. The key to making a diagnosis at autopsy is to have a high index of suspicion for sepsis and to order appropriate post-mortem tests, including blood cultures and occasionally blood or cerebrospinal fluid serology. **(3)**

An indication of potential sepsis at autopsy is the finding of extremely rapid decomposition, particularly if the body has been properly refrigerated. Investigation of possible sepsis-related deaths should include inquiries at the hospital laboratory as to the results of any antemortem blood cultures. Postmortem blood cultures do work, though, particularly if one obtains a pure

culture of a pathogenic organism that fits with the clinical picture and the autopsy findings. Post-mortem blood cultures can be taken via sub clavian or femoral puncture, after sterilization of the skin with povidine followed by an alcohol swab. Blood cultures can also be obtained internally by opening the pericardial sac, having an assistant lift the heart by its apex, and obtaining blood from the right atrium. Because the pericardial sac is sterile, with the possible exception of any bacteria causing a septic process, there is no need to sterilize the pericardial surface of the atrium with povidine and alcohol or with heat. **(3)**

Urosepsis is most commonly encountered in those with neurogenic bladders or obstructive uropathy. On occasion, the forensic pathologist will see the sudden death of a previously healthy person caused by sepsis arising from undiagnosed acute pyelonephritis. Use of the diagnosis **"urosepsis**" and other infections as cause of death statements on **clinician-provided death certificates should cause death investigators to ask for more information as to the proximate cause of death.** Such phraseology is commonplace amongst clinicians, particularly those who are involved in the treatment of paraplegic and quadriplegic patients. This particular group of patients is most likely to have an underlying cause of death of unnatural origin (motor vehicle accidents, gunshot wounds, etc.). **(3)**

Do

Obtain post-mortem blood cultures whenever you have reason to believe an individual has died as a result of sepsis. Interpret post-mortem blood cultures with caution, paying particular attention to whether an organism grown in a post-mortem culture makes clinical sense and whether it grows as a pure or mixed culture. Report any death where you suspect infection with Neisseria meningitidis to a public health official. **(3)**

Don't

Assume that an individual dying with autopsy evidence of the Waterhouse-Friderichsen syndrome will always have sepsis due to N. meningitidis. **(3)**

CHRONIC ETHANOL ABUSE

Deaths arising from the acute toxic effects of ethanol and from the wide variety of injuries that acutely intoxicated individuals can inflict upon themselves and others presented elsewhere in this volume. The propensity of chronic alcoholics to develop lobar pneumonia has been covered previously under diseases of the respiratory system, while alcoholic cardiomyopathy was briefly mentioned in the cardiovascular system. **(3)**

As with bleeding peptic ulcers and broncho arterial fistulas, bleeding esophageal varices can produce a dramatic scene that may initially appear to be suspicious for violence. It will be rapidly evident that the blood originates from the mouth, as opposed to any injury, and the autopsy findings of bleeding

esophageal varices associated with micro nodular cirrhosis of the liver will confirm this impression. **(3)**

Chronic alcoholics can die suddenly and unexpectedly, with the only significant findings at autopsy being diffuse fatty change of the liver (and occasionally cirrhosis and/or background alcoholic hepatitis) together with toxicologic findings of either no or a nonlethal ethanol level in the blood. These deaths are attributed to chronic ethanol abuse; however, the exact mechanism by which death occurs is still not clearly understood. **(3)**

In some cases, there may be evidence of alcoholic ketoacidosis, whereas in others there may be an observation of seizure-like activity, suggestive of withdrawal seizures, prior to death. In most, however, no clear pathophysiologic derangement can be identified.

Theories as to potential mechanisms of death include QT prolongation with cardiac arrhythmias, hypoglycemia, electrolyte imbalances, and even pulmonary fat emboli arising from the liver. Ethanol abuse is one of the major causes of both acute and chronic pancreatitis, but acute hemorrhagic pancreatitis is seen less commonly as an immediate cause of death in alcoholics than those deaths associated with diffuse fatty change of the liver or with complications of cirrhosis. **(3)**

Do

Realize that chronic alcoholics can die suddenly and unexpectedly with the only significant findings at autopsy being diffuse fatty change of the liver

and a nonlethal level of ethanol in the blood. The cause of death is chronic ethanol abuse; the mechanism of death is not known. **(3)**

Don’t

Automatically assume that a chronic alcoholic has died as a result of their ethanol abuse—some proportion of alcoholics die of completely unrelated disease. Automatically assume that a chronic alcoholic with an upper gastrointestinal hemorrhage has cirrhosis and bleeding esophageal varices—many will have other causes of upper gastrointestinal hemorrhage.

Automatically assume that a chronic alcoholic has diffuse fatty change of the liver and/or micro nodular cirrhosis—many chronic alcoholics have grossly and histologically normal livers at autopsy. **(3)**

REVIEW OF LITERATURE

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MATERIALS AND METHODS

Material for the present study comprise 100 cases of Sudden Death cases with no known history of any pre-existing diseases autopsied in Government Kilpauk Medical College Hospital, Chennai. All Post-Mortem Certificates and case records in the mortuary files were studied for Sudden death cases with no known history of any pre-existing disease over a period from September 2015 to August 2016.

The Data Consists of:

- History furnished by the police while submitting the requisition for Post-Mortem Examination. (History of the case, Form – 86, Hospital Case Sheets if any and other relevant police documents).
- Detailed examination findings of the deceased who died due to sudden death with no history of any pre-existing diseases autopsied in the Department of Forensic Medicine and Toxicology. Government, Kilpauk Medical College & Hospital, Chennai, during the prospective study period.
- Post-mortem report of the above said cases (Cause of Death).
- Photographs of the organs (Gross Photographs) Taken during Post-Mortem Examination.
- Specimens which were sent to pathology department and Histopathological Photographs pertained to the specimen obtained from the Department of Pathology in Government, Kilpauk Medical College & Hospital – Chennai.

INCLUSION CRITERIA:

- All Brought Dead Cases of Age Group between 18 Years to 40 Years with no history of any pre-existing diseases autopsied in Government, Kilpauk Medical College & Hospital, Chennai.

EXCLUSION CRITERIA:

- Age group less than 18 years.
- Age group more than 40 years.
- Unnatural deaths due to accidents, suicides and homicide.
- Treated cases for any previous ailments.
- Poisoning cases.
- Animal Strike / Sting (Snakes, Scorpion etc).

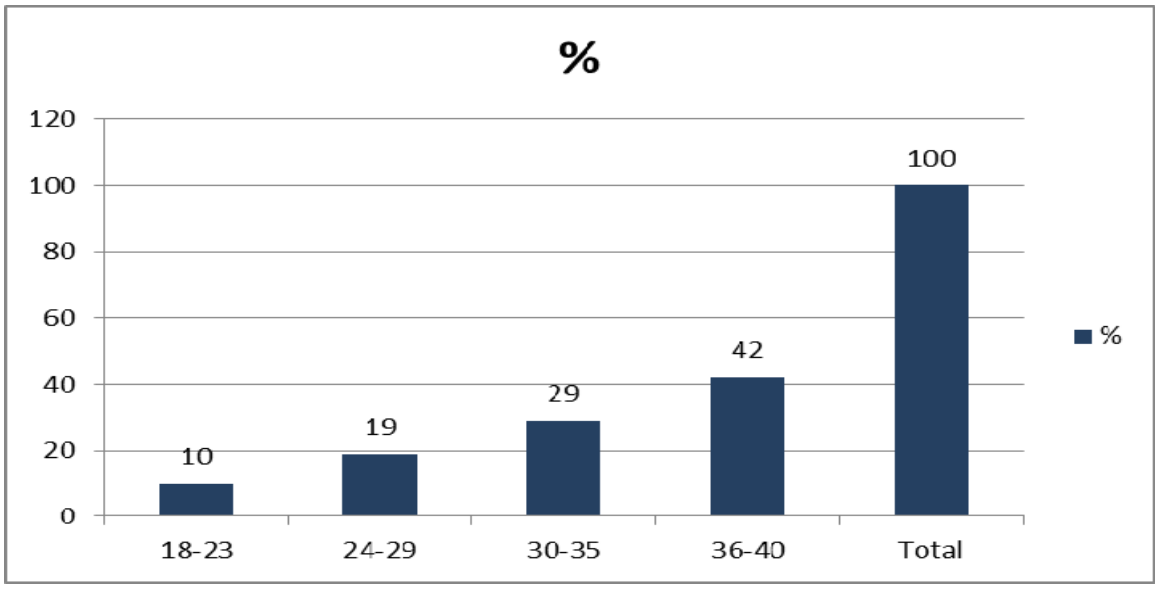
RESULTS

Table-1: AGE WISE DISTRIBUTION

YEARS	NUMBER OF CASES	%
18-23	10	10
24-29	19	19
30-35	29	29
36-40	42	42
Total	100	100

From the above table, it can be noted that maximum number of deaths i.e. (42%) occurred in the age group of 36-40 Years, followed by 30-35 Years age group (29%)

Figure-1: AGE WISE – DISTRIBUTION



AGE WISE DISTRIBUTION: PIE DIAGRAM:

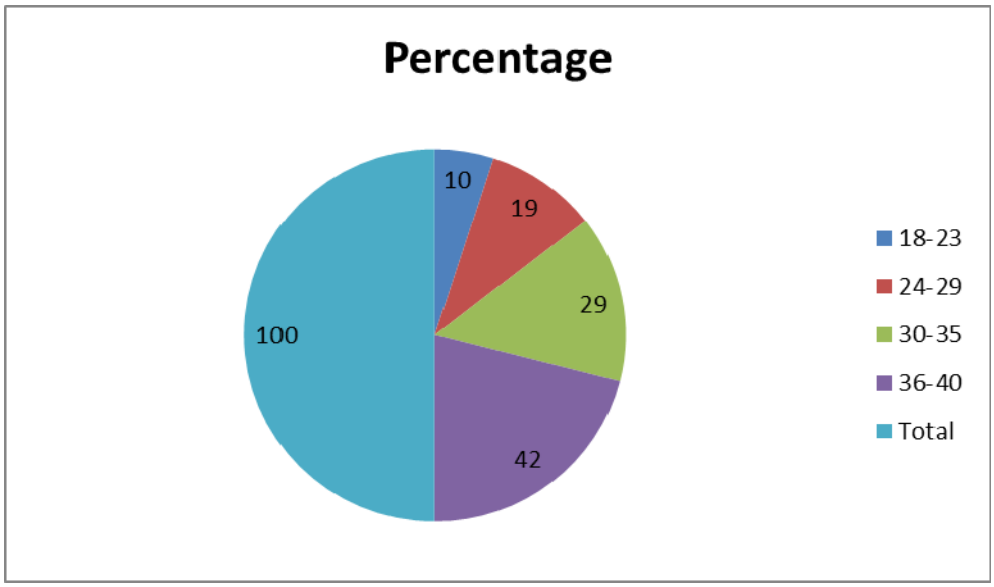
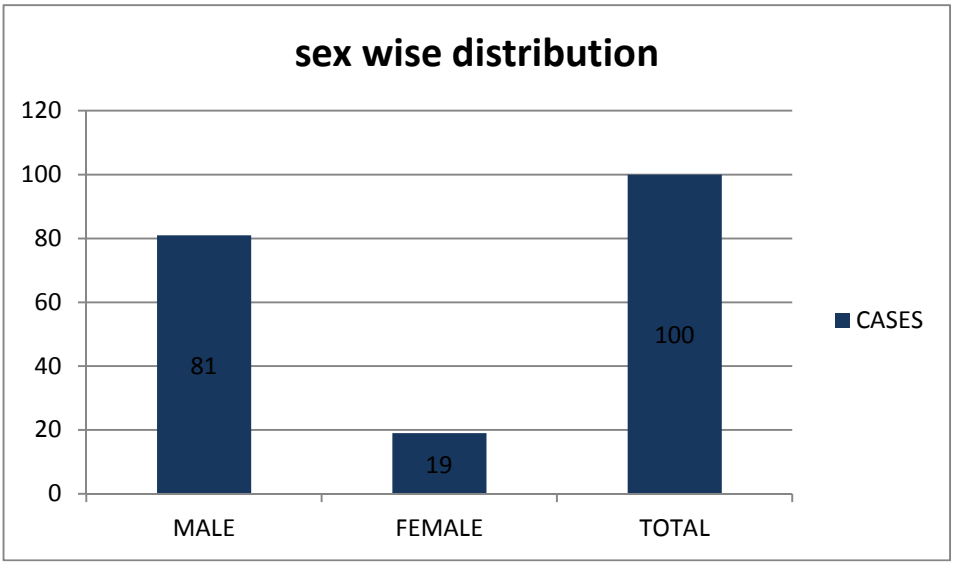


TABLE-2: (SEX WISE DISTRIBUTION)

SEX	CASES	%
MALE	**81**	**81**
FEMALE	19	19
TOTAL	100	100

From the above table, it can be noted that maximum number of cases who died due to sudden death **(81%) were more in males** when compared to that of females.

FIGURE- 2: (SEX WISE DISTRIBUTION)



SEX WISE DISTRIBUTION: (PIE DIAGRAM)

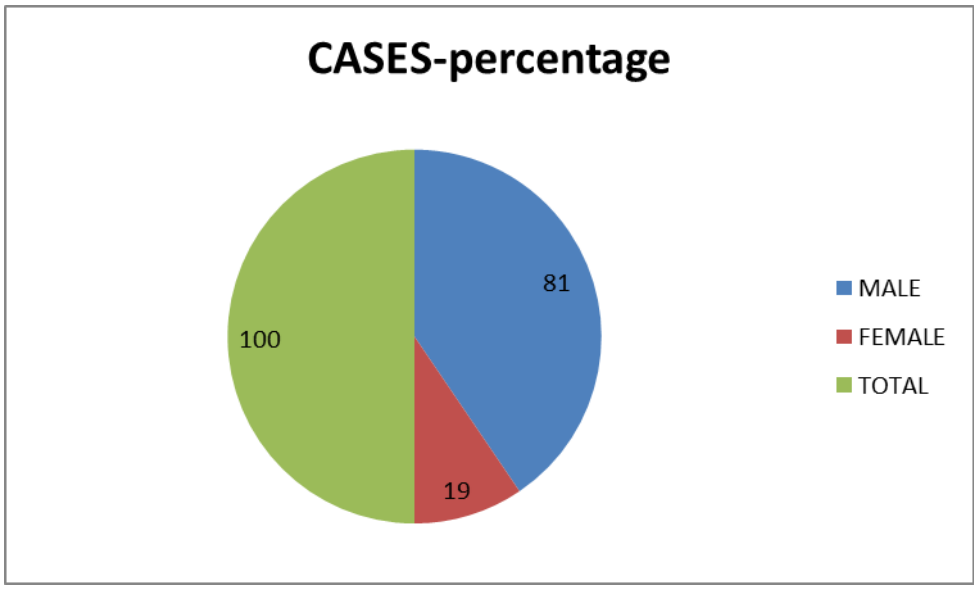
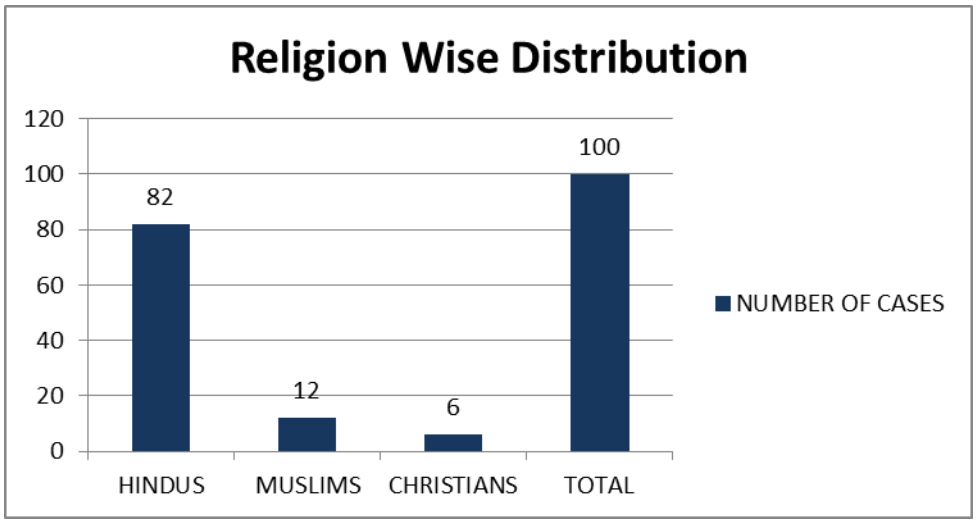


Table-3: (RELIGION WISE DISTRIBUTION)

RELIGION	NUMBER OF CASES	%
HINDUS	**82**	**82**
MUSLIMS	12	12
CHRISTIANS	06	06
TOTAL	100	100

The above table shows that majority of the Sudden Death victims (82%) belongs to Hindu religion.

FIGURE-3: (RELIGION WISE DISTRIBUTION)



RELIGION WISE DISTRIBUTION: (PIECHART):

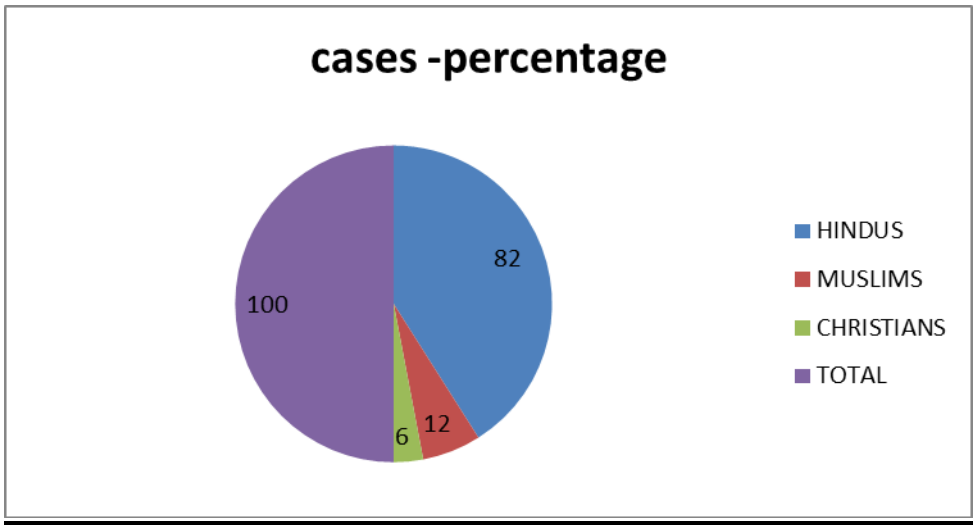
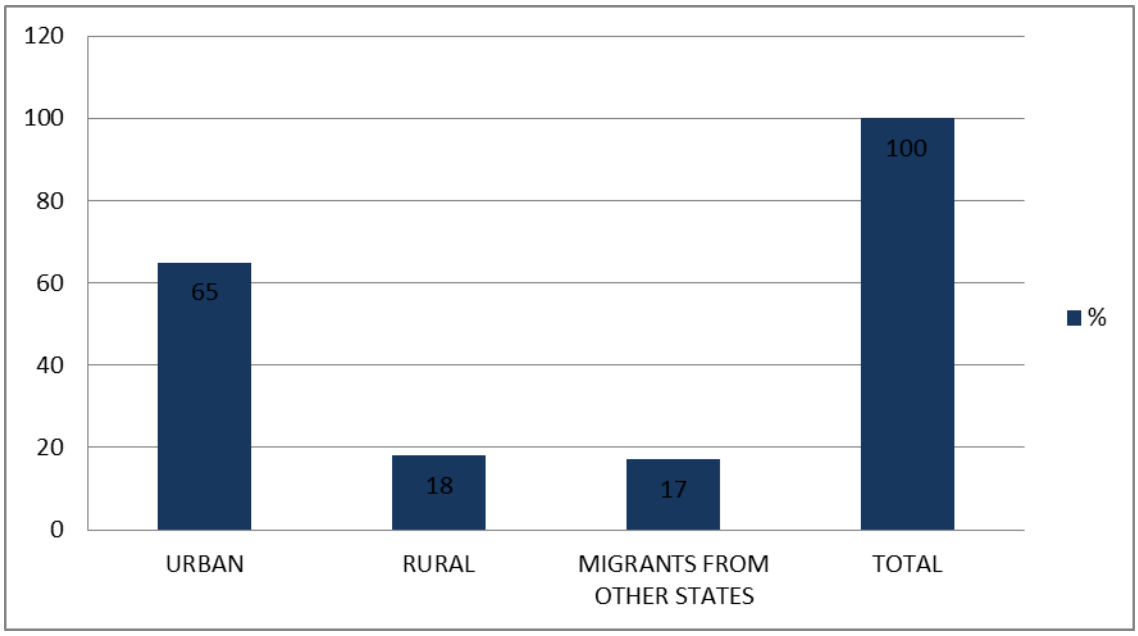


Table-4: (DOMICILIARY PROFILE WISE DISTRIBUTION)

DOMICILIARY	**NUMBER OF CASES**	**%**
URBAN	**65**	**65**
RURAL	18	18
MIGRANTS FROM OTHER STATES	17	17
TOTAL	100	100

The above table shows that majority (65%) of the victims of sudden death belongs to URBAN area.

Figure: 4. (DOMICILIARY PROFILE WISE DISTRIBUTION):



DOMICILIARY PROFILE WISE DISTRIBUTION: (PIE DIAGRAM)

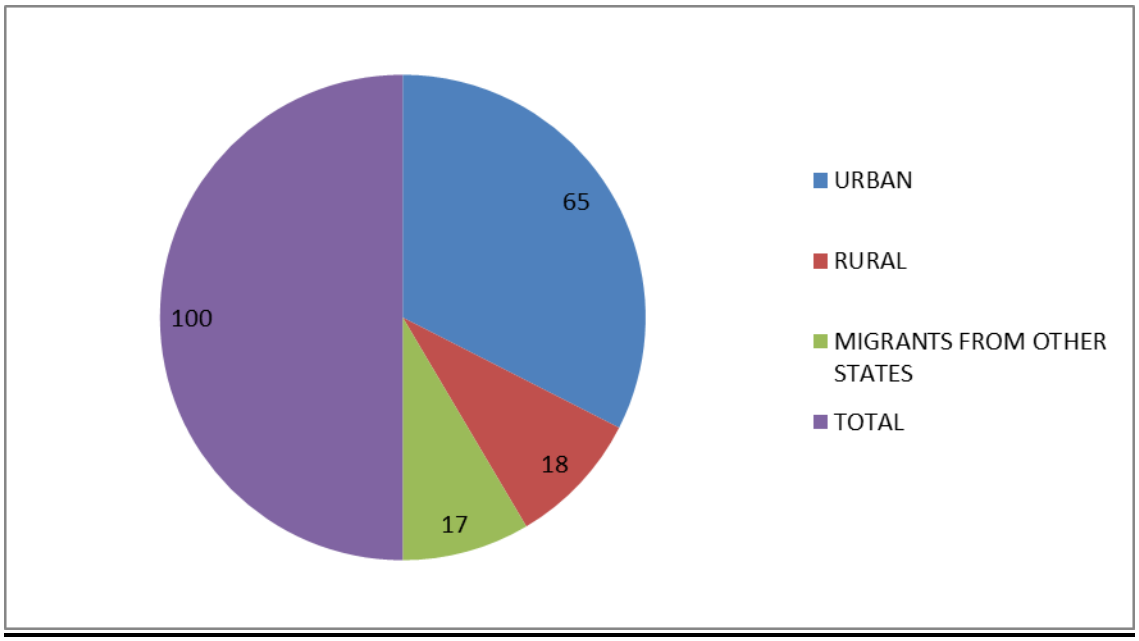
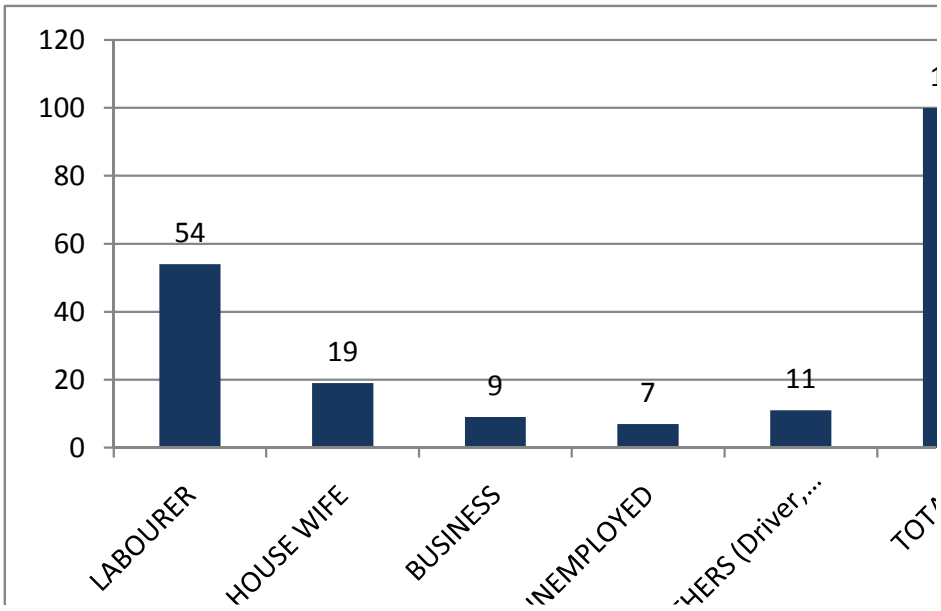


Table-5: (OCCUPATION WISE DISTRIBUTION)

OCCUPATION	NUMBER OF CASES	%
LABORER	**54**	**54**
HOUSE WIFE	19	19
BUSINESS	09	
UNEMPLOYED	07	07
OTHERS (Driver, Mechanic, Electrician, Carpenter etc.)	11	11
TOTAL	100	100

The above table shows that 54% of the victims who died due to sudden death were Laborer's.

Figure-5: (OCCUPATION WISE DISTRIBUTION)



OCCUPATION WISE DISTRIBUTION: PIE DIAGRAM

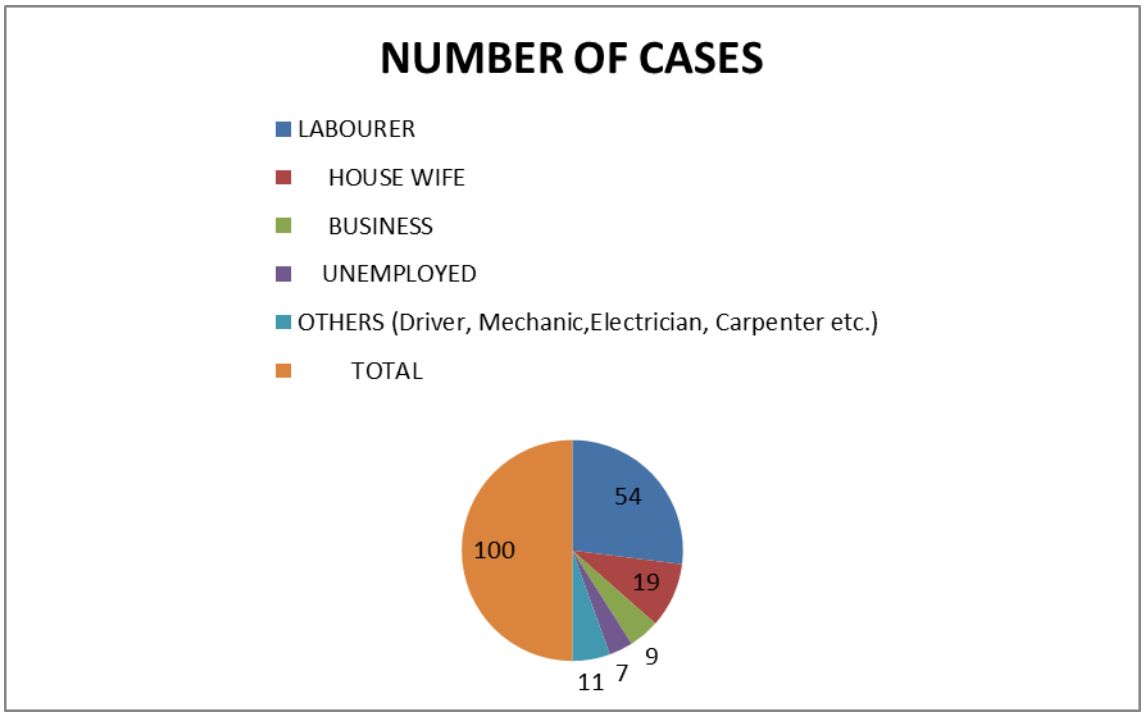
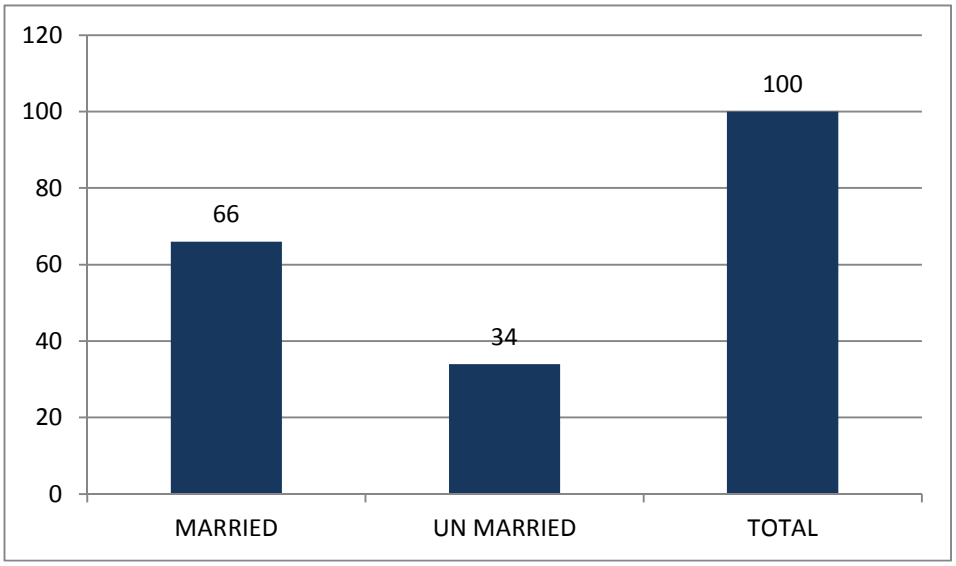


Table-6: (MARITAL STATUS WISE DISTRIBUTION)

MARITAL STATUS	NUMBER OF CASES	%
MARRIED	**66**	**66**
UN MARRIED	34	34
TOTAL	100	100

The above table shows that 66% of the cases who died due to sudden death were Married.

Figure-6: (MARITAL STATUS WISE DISTRIBUTION)



MARITAL STATUS WISE DISTRIBUTION (PIE DIAGRAM):

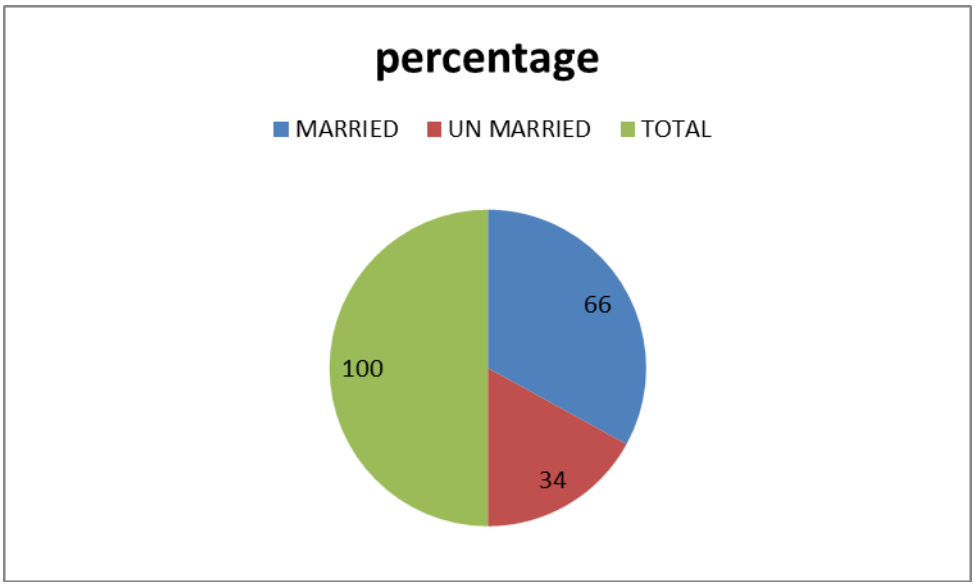
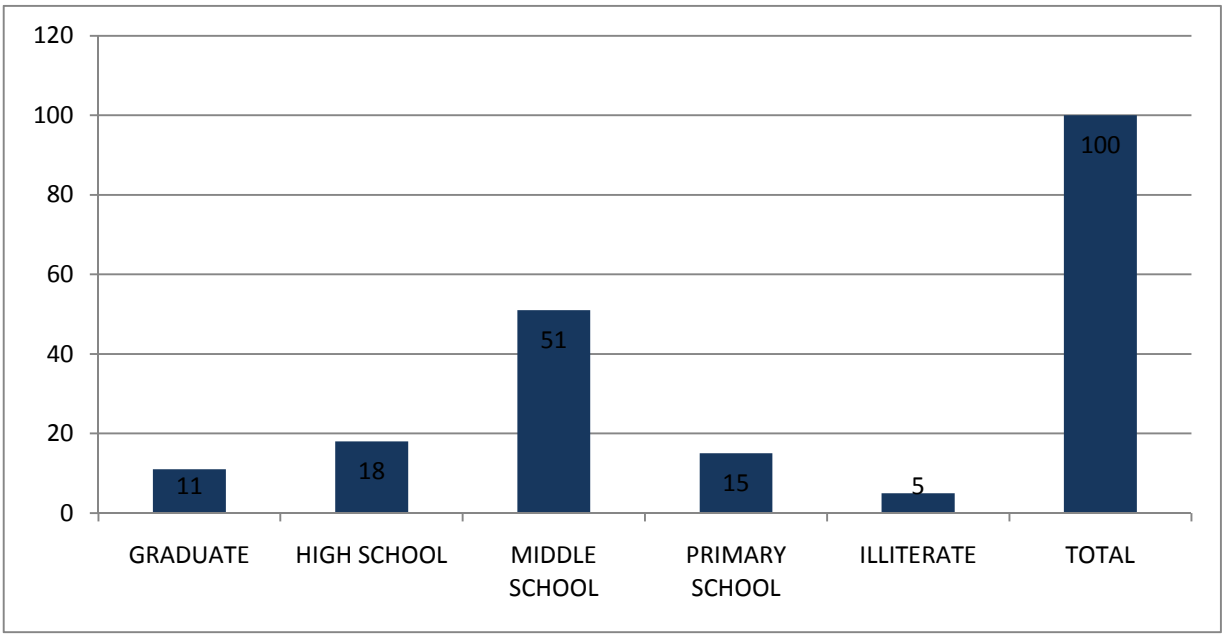


Table-7: (EDUCATIONAL STATUS WISE DISTRIBUTION)

EDUCATION	NUMBER OF CASES	%
GRADUATE	11	11
HIGH SCHOOL	18	18
MIDDLE SCHOOL	**51**	**51**
PRIMARY SCHOOL	15	15
ILLITERATE	05	05
TOTAL	100	100

The above table shows that maximum cases who died due to sudden death belongs to Middle School Education (51%) followed by High School Education (18%).

Figure -7: (EDUCATIONAL STATUS WISE DISTRIBUTION)



EDUCATIONAL STATUS WISE DISTRIBUTION (PIE DIAGRAM)

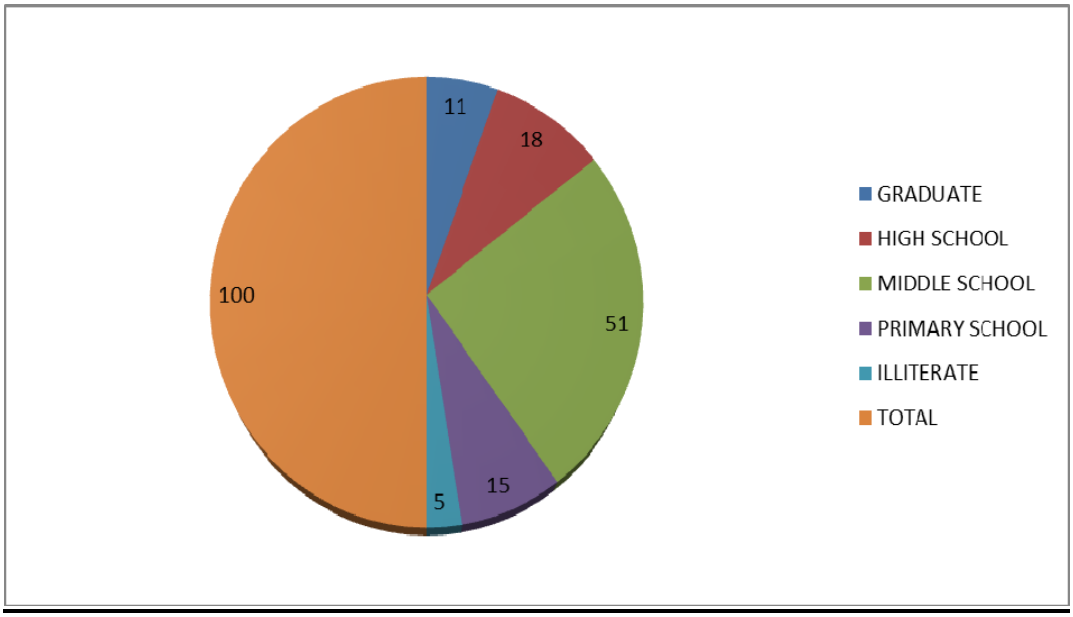
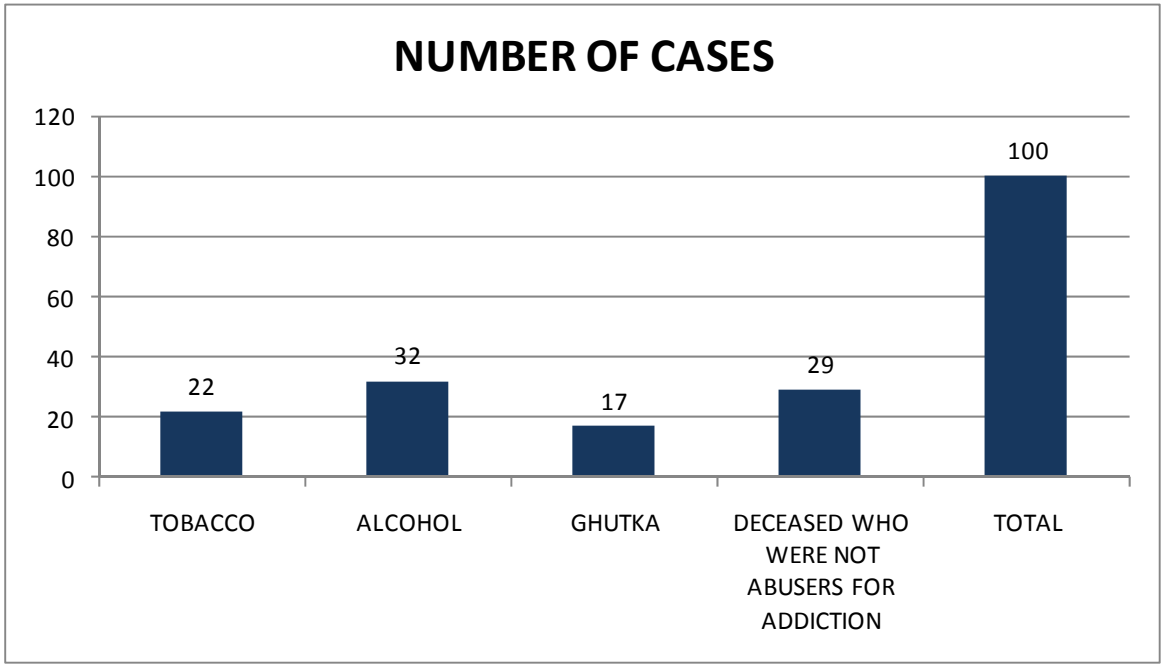


Table-8: (ADDICTION WISE DISTRIBUTION)

ADDICTION SUBSTANCES USED	NUMBER OF CASES	%
TOBACCO	22	22
ALCOHOL	**32**	**32**
GHUTKA	17	17
DECEASED WHO WERE NOT ABUSERS FOR ADDICTION	29	29
TOTAL	100	100

The above table shows that deceased who were addicted to Alcohol (32%) were at high risk for sudden death.

Figure-8: (ADDICTION WISE DISTRIBUTION)



ADDICTION WISE DISTRIBUTION (PIE DIAGRAM)

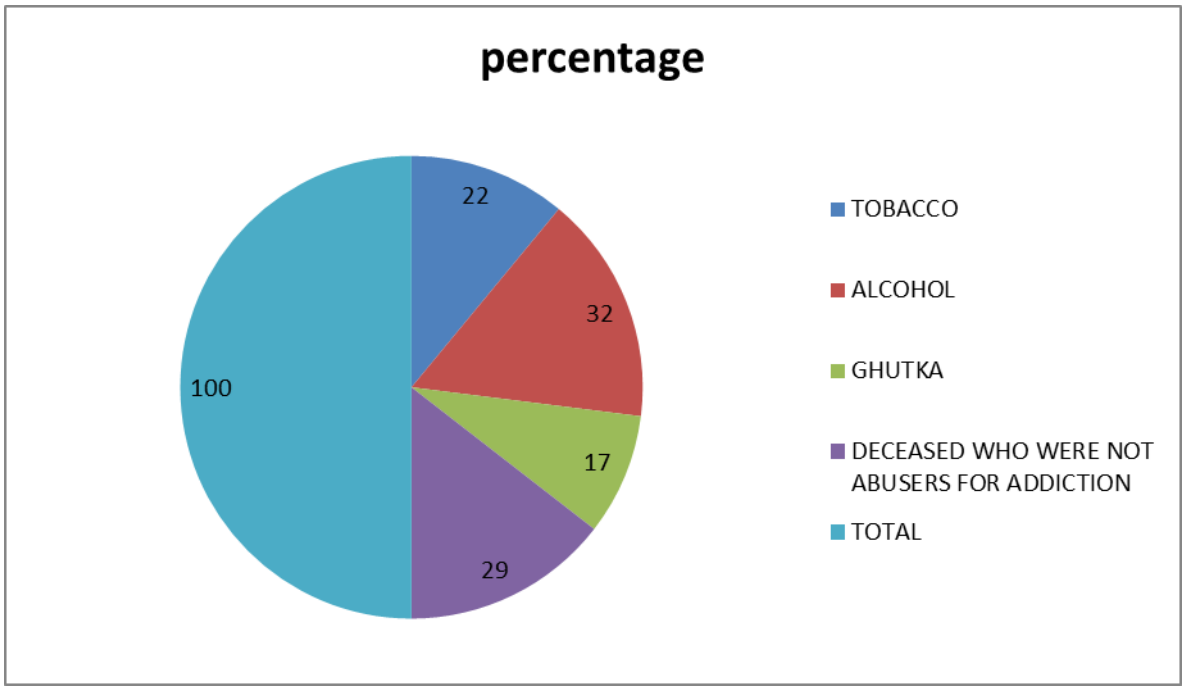
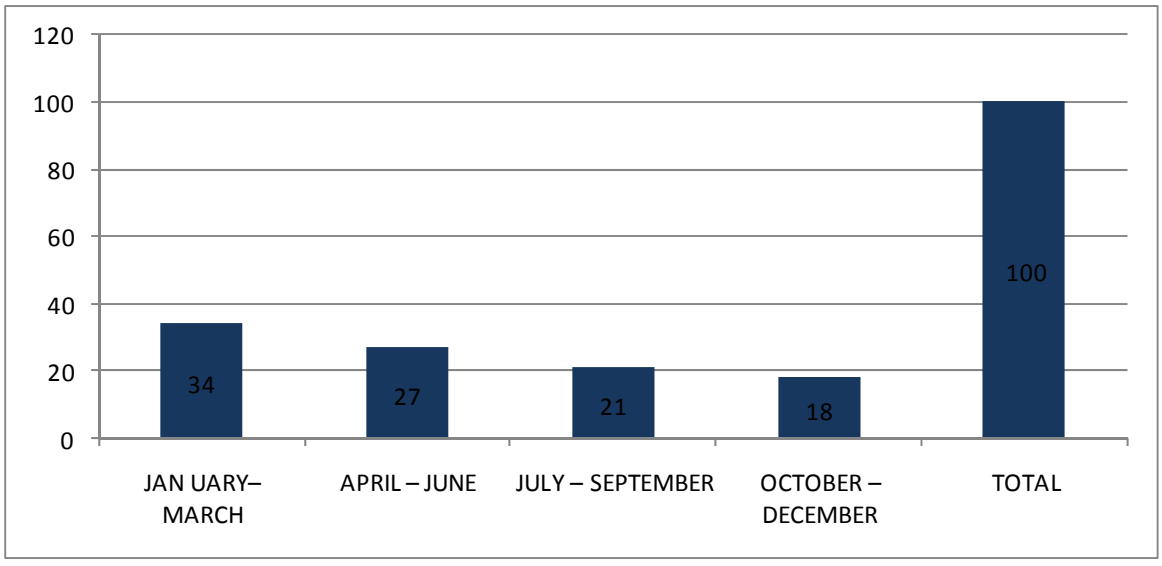


Table-9: (MONTH WISE DISTRIBUTION OF SUDDEN DEATH)

MONTH OF DEATH	NUMBER OF CASES	%
JAN UARY– MARCH	**34**	**34**
APRIL – JUNE	27	27
JULY – SEPTEMBER	21	21
OCTOBER – DECEMBER	18	18
TOTAL	100	100

The above table shows that maximum incidents of sudden death (34%) occurred during January to March

Figure-9: (MONTH WISE DISTRIBUTION OF SUDDEN DEATH)



MONTH WISE DISTRIBUTION : (PIE CHART)

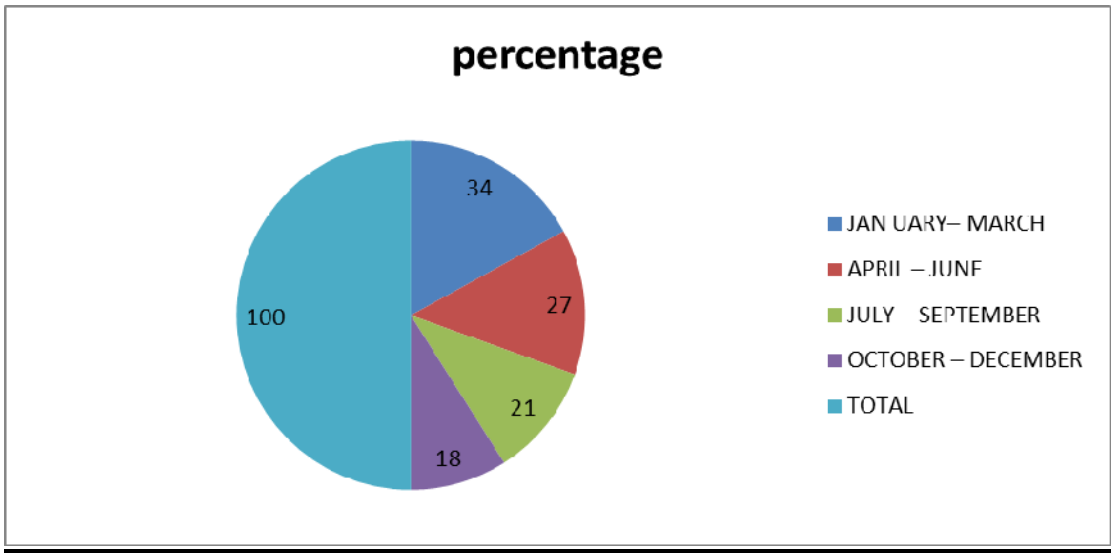
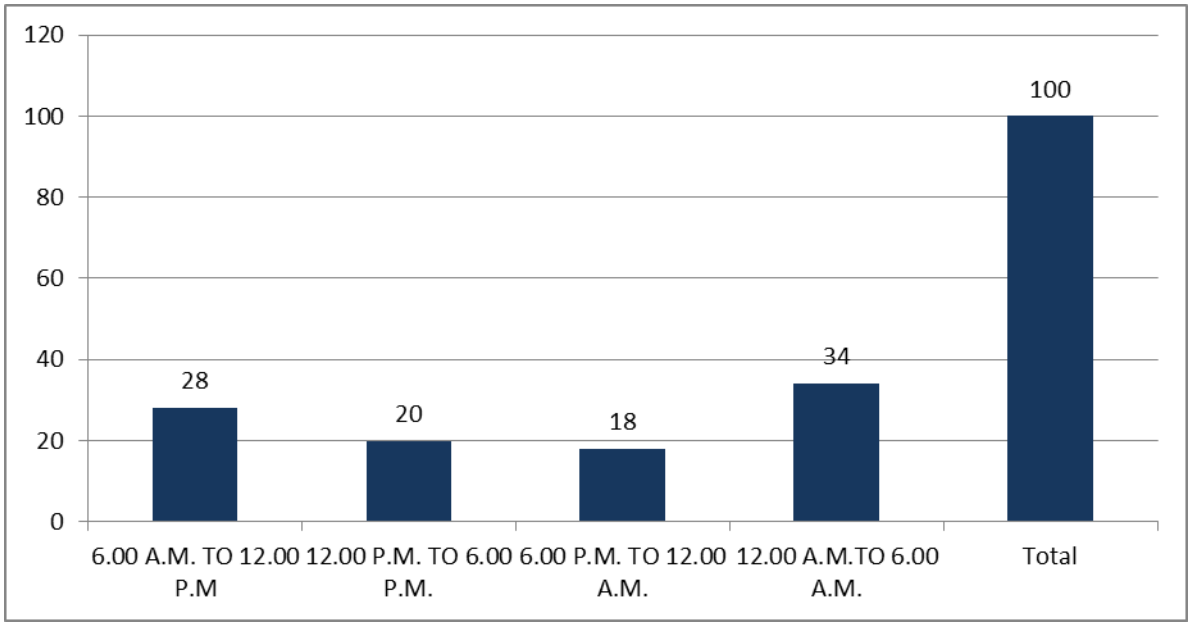


Table-10: (TIME WISE DISTRIBUTION OF SUDDEN DEATH)

TIME	NUMBER OF CASES	%
6.00 A.M. TO 12.00 P.M	28	28
12.00 P.M. TO 6.00 P.M.	20	20
6.00 P.M. TO 12.00 A.M.	18	18
12.00 A.M.TO 6.00 A.M.	**34**	**34**
Total	100	100

The above table shows majority of incidence of sudden death occurred between **12.00 P.M. to 06.00 A.M (34%)** followed by **6.00 A.M. to 12.00 P.M. (28%).**

Figure-10: (TIME WISE DISTRIBUTION OF SUDDEN DEATH)



TIME WISE DISTRIBUTION : (PIE CHART)

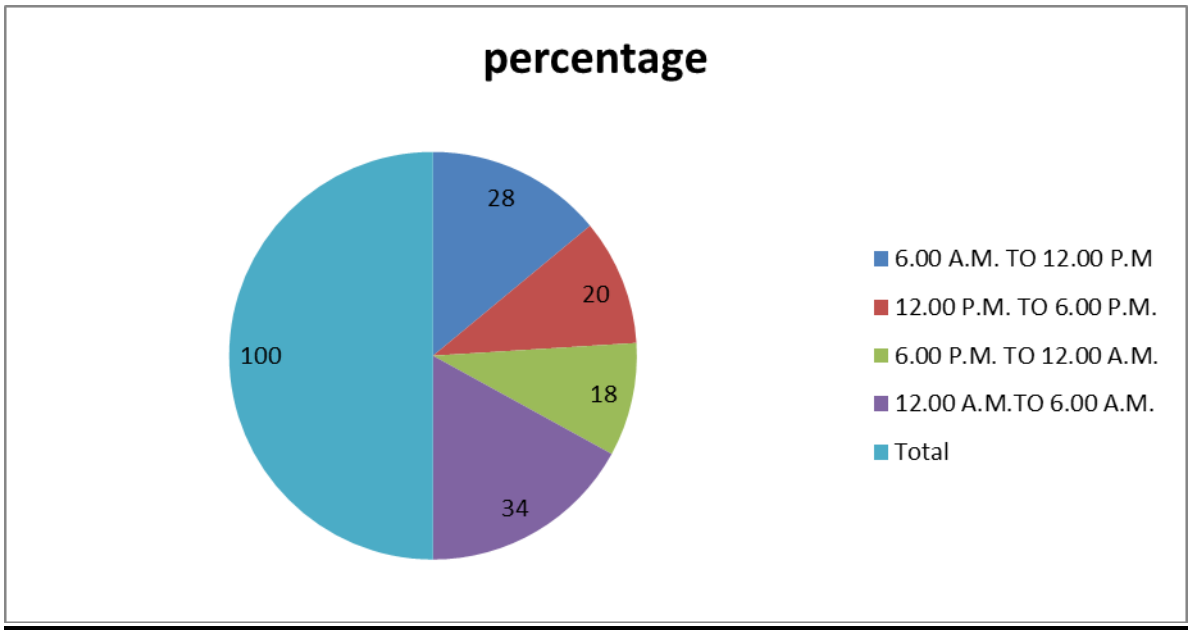
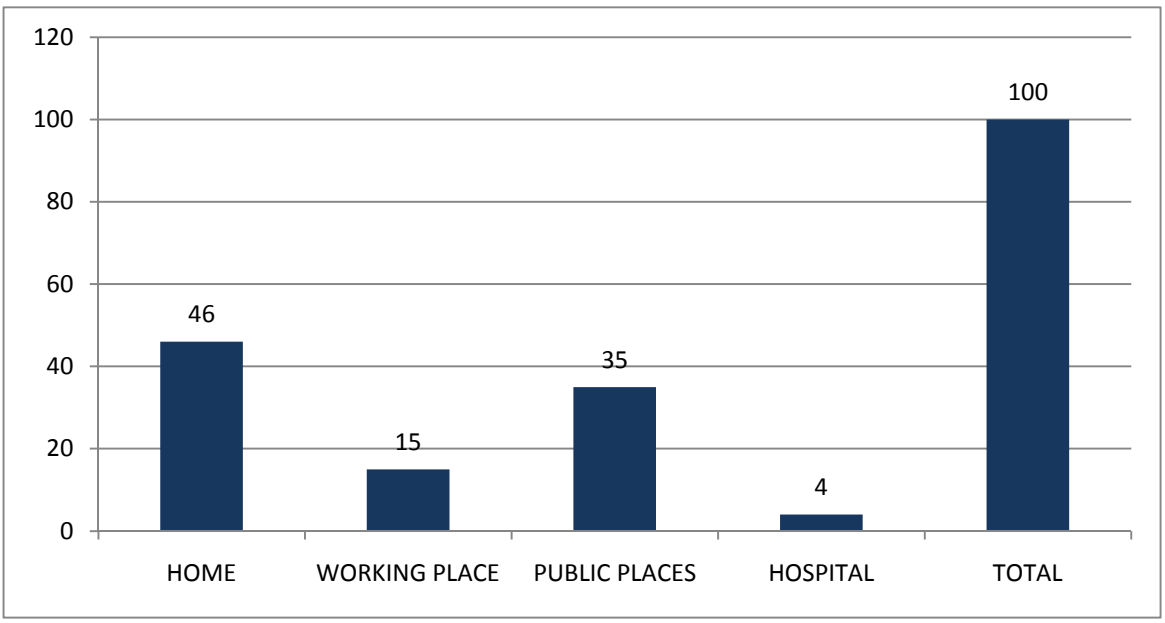


Table-11: (PLACE WISE DISTRIBUTION OF DEATH)

PLACE OF DEATH	NUMBER OF CASES	%
HOME	**46**	**46**
WORKING PLACE	15	15
PUBLIC PLACES	35	35
HOSPITAL	04	04
TOTAL	100	100

The above table shows majority of sudden death incidents took place at home (46%).

Figure-11: (PLACE WISE DISTRIBUTION OF DEATH)



PLACE WISE DISTRIBUTION: PIE DIAGRAM

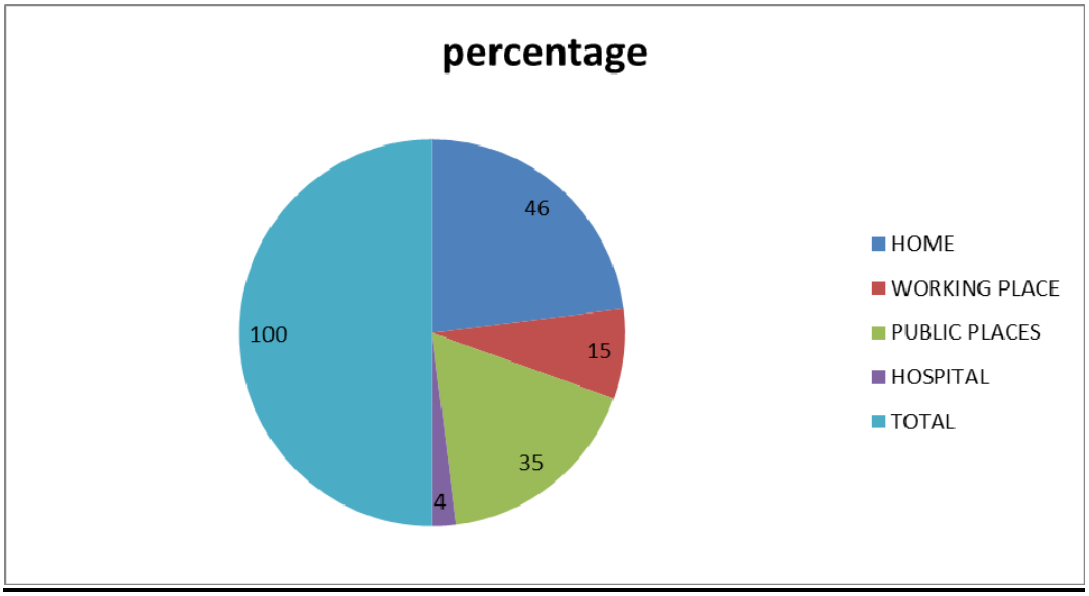
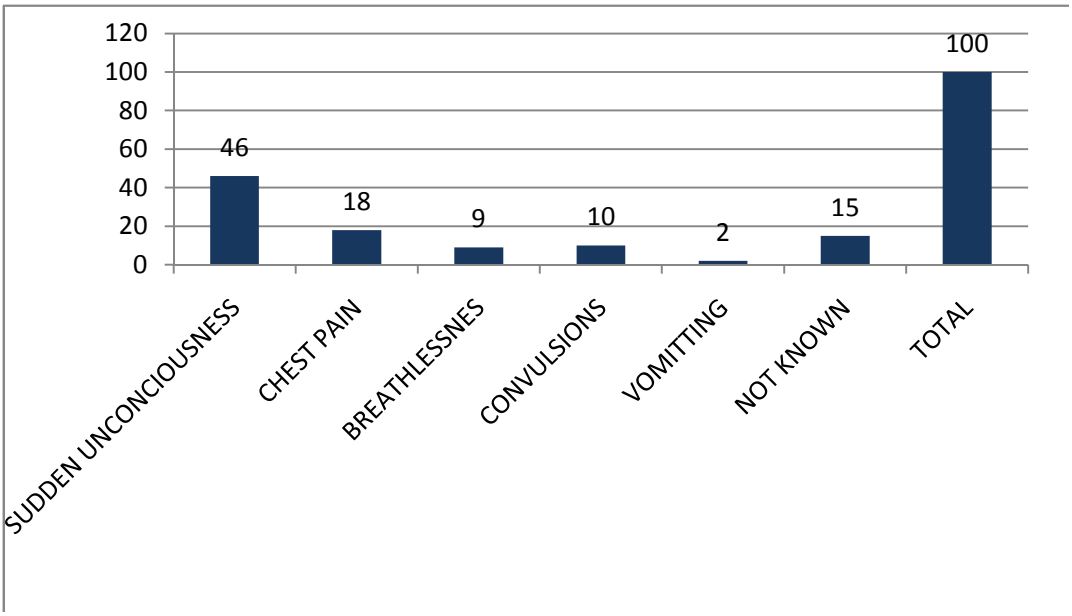


Table-12: (DISTRIBUTION OF SUDDEN DEATH CASES ACCORDING TO PRESENTING COMPLAINTS)

PRESENTING COMPLAINTS	**NUMBER OF CASES**	**%**
SUDDEN UNCONCIOUSNESS	**46**	**46**
CHEST PAIN	18	18
BREATHLESSNES	09	09
CONVULSIONS	10	10
VOMITTING	02	02
NOT KNOWN	15	15
TOTAL	100	100

The above table shows maximum number of cases died who presented with complaints of sudden unconsciousness (**46%)** as presenting complaints.

Figure-12: (DISTRIBUTION OF SUDDEN DEATH CASES ACCORDING TO PRESENTING COMPLAINTS)



DEATH ACCORDING TO PRESENTING COMPLAINTS: (PIE DIAGRAM):

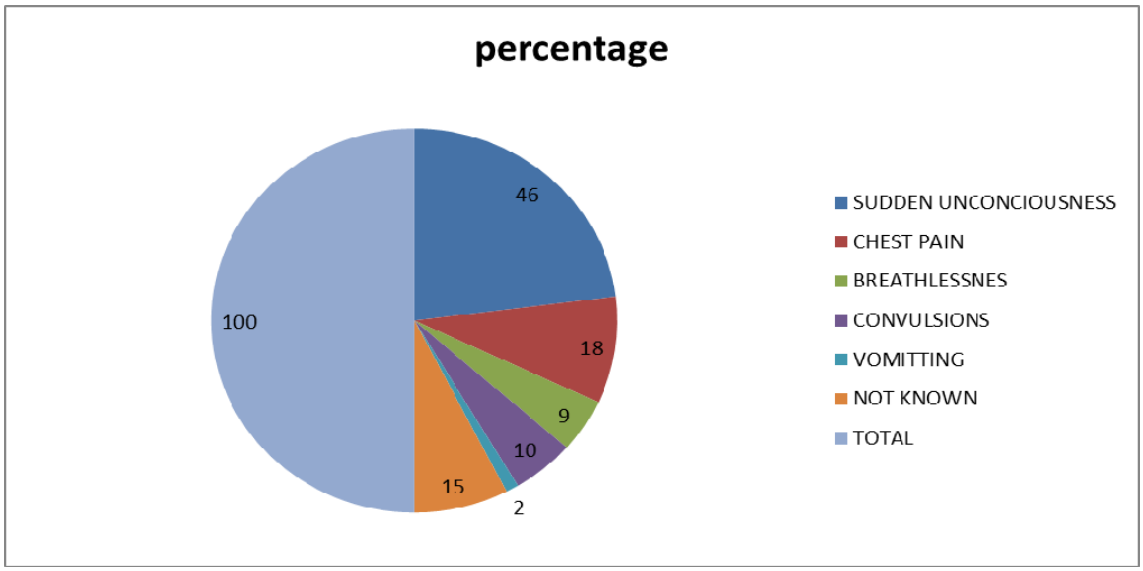
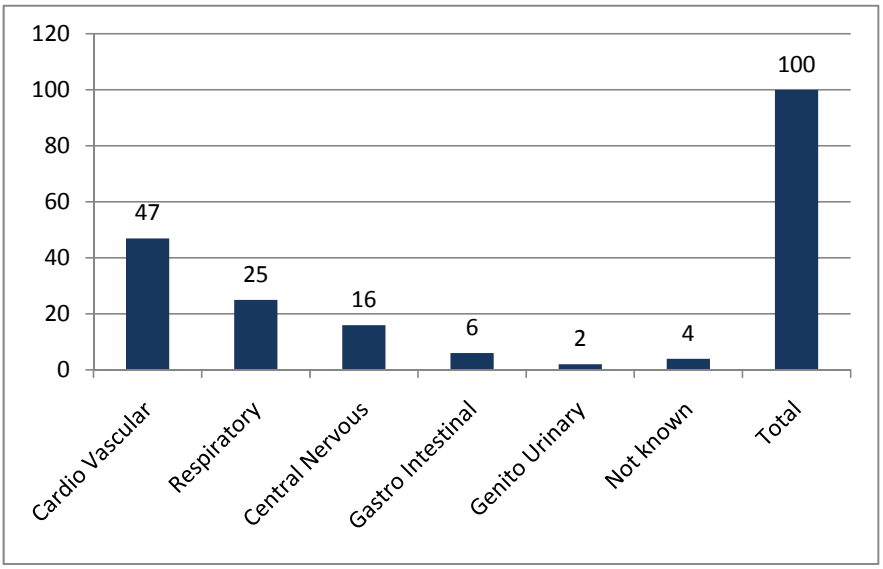


Table-13: (DISTRIBUTION OF SUDDEN DEATH CASES ACCORDING TO PREDOMINANTLY INVOLVED BODY SYSTEM)

BODY SYSTEM INVOLVED	NUMBER OF CASES	%
Cardio Vascular	**47**	**47**
Respiratory	25	25
Central Nervous	16	16
Gastro Intestinal	06	08
Genito Urinary	02	02
Not known	04	04
Total	100	100

The above table shows the most common system in the body resulting in sudden death. Majority of the cases were confined to the Cardio Vascular system **(47%)**

Figure-13: (DISTRIBUTION OF SUDDEN DEATH CASES ACCORDING TO PREDOMINANTLY INVOLVED BODY SYSTEM)



(DISTRIBUTION OF SUDDEN DEATH CASES ACCORDING TO PREDOMINANTLY INVOLVED BODY SYSTEM) : (PIE DIAGRAM)

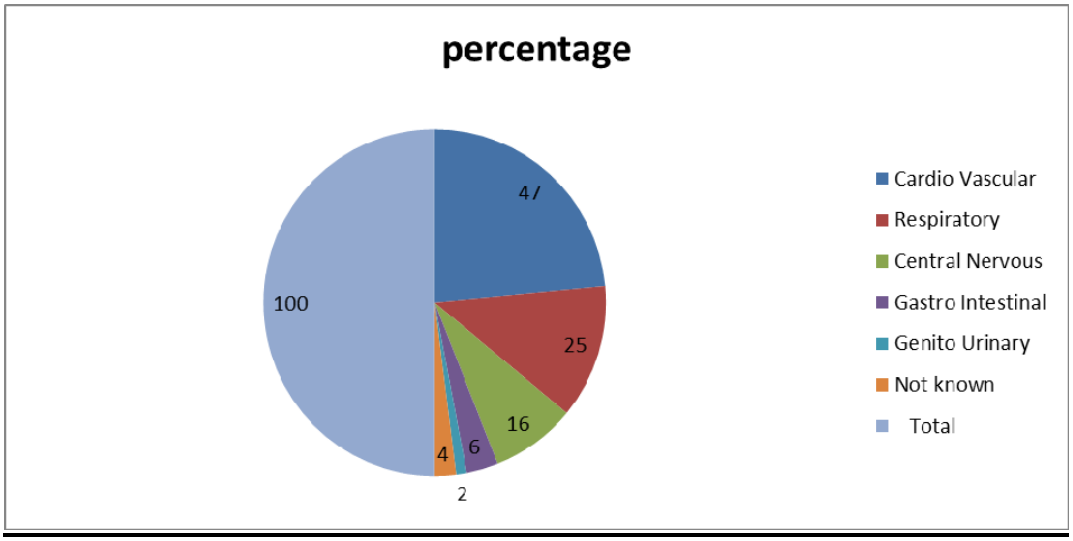
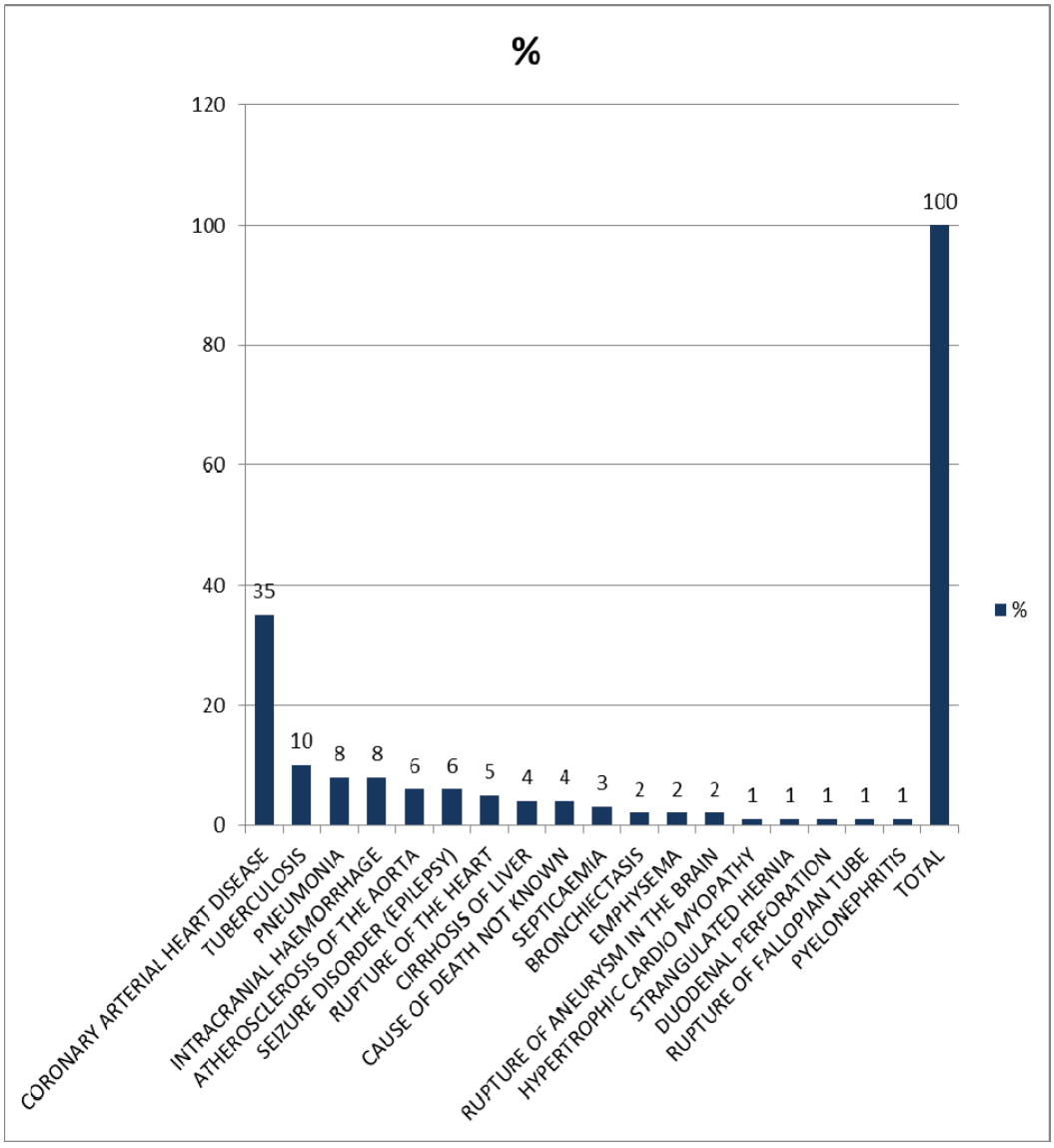


Table 14: (CAUSE OF DEATH DISTRIBUTION)

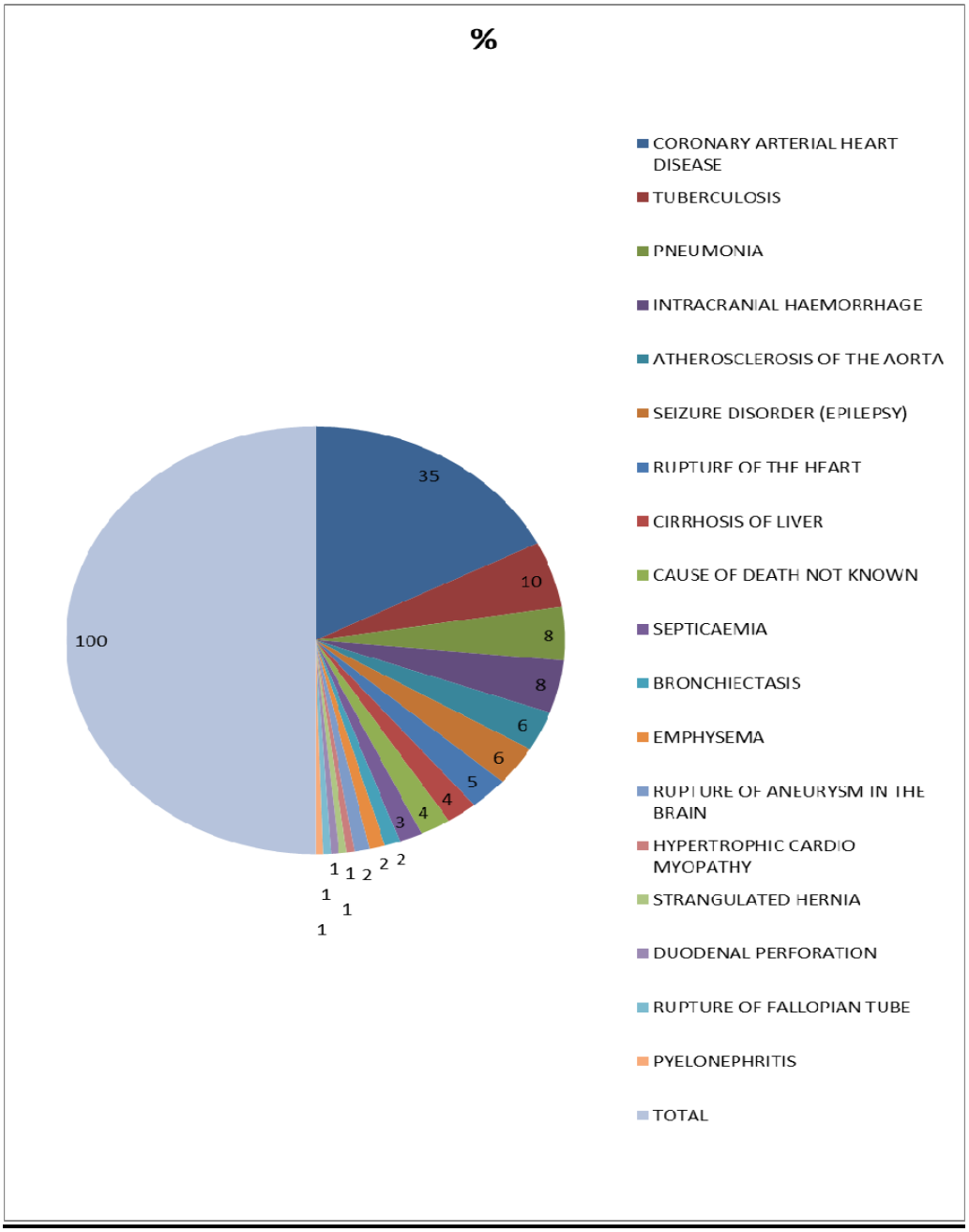
CAUSE OF DEATH	NUMBER OF CASES	%
CORONARY ARTERIAL HEART ISEASE	**35**	**35**
TUBERCULOSIS	10	10
PNEUMONIA	08	08
INTRACRANIAL HAEMORRHAGE	08	08
ATHEROSCLEROSIS OF THE AORTA	06	06
SEIZURE DISORDER (EPILEPSY)	06	06
RUPTURE OF THE HEART	05	05
CIRRHOSIS OF LIVER	04	04
CAUSE OF DEATH NOT KNOWN	04	04
SEPTICAEMIA	03	03
BRONCHIECTASIS	02	02
EMPHYSEMA	02	02
RUPTURE OF ANEURYSM IN THE BRAIN	02	02
HYPERTROPHIC CARDIO MYOPATHY	01	01
STRANGULATED HERNIA	01	01
DUODENAL PERFORATION	01	01
RUPTURE OF FALLOPIAN TUBE	01	01
PYELONEPHRITIS	01	01
TOTAL	100	100

The above table shows that the majority of the deceased died due to **Coronary Arterial Heart Disease (35%) followed by Tuberculosis (10%) and Intracranial Haemorrhage (8%).**

Figure-14: (CAUSE OF DEATH DISTRIBUTION)

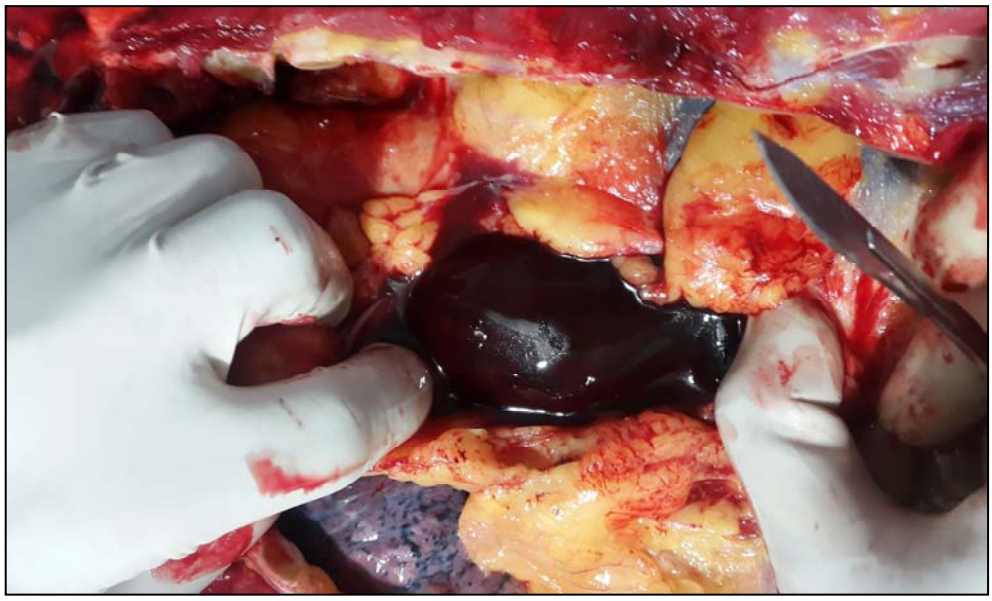


CAUSE OF DEATH: PIE DIAGRAM:

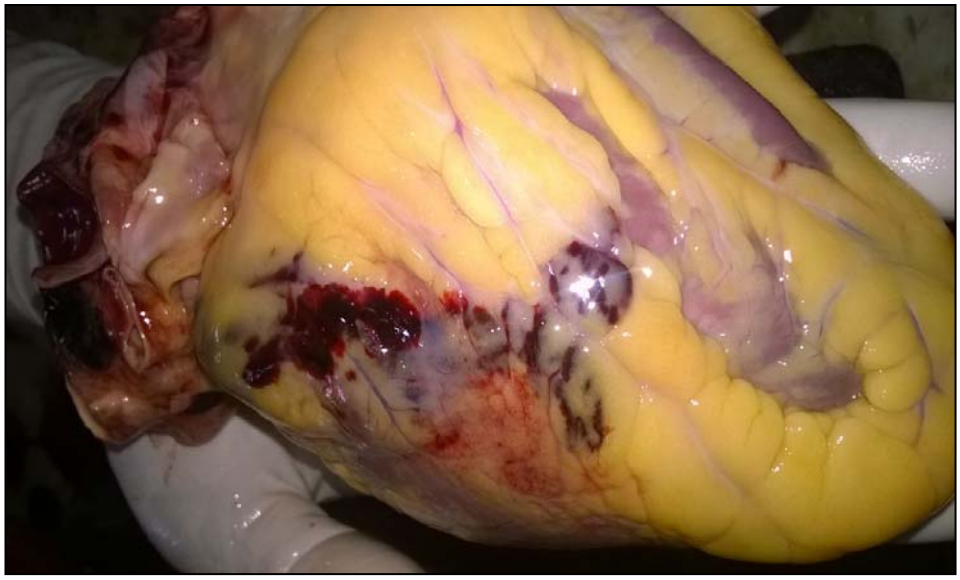


POST MORTEM - PHOTOGRAPHS:

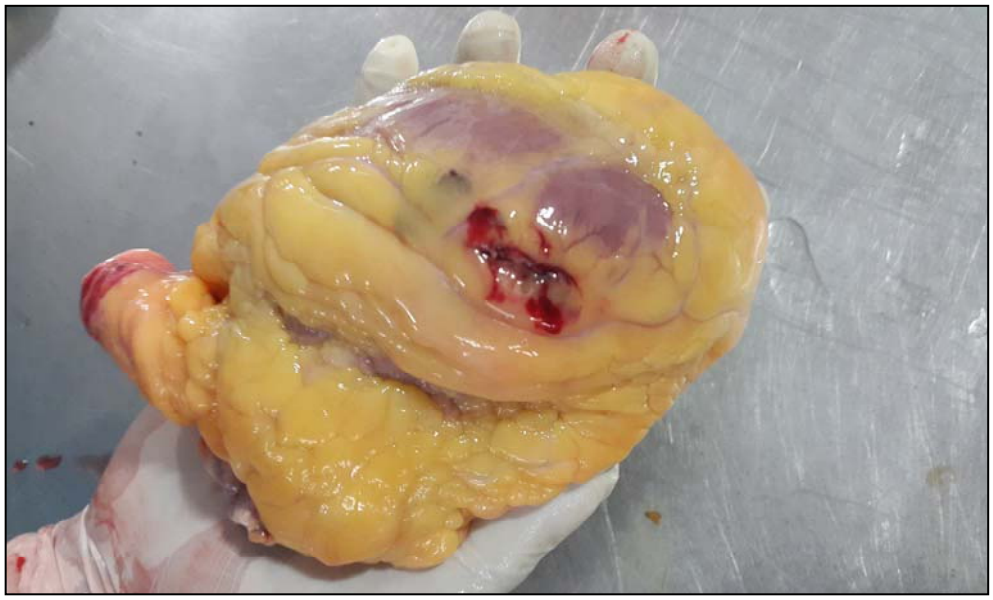
1. **CASE OF RUPTURE OF THE HEART SHOWING COLLECTION OF DARK RED COLOUR HAEMATOMA IN THE PERICARDIAL SAC**



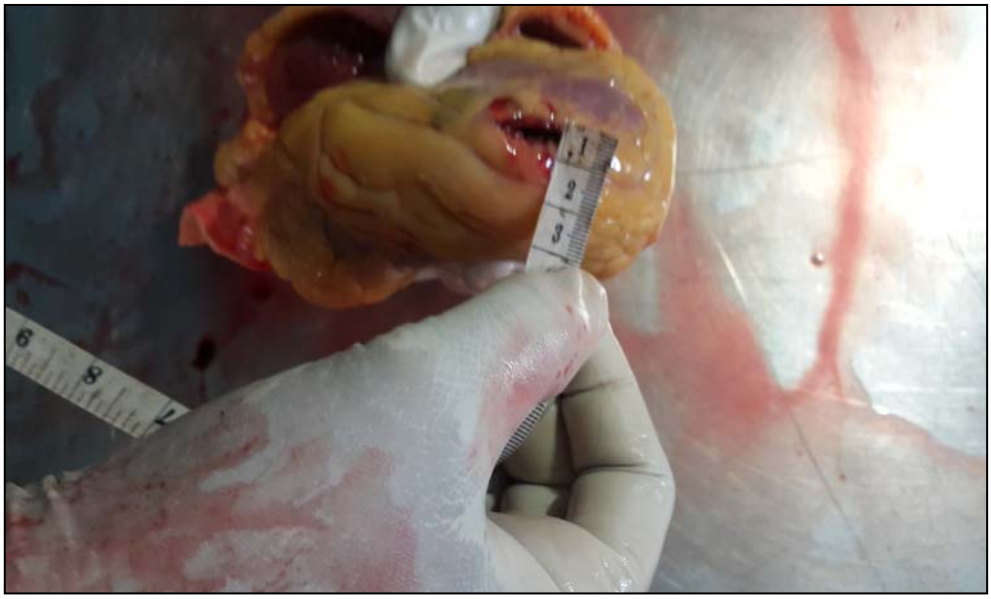
2. **A CASE OF RUPTURE OF THE HEART SHOWING RUPTURE AT THE ANTERIOR ASPECT OF RIGHT ATRIUM WITH SURROUNDING EXTRAVASATION OF BLOOD**



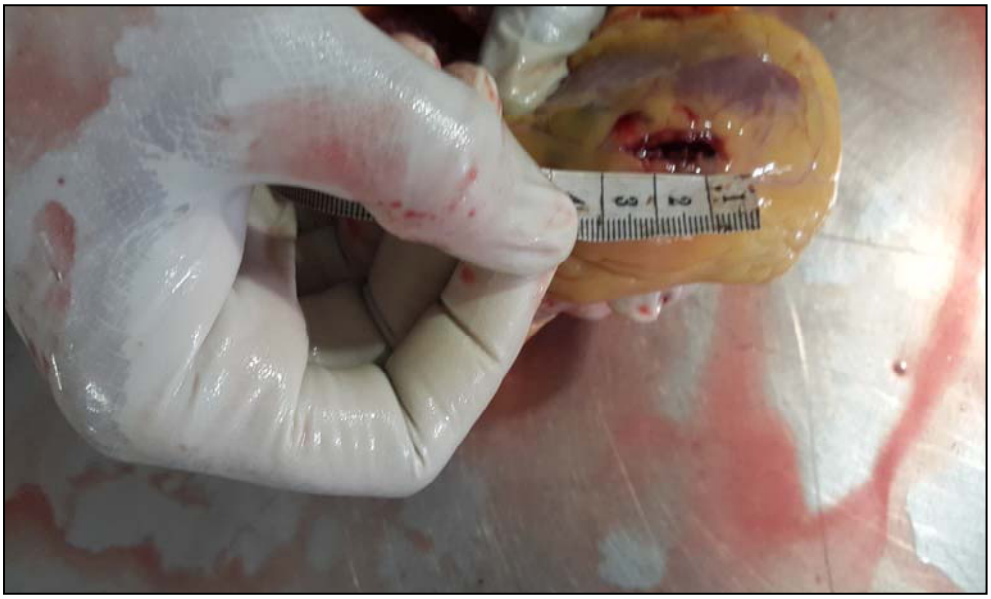
3. **A CASE OF RUPTURE OF THE HEART SHOWING RUPTURE AT THE ANTERIOR ASPECT OF RIGHT VENTRICLE WITH SURROUNDING EXTRAVASATION OF BLOOD**



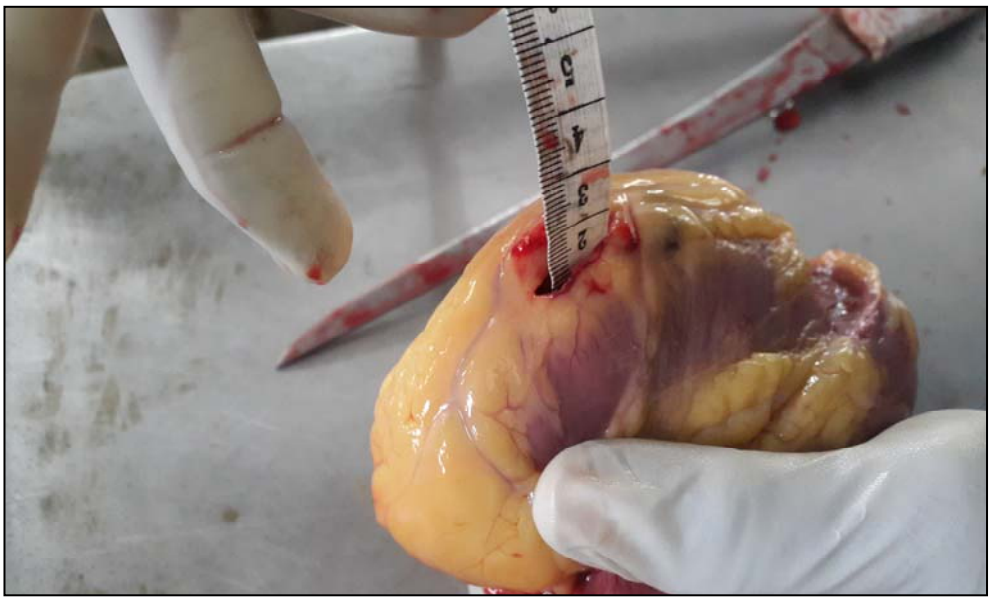
4. **A CASE OF RUPTURE OF THE HEART SHOWING RUPTURE AT THE ANTERIOR ASPECT OF RIGHT VENTRICLE WITH SURROUNDING EXTRAVASATION OF BLOOD**



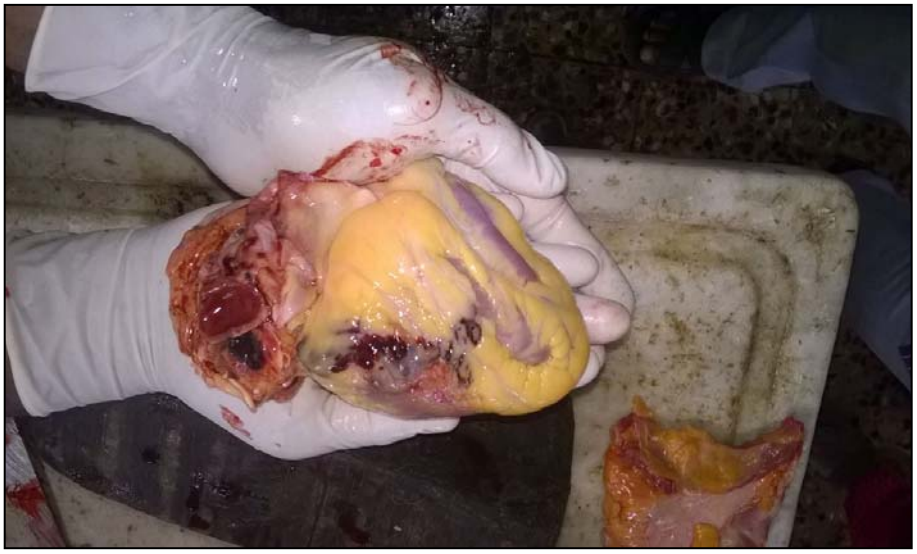
5. **A CASE OF RUPTURE OF THE HEART SHOWING RUPTURE AT THE ANTERIOR ASPECT OF RIGHT VENTRICLE WITH SURROUNDING EXTRAVASATION OF BLOOD**



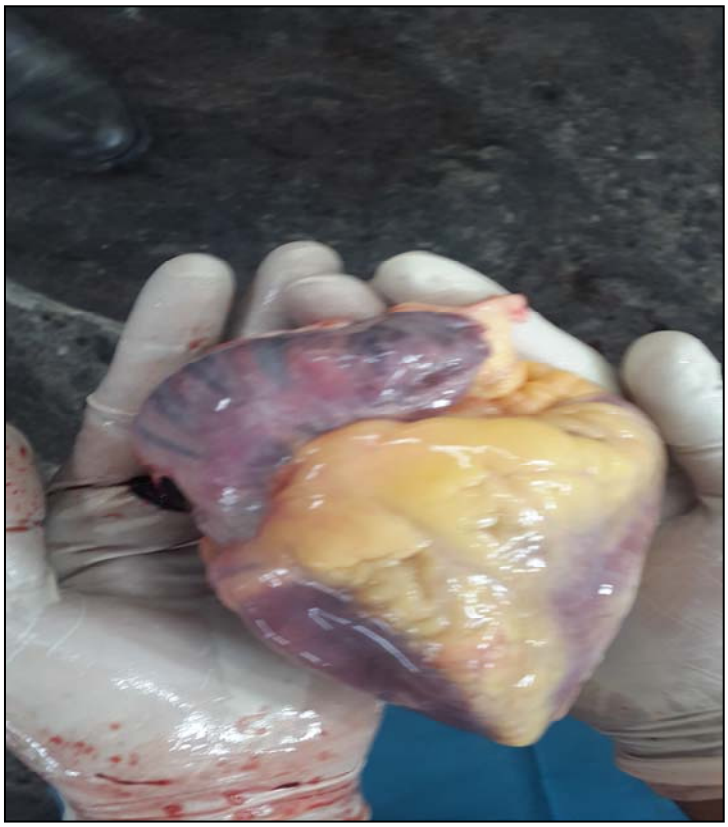
6. **A CASE OF RUPTURE OF THE HEART SHOWING RUPTURE AT THE ANTERIOR ASPECT OF RIGHT VENTRICLE WITH SURROUNDING EXTRAVASATION OF BLOOD**



7. **A CASE OF RUPTURE OF THE HEART SHOWING RUPTURE AT THE ANTERIOR ASPECT OF RIGHT VENTRICLE WITH SURROUNDING EXTRAVASATION OF BLOOD**

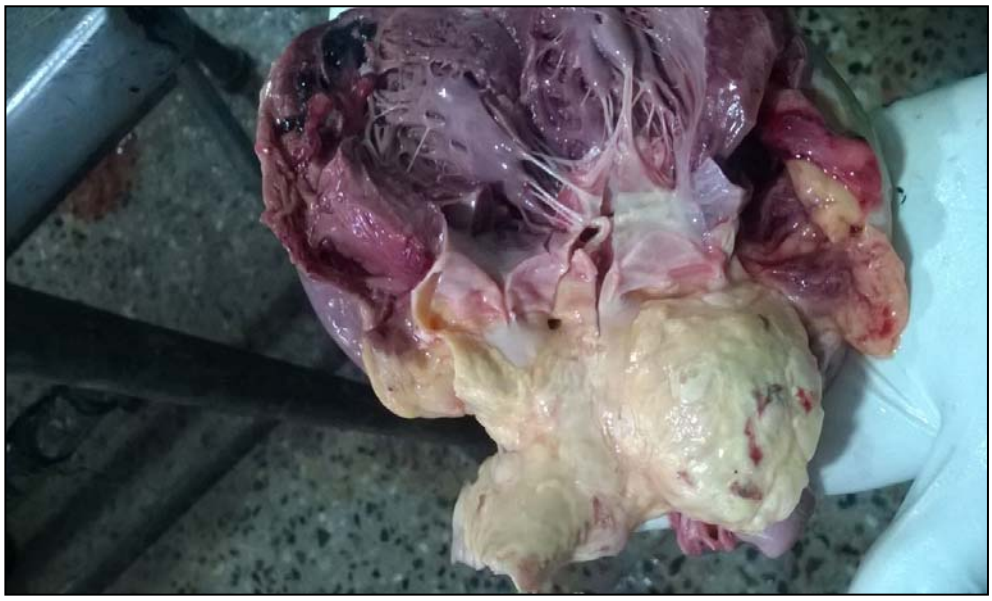


8. **A CASE OF ISCHAEMIC HEART DISEASE SHOWING ENLARGED RIGHT ATRIAL APPENDAGE.**

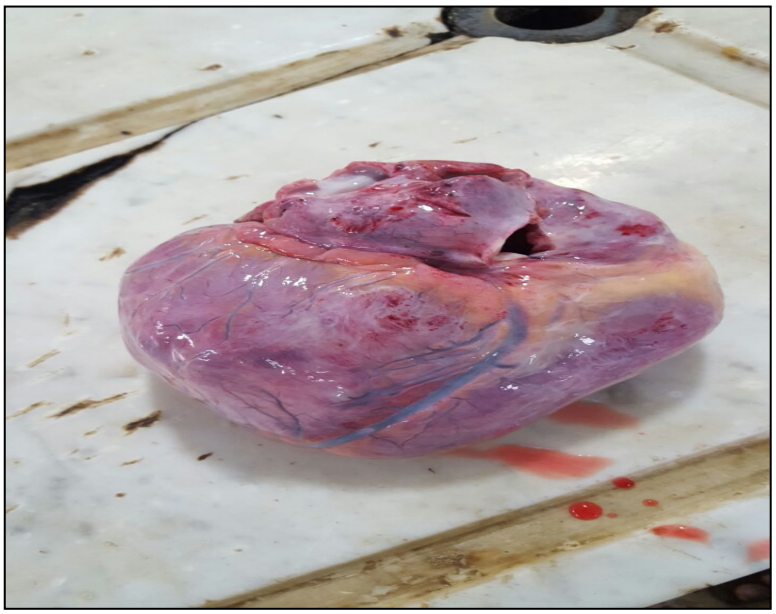


9. A CASE OF HYPERTROPHIC CARDIOMYOPATHY SHOWING INCREASED THICKNESS OF LEFT VENTRICLE.

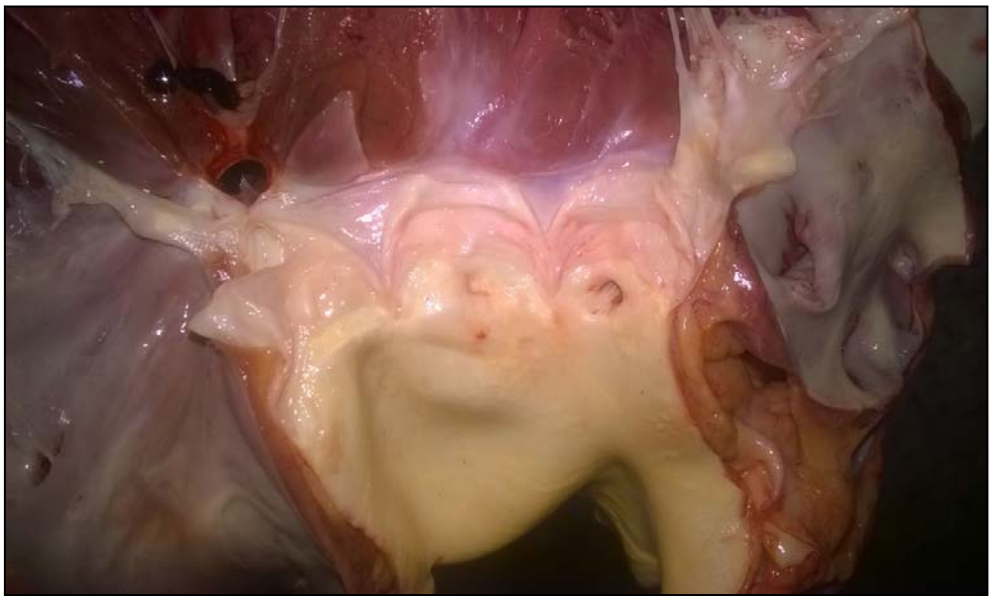
10. A CASE OF ATHEROSCLEROSIS OF AORTA SHOWING MULTIPLE ATHEROMATOUS PLAQUES IN THE ROOT OF AORTA.



11. A CASE OF HYPERTROPHIC OBSTRUCTIVE CARDIOMYOPATHY – EXTERNAL APPEARANCE (FOOT BALL SHAPE OF THE HEART)

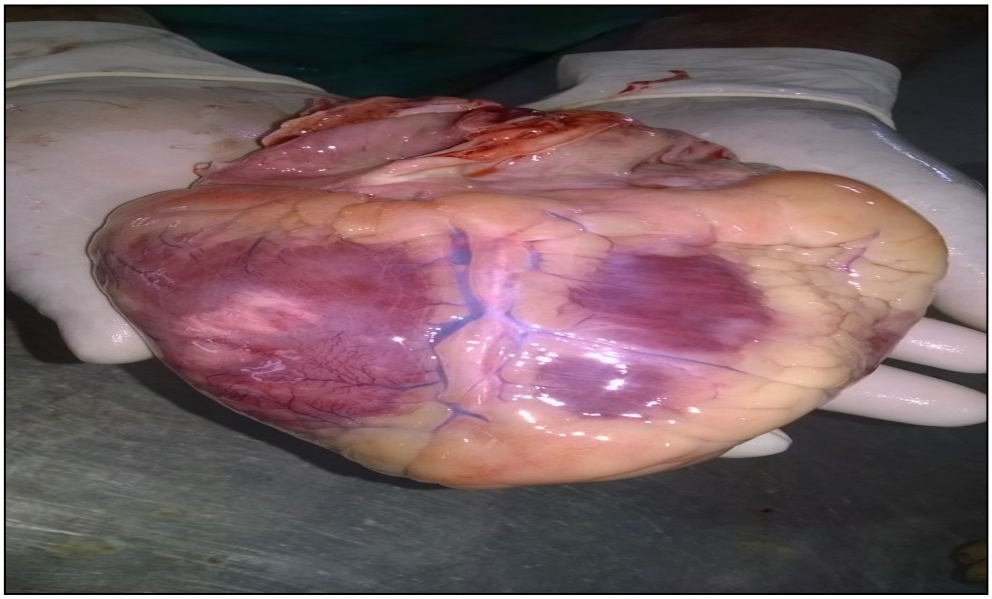


12. CUT OPENED SECTION OF THE HEART SHOWING OCCLUSION OF LEFT CORONARY ARTERY

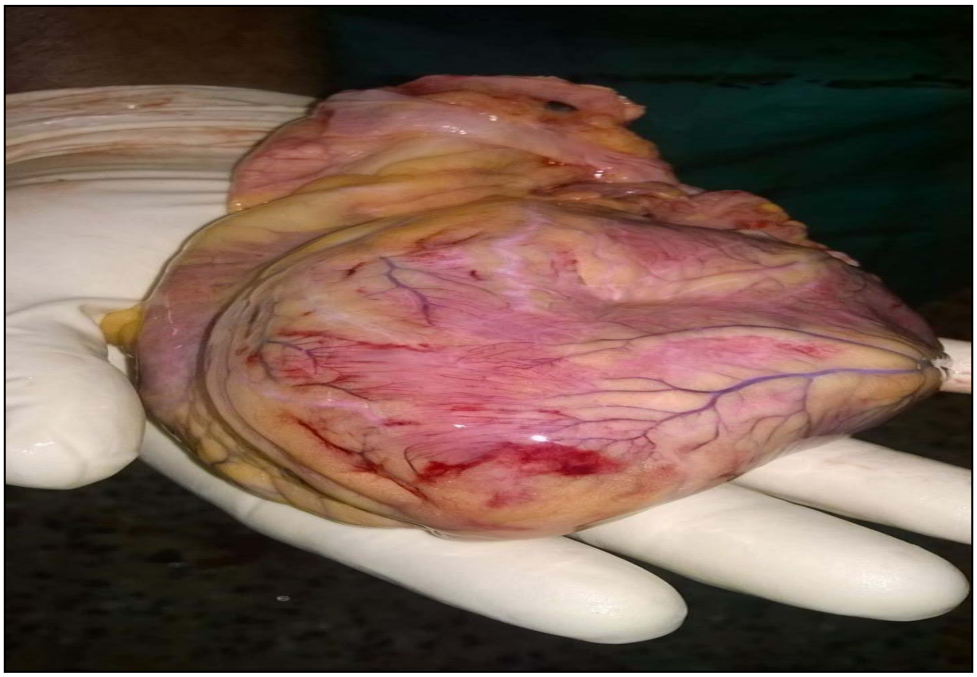


13. CUT OPEN SECTION OF THE HEART SHOWING PAPILLARY MUSCLE AND CHORDAE TENDINAE.

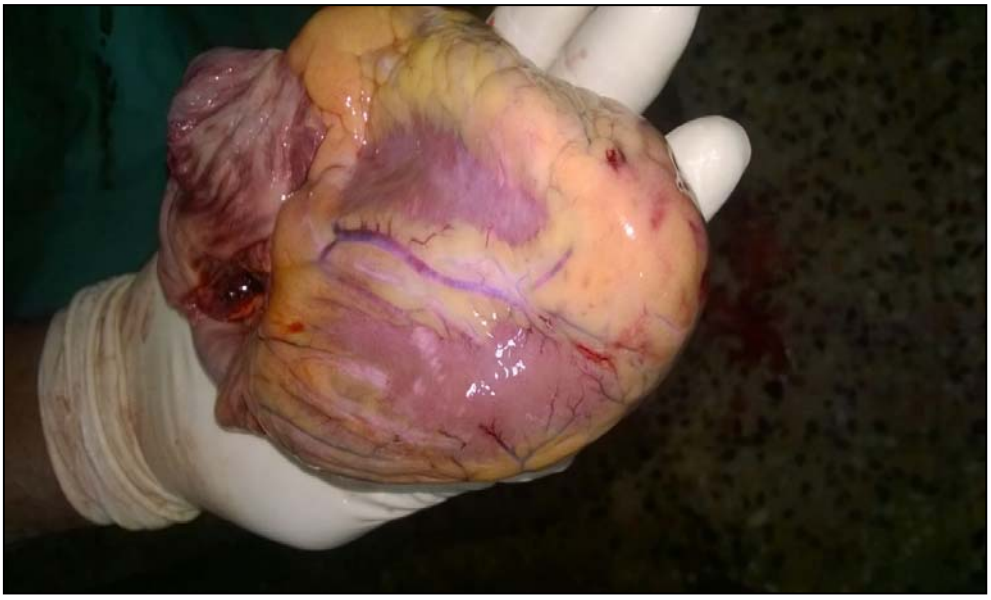
14. HEART SHOWING OLD ISCHAEMIC CHANGES OVER THE ANTERIOR ASPECT OF RIGHT VENTRICLE.



15. HEART SHOWING OLD ISCHAEMIC CHANGES IN THE RIGHT VENTRICLE

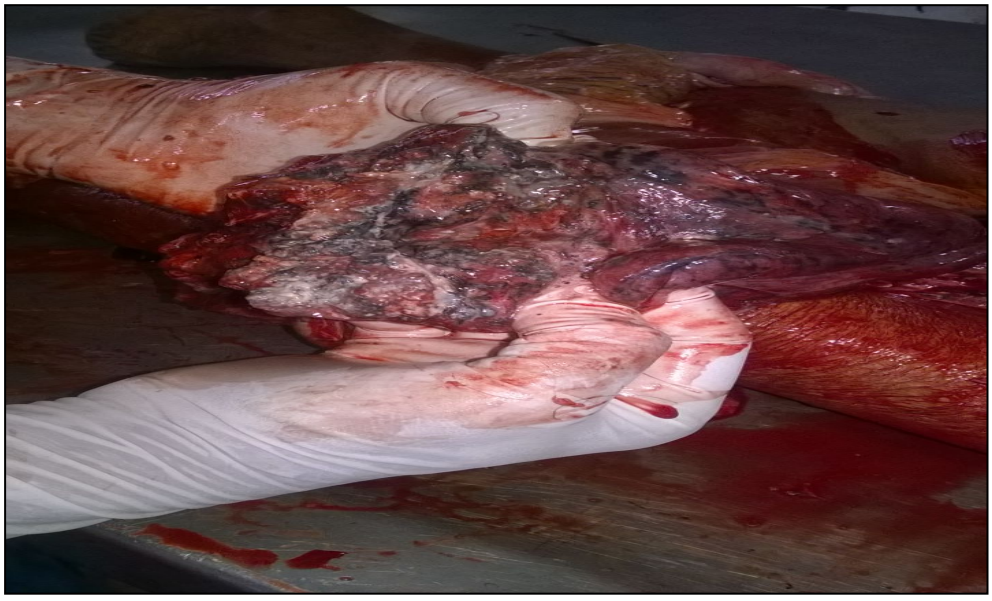


16. HEART SHOWING OLD ISCHAEMIC CHANGES IN THE RIGHT VENTRICLE

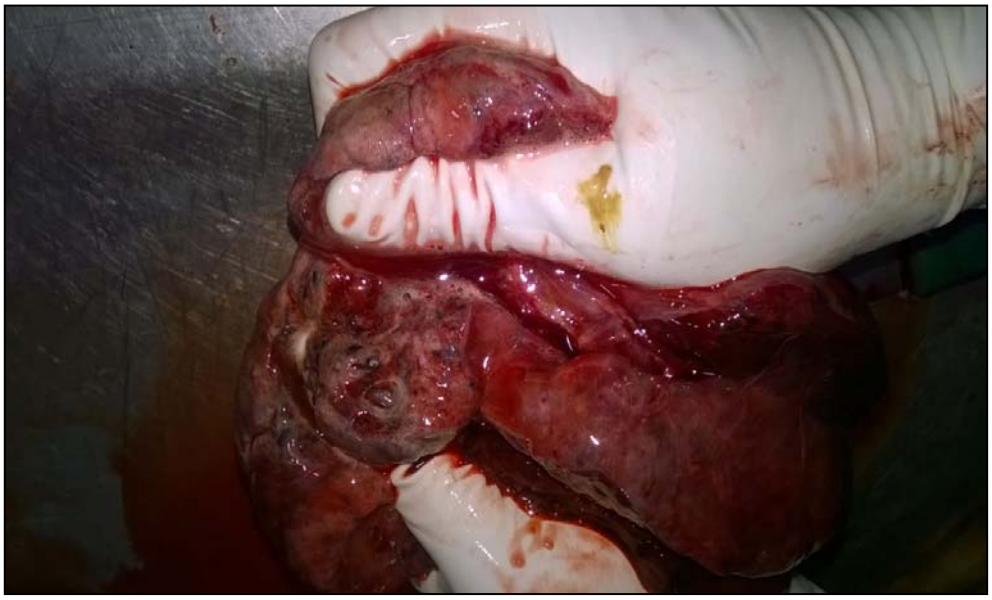


17. LEFT LUNG ADHERENT TO CHEST WALL

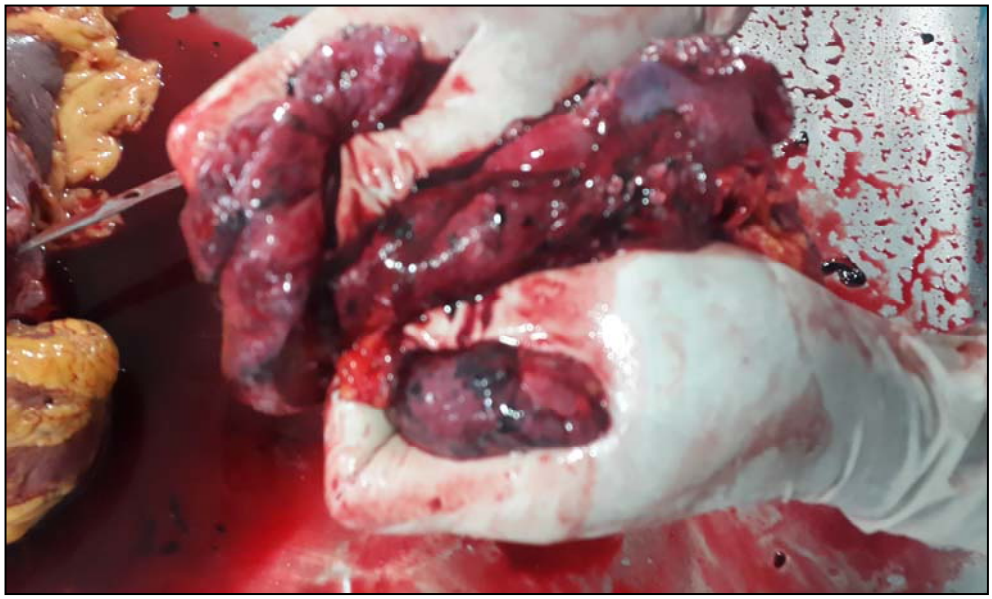
18. A CASE OF PULMONARY TUBER CULOSIS SHOWNING MULTIPLE CAITIES IN THE UPPER LOBE OF THE RIGHT LUNG



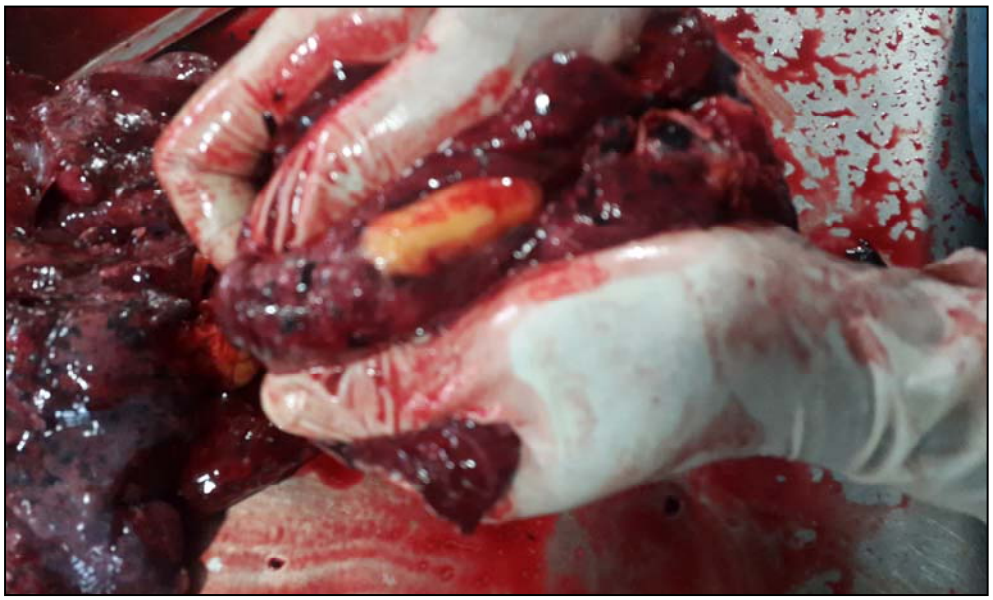
19. A CASE OF TUBERCULOSIS SHOWING PUS ON CUT SECTION OF THE LUNGS.



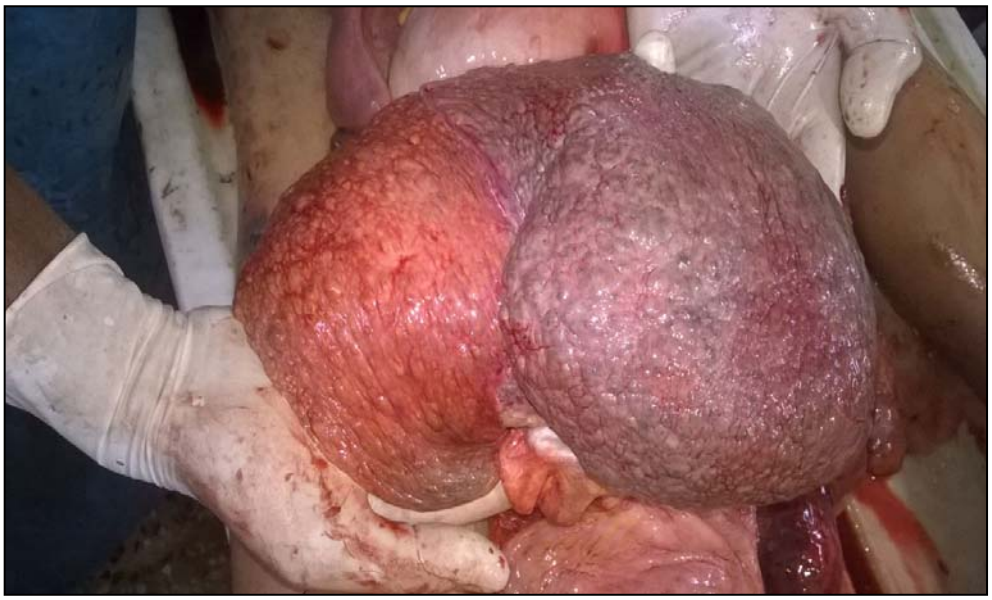
20. A CASE OF EMPHYSEMATOUS LUNG SHOWING PULMONARY OEDEMA



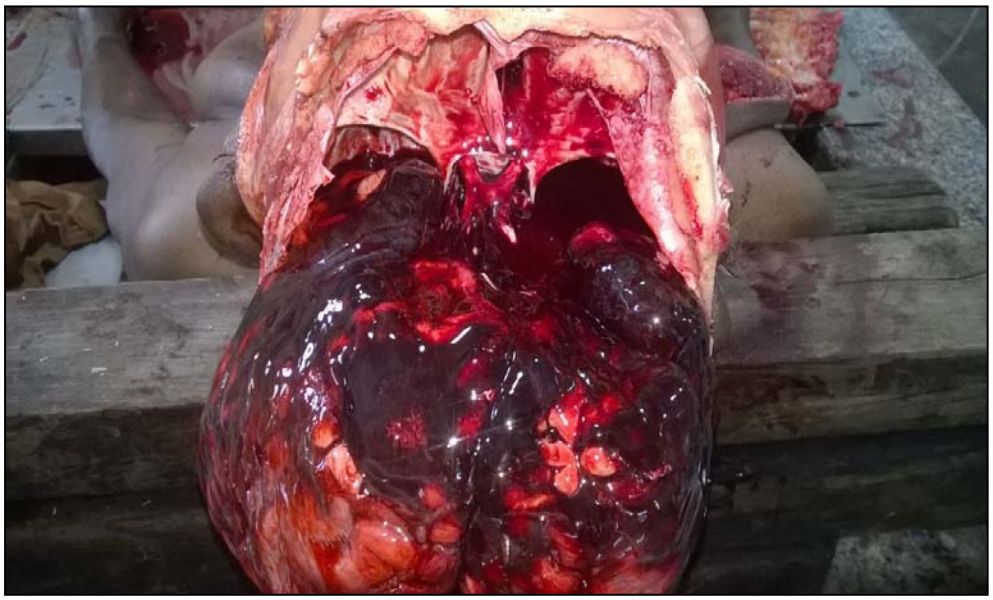
21. A CASE OF PNEUMONIA SHOWING PULMONARY OEDEMA



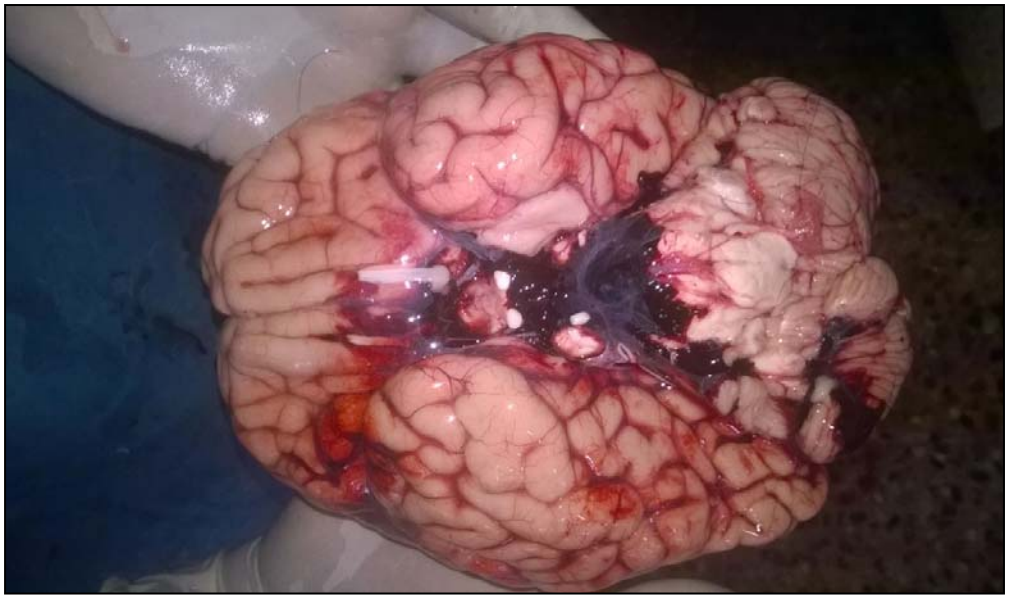
22. CIRRHOSIS OF LIVER



23. RUPTURE OF BERRY ANEURYSM IN THE CIRCLE OF WILLIS

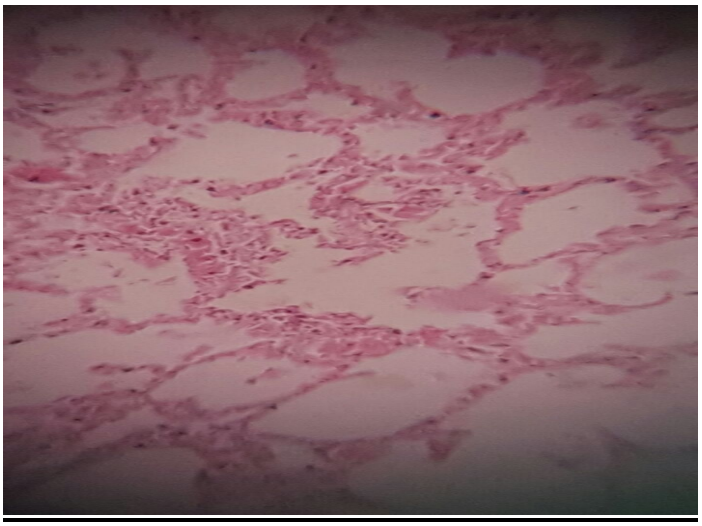


24. RUPTURE OF BERRY ANEURYSM IN THE CIRCLE OF WILLIS

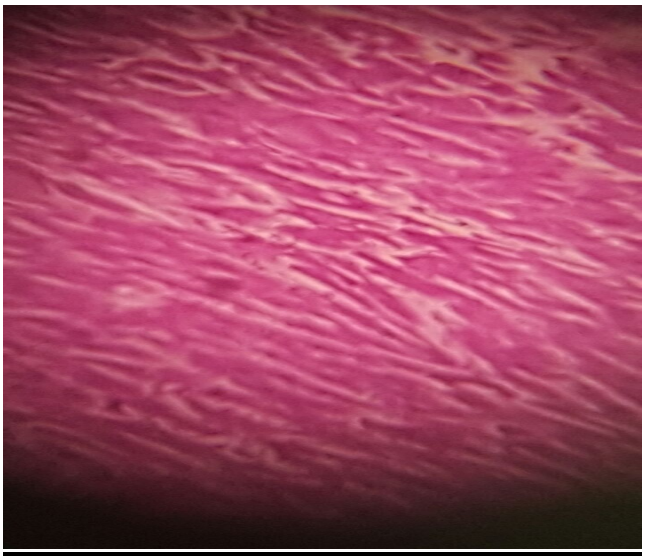


HISTOLOGY PHOTOGRAPHS

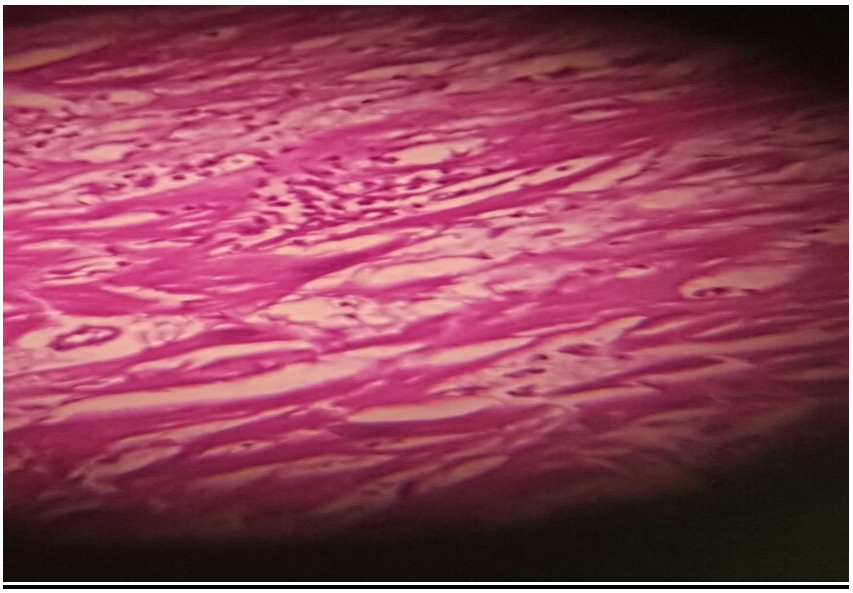
1. **HISTO - PATHOLOGYPHOTOGRAPH SHOWING PANACINAR (PANLOBULAR) EMPHYSEMA SHOWING INVOLVEMENT OF THE ENTIRE LOBULES AND WHOLE OF ACINUS.**



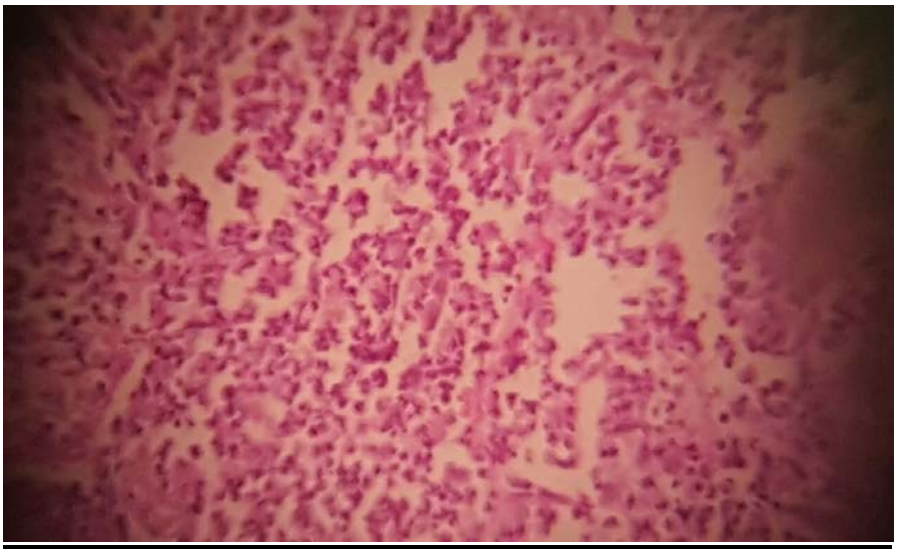
2. **RECENT MYOCADIAL INFARCTION WIYHIN 24 HOURS SHOWING SHRUNKEN EOSNOPHILIC CYTOPLASM AND PYKNOSIS OF THE NUCLEI. THE NEUTROPHIC INFILTERATE AT THE MARGIN OF THE INFARCT IS SLIGHT.**



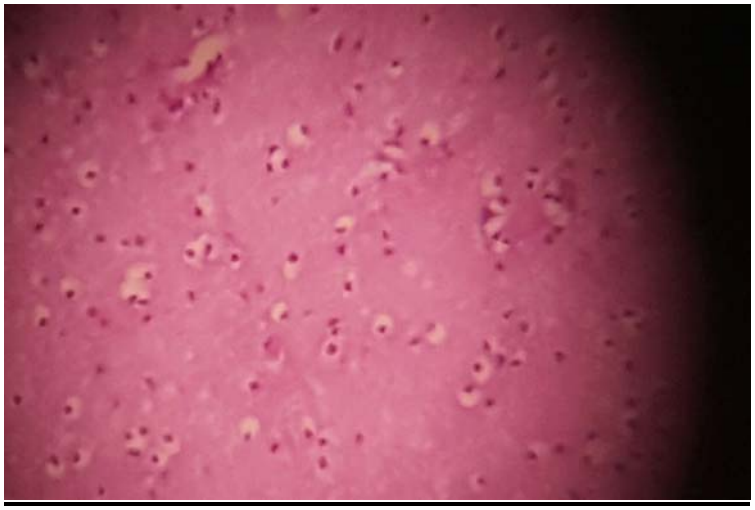
3. **HISTO-PATHOLOGICAL PHOTOGRAPH SHOWING PATCHY MYOCARDIAL FIBROSIS ESPECIALLY AROUND SMALL BLOOD VESSELS IN THE INTERSTITIUM. THE INTERVENING SINGLE CELLS AND GROUP OF MYOCARDIAL CELLS SHOWS MYOCYTOLYSIS.**



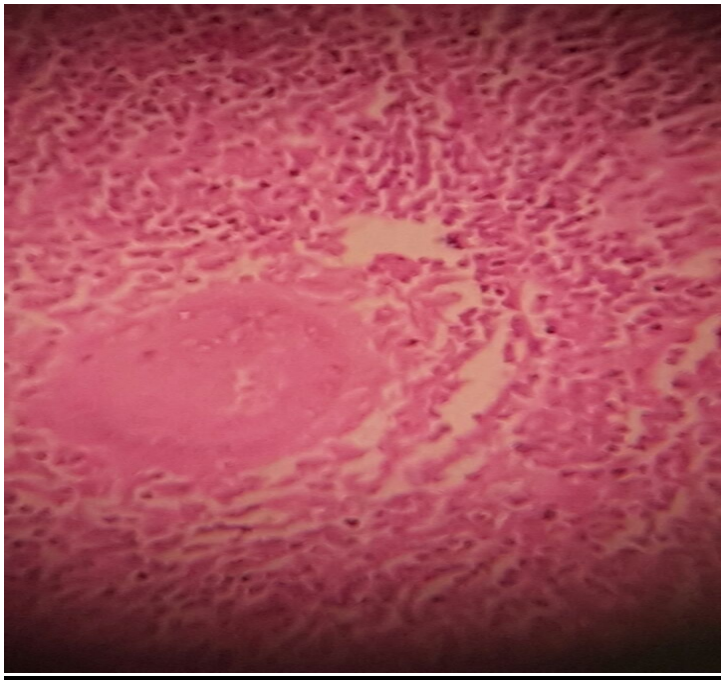
4. **HISTO – PATHOLOGICAL PHOTOGRAPH OF LOBAR PNEUMONIA SHOWING DILATED ALVEOLI, WIDENED SEPTA, OEDEMA FLUID WITH SOME RBC AND POLYMORPHO NEUTROPHILS**



5. **HISTO- PATHOLOGICAL PHOTOGRAPH SHOWING REACTIVE GLIOSIS, NEOVASCULARISATION, CYSTIC SPACE AND CHRONIC INFLAMMATION IN A CASE OF SEIZURE DISORDER.**



6. **HISTO-PATHOLOGICAL PHOTOGRAPH OF A CASE OF PULMONARY TUBERCULOSIS SHOWING CAEATION NECROSIS, FIBROSIS, ALVEOLAR SACS, EPITHELOID CELLS AND LANGHAN'S GIANT CELLS.**



DISCUSSION

In my study maximum number of deaths i.e., 42 Cases (42%) occurred in the age group of 36-40 Years, followed by 30-35 Years age group. Where as in the study conducted by review no 1,2, 13, 14 Maximum number of deaths occurred in the age group of 36-40 years followed by 31-35 years. This clearly indicates that the age group of 30 years – 40 years where at high risk for Sudden death. Determination of the cause of death in younger aged people is difficult to opine and in most of these cases post- mortem examination can provide valuable information in the interest of the society and public health by identifying the risk group and major contributory causes of death in these victims.

In my study maximum number of cases who died due to sudden death, 81 cases (81%) was more in males when compared to that of females which is similar to the study conducted by review no 1,2,3,4,5,6,9,11,12,13,16. This may be due to increased stress pertained to their job and dependence towards any substance abuse in males.

In my study majority of the Sudden Death victims were belonging to Hindu religion which is similar to the study conducted by review no 1,2,3,4,5,6,910. This may be attributed to the nation (India) .Where majority belong to the Hindu religion and also confined to the study locality in which majority were Hindus.

In my study majority (65%) of the victims of sudden death belongs to Urban area which is similar to the study conducted by review no 1, 3, 4, 9, 13, 15. This is because of sedentary life style and westernization adopted by the people in urban area.

In my Study majority of the victims who succumbed to sudden death were married. Which is similar to the study conducted by review no 1,2,3,4,5,6,7,9,13,14. This clearly shows that married people have more amount of responsibility and require adequate money to fulfill their day to day needs. The reason for sudden death in married subjects may be attributed to stress and financial constraints.

In my study majority of the victims who succumbed to sudden death were Laborer's by occupation. Where as in the study conducted by review no 1, 2,3 9, 11. Jobless and sedentary work style people were the victims of study death. The increased incidence of Laborers may be attributed to the type of work (Stressful) as well as increased dependence to alcohol and tobacco consumed to reduce body pain / to relieve tension for the type of work in which they are employed.

In my study majority of the victims who succumbed to sudden death have studied only up to Middle school education (51%), followed by High School Education (18%). Where as in the study conducted by review no 1, 9, 11 Primary School Education was more followed by people who have

completed degree and in good profession. This may be attributed to sedentary life style which was adopted by the qualified professionals for sudden death.

In my study majority of the victims who succumbed to sudden death were addicted to Alcohol (32%), followed by tobacco addiction (22%). Surprisingly those who were not addicted to any substance abuse constituted (29%). Predominantly females. Where as in the study conducted by review no 1, 8,9,15,16 tobacco addiction was the major cause of sudden death followed by alcohol. This clearly proves that substance abuse has got high fatality for sudden death.

Regarding less predominance of substance abuse in females at India may be due to the culture in India whereas in the western literature even female predominance was high for substance abuse which may be attributed to the life style adopted in the west.

In my study maximum incidents of sudden death (34%) occurred during January to March. Which is similar to the study conducted by review no 1,2,3,7,8,9,15,16. In India January to March is considered as Summer Season. The reason for increased incidence of sudden death during this season may be attributed to the heat (Increased Perspiration) resulting in dehydration, stroke etc which may cause sudden death.

In my study maximum incidence of sudden death occurred between 12.00 P.M. to 06.00 A.M (34%), followed by 6.00 A.M. to 12.00 P.M. (28%). Which is similar to the study conducted by review no 1, 9. The high incidence

of sudden death during 12.00P.M – 06.00 A.M could not be explained because maximum data was not available to compare about the time of sudden death. However if vast data is available and the reasons for sudden death pertained to this time period is compiled a good precaution about the timing pertained to sudden death can be explained.

In my study maximum incidence of sudden death took place at home (46%).Which is similar to the study conducted by review no 1,2,3,4,5,12. This clearly says that most of the cases of sudden deaths are silent without any pre-existing symptoms and the person may collapse all of a sudden while relaxing particularly during sleep.

In my study Maximum number of cases died due to sudden death presented with complaints of sudden unconsciousness (46%) as presenting complaints. Which is similar to the study conducted by review no 1, 2 and 3. The sudden unconsciousness may be attributed to any silent pre-existing pathology like aneurysm, Rupture etc. This clinical symptom should be dealt seriously and immediate attention should be drawn so that the necessary investigations shall be taken and treat conservatively/ surgically for the survival of the person.

In my study majority of the cases who died of sudden death was confined to the Cardio Vascular system (47%) which is similar to all my review studies. This indicates that Cardio Vascular system is the most involved system in sudden death cases. This may be attributed to sedentary life style,

lack of exercise and high intake of fatty food substances which may lead to the formation of atheromatous plaques in the vessels there by leading to Hypertension, Myocardial Infarction etc.

In my study majority of the deceased died due to Coronary Arterial Heart Disease (35%) followed by Tuberculosis (10%) and Intracranial Haemorrhage (8%). This is similar to the study conducted by 1,2,3,4,6,7,9,14,15,16. This is due sedentary life style, lack of exercise and high fatty intake leading to the formation of atheromatous plaques. In recent studies it has shown that majority of the people die due to stress resulting in sudden spasm of the coronary arteries there by resulting in coronary stenosis and death.

SUMMARY

1. Maximum number of deaths i.e., 42 Cases (42%) occurred in the age group of 36-40 Years, followed by 30-35 Years age group.

2. Maximum number of cases who died due to sudden death, 81 cases (81%) was more in males when compared to that of females.

3. Majority of the Sudden Death victims were belonging to Hindu religion.

4. Majority (65%) of the victims of sudden death belongs to urban area.

5. Majority of the victims who succumbed to sudden death were married

6. Majority of the victims who succumbed to sudden death were laborers by occupation.

7. Majority of the victims who succumbed to sudden death have studied only up to Middle school education (51%), followed by High School Education (18%).

8. Majority of the victims who succumbed to sudden death were addicted to Alcohol (32%).

9. Maximum incidents of sudden death (34%) occurred during January to March.

10. Maximum incidence of sudden death occurred between 12.00 P.M. to 06.00 A.M (34%), followed by 6.00 A.M. to 12.00 P.M. (28%).

11. Maximum incidence of sudden death took place at home (46%).

12. Maximum number of cases died due to sudden death presented with complaints of sudden unconsciousness (46%) as presenting complaints.

13. Majority of the cases who died of sudden death was confined to the Cardio Vascular system (47%).

14. Majority of the deceased died due to Coronary Arterial Heart Disease (35%) followed by Tuberculosis (10%) and Intracranial Haemorrhage (8%)

CONCLUSION

1. The above observations suggest that most of the Sudden Deaths were due to preventable causes including infections (Tuberculosis, Pneumonia, Bronchiectasis, Emphysema etc.).
2. Involvement of Cardio Vascular System which still appears to be the major risk factors of sudden death was mostly due to mismanaged blood pressure level.
3. Lack of patient awareness, timely seeking health care services and poverty still predominate the majority of deaths in our study population.
4. Before coming to the autopsy diagnosis of sudden death, reliable history along with detailed clinical history from hospital records and previously available health records must be retrieved from the police.
5. A meticulous post-mortem examination along with histo pathological study and detailed toxicological analysis can pinpoint the actual cause of death.
6. Inspite of meticulous post – mortem examination in certain sudden death cases the autopsy becomes negative, because to conclude through histo pathological examination also there is no sufficient time for the markers like inflammatory cells which can be interpreted through microscopic examination. Endocrine disturbances leading to sudden death also cannot be explained because of non-availability of postmortem sample data for comparison confined to that specific area.

RECOMMENDATIONS

1. The incidence of sudden death is about 35% of all cases of death which is really a matter of concern for the death investigators.

2. In a small percentage of cases, there may not be any obvious cause of death. These autopsies are called obscure autopsies. The cause may be endocrine dysfunctions, biochemical disturbances, viral infections etc. which will not always provide positive autopsy findings. In such cases tissues for histo pathological examination along with blood for biochemical, hematological and microbiological examinations are highly required.

3. Though in my one year study period i have not encountered any such cases where cause of death was dependent on such supportive investigations, but I am interested to expand my study period from 1 year to 5 years, so that i could provide a better ideation regarding the causes of Sudden death cases with no known history of any preexisting diseases over a decade, as well as any major change in socio demographic profiles (specially in age and sex distribution) and risk factors.

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