Original Research Article

A clinical study of cerebral hemorrhage in non-diabetic normotensives vs non–diabetic hypertensives and the role of nimodipin

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Abstract

Background: Intra cerebral hemorrhage accounts for 10-15% of all cases of stroke and is associated with highest mortality rate, with only 38% of affected patients surviving the first year.
Materials and methods: All cases of cerebro vascular accident (CVA) presented to Gandhi Hospital, Musheerabad, were considered, and the cases of CVA with intra cerebral hemorrhage evidenced by lumbar puncture and CT scan brain were prospectively studied at Department of Medicine, Gandhi Hospital Muheerabad, between May 2002 and April 2004.
Results: Out of 63 cases, 22 cases were with non-diabetic normotensive, and non-risk factors; and 41 cases were with non-diabetic hypertension; two groups of cases were studied separately and the comparison was made. Total number of 254 Cerebro Vascular Accident (CVA) cases was presented to Gandhi Hospital, Musheerabad. Cerebral thrombosis were 171, Cerebral embolism were 20, and intra cerebral hemorrhage were 63. Out of 63 intra cerebral hemorrhage cases, 41 were hypertensive and 22 were normotensive. Out of 41 hypertensive patients, 23 died and 18 survived; out of 22 normotensive patients, 3 died and 19 survived. All cases were given broad spectrum antibiotic (inj. Ampicillin 500 mg IV 6th hourly, Inj. Flagyl 500 mg IV 8th hourly and Inj. Gentamycin 80 mg IV 12th hourly), Inj. Mannitol 100 mg IV 8th hourly, tablet Nimodipin 30 mg 8th hourly, proper care was taken and appropriate physiotherapy was given.
Conclusion: It is useful to identify the risk factors for intra cerebral hemorrhage in normotensives, because the cause was identified in 5 cases, 4 cases with aneurysm, one case with arterio venous

malformations (AVM's) and most of the cases who belong to 5th and 6th decade, the cause for intra cerebral hemorrhage was not known. Nimodipin was used in all the cases as initial medical treatment, with good results, when compared to 50% mortality in other studies where Nimodipin was not given.

Key words

Cerebral hemorrhage, Non–diabetic normotensives, Non–diabetic hypertensives, Nimodipine.

Introduction

Intracranial hemorrhage includes intraparenchymal hemorrhage, sub arachnoid hemorrhage (SAH), subdural, and epidural hemotomas. Intra parenchymal hemorrhage is the most common type of intracranial hemorrhage. Hypertension (HTN), trauma, cerebral amyloid angiopathy cause the majority of these hemorrhages and aneurysms, arteriovenous malformations, vascular hamartomas are the other causes of intra cerebral hemorrhage (ICH) [1]. Advanced age and heavy alcohol consumption increase the risk, and cocaine use is one of the most important causes in the young [1].

Intra cerebral hemorrhage accounts for 10-15% of all cases of stroke and is associated with highest mortality rate, with only 38% of affected patients surviving the first year [2, 3]. Depending on the underlying cause of bleeding, intra cerebral hemorrhage is classified as either primary or secondary. Primary intra cerebral hemorrhage (PICH), accounting for 78 to 88% of cases, originates from the spontaneous rupture of small vessels damaged by chronic hypertension or amyoid angiopathy. Secondary intra cerebral hemorrhage occurs in a minority of patients in association with vascular abnormalities such as arteriovenous malformations (AVM's) and aneurysms, tumours, or impaired coagulation [2, 4].

Although hypertensive intra cerebral hemorrhage remains the most common form of intra cerebral hemorrhage, underlying vascular abnormalities should always be considered in appropriate circumstances because of the high risk of recurrent hemorrhage and available treatment options [2, 5, 6]. The world wide incidence of intra cerebral hemorrhage ranges from 10 to 20 cases per 1,00,000 population and increases with age. Intra cerebral hemorrhage is more common in men than women, particularly those older than 55 years of age, and in certain populations, including blacks and Japanese [7, 8].

Persons with hypertension who were 30 to 69 years of age and who received standardized antihypertensive therapy had a risk of stroke including intra cerebral hemorrhage. Intra cerebral hemorrhage commonly occurs in the cerebral lobes, basal ganglia, thalamus, brain stem, predominantly pons, and cerebellum. Extension into the ventricles occurs in association with deep, large hematomas [2, 9]. Intra cerebral hemorrhage most commonly involve cerebral lobes, originating from penetrating cortical branches of the anterior, middle or posterior cerebral arteries; Basal ganglia, originating from ascending lenticulostriate branches of middle cerebral artery (MCA), the thalamus from ascending thalamogeniculate branches of posterior cerebral artery (PCA), the pons from paramedian branches of the basilar artery (BA), and the cerebellum from penetrating branches of the posterior inferior, anterior inferior, or superior cerebellar arteries [2, 9].

Hematomas of many sites common to both primary intra cerebral hemorrhage and aneurismal bleeding, difference between the two is [10] frontal and temporal hematomas are common with aneurysms and infrequent with PICH, hematomas of the parietal lobe or corona radiata are frequent with PICH and uncommon with aneurysms, hematomas of occipital region are rare with both diseases, isolated hemorrhages into the thalamus or caudate nucleus are seen

characteristically in PICH, caval, collosal, and bilateral frontal lobe hematomas usually indicate a ruptured aneurysm, and hematomas of external capsule are frequent with both and particularly with PICH.

Although intra cerebral hemorrhage is a less frequent cause of stroke than cerebral infarction, it is more often fatal [11]. It is usually attributed to hypertensive small vessel disease, and the most common sites of hemorrhage are the basal ganglia, cerebellum and pons. Treatment is largely restricted to the control of hypertension and rehabilitation. In some patients with ICH, however the hemorrhage is lobar in location, such as in the frontal, parietal, temporal, or occipital cortex, and such patients often do not have hypertension. This category of hemorrhage referred to as lobar intra cerebral hemorrhage, may represent a distinct pathogenic sub group [11, 12]. Cerebral amyloid angiopathy (CAA), also known as cerebral congophilic angiopathy or cerebral amyloidosis, has been recognised as a frequent cause of lobar intra cerebral hemorrhage, the clinical syndrome is characterised by lobar intra cerebral hemorrhage, with no other definite cause of the hemorrhage [11]. Affected patients are usually over the age of 60 years and may have antecedent memory loss. In the Northern Manhattan stroke study, the annual incidence of lobar hemorrhage was 8.4 cases per 1,00,000 persons over the age of 20 years and lobar hemorrhage accounted for a third of all cases of primary non traumatic intra cerebral hemorrhage. A polypeptide called amyloid β peptide, which originates from the amyloid precursor protein and is encoded by a gene on chromosome 21, has been implicated in the pathogenesis of cerebral amyloidosis [11]. Furthermore, the association between apolipoprotein E polymorphism (ε4 or ε2) and the occurrence of cerebral amyloid angiopathy and intra cerebral hemorrhage suggests the presence of a genetic mechanism [11, 13-15].

Recurrent lobar hemorrhage is the hallmark of cerebral amyloid angiopathy, apolipoprotein genotype as a potential risk factor for recurrent lobar hemorrhage. Cerebral amyloid angiopathy is defined by the deposition of congophilic material in vessels of the cortex and leptomeninges, and is the major cause of ICH in elderly [4, 6]. Cortical and subcortical regions are common sites because where the vascular amyloid deposits are most frequent, and occur less commonly in the cerebellum [6].

The main difference between hypertensive hemorrhage and amyloid angiopathy hemorhage is, in the former most common site is basal ganglia, occur during day time activities and rupture into the ventricle, but in the latter commonest site is lobar region, occur at night and rupture into the sub arachnoid space [4, 11].

Occurrence of mucoid degenerative changes in the aneurysm wall,similar to mucoid vasculopathy identified by BJ Rajesth, Sandhya mani, et al. [16]. Mucoid changes were commonly seen in patients with 5th and 6th decades, this mucoid and elastic tissue degeneration or mucoid arteriosclerotic vasculopathy, weakens the vessel wall and it may lead to formation of aneurysm. When this change combined with risk factors, risk of formation of aneurysm is further high [16].

Arteriovenous malformations are a complex tangle of abnormal arteries and veins, arteriovenous malformations are commonly grouped and treated according to their appearance on angiography (angio architecture). Small arteriovenous malformations located on the surface of the brain are the best candidates for direct surgical removal. Large arteriovenous malformations are wedge shaped with the base in the cortex and the bulk of the abnormality extending deeper into the brain. Malformations confined to the cortex almost always drain superficially through cortical veins; Malformations in the basal ganglia, thalamus, and brain stem are usually fed in the small, deep, perforating arteries. Arteriovenous malformations of the brain typically present before the age of 40 years and affect both sexes in nearly equal proportions. Intracranial

hemorrhage is the most common clinical presentation of arteriovenous malformations with a reported frequency ranging from 30 to 82 percent [17].

Pathogenesis and pathology of intra cerebral hemorrhage

Brain hemorrhage is often divided into two broad categories, intra parenchymal and sub arachnoid space, denoting the site of vascular rupture. They are associated with diverse pathologic conditions. Combination of the two may occur. ICH may rupture into a ventricle and reach the sub arachnoid space (SAS) via the 4th ventricle or the blood from a ruptured aneurysm in the SAS may dissect into the adjoining brain. Vascular malformations may be a source of hemorrhage and often induce both sub arachnoid hemorrhage and intra cerebral hemorrhage.

Acute hemorrhages are characterized by extravasations of blood with compression of adjacent parenchyma. Old hemorrhages show an area of cavitary destruction of brain with a rim of brownish discolouration. Microscopically the early lesion is characterized by a central core of clotted blood surrounded by a rim of brain tissue showing anoxic neuronal and glial changes as well as edema. Eventually edema resolves, pigment laden and lipid laden macrophages appear and proliferation of reactive astrocytes is seen at the periphery [22].

Sub arachnoid hemorrhage

Sub arachnoid hemorrhage due to ruptured berry aneurysm is most frequent in the fifth decade (about one third) and is slightly more frequent in females. In the early post sub arachnoid hemorrhage period, regardless of the etiology of the hemorrhage, there is an increased risk of vasospastic injury involving vessels other than those originally injured. Berry aneurysm measure from a few millimeters to 2 or 3 cms and have a bright red, shiny surface and a thin translucent wall [22]. Hypertension is the most probable contributory factor to the death of patients with SAH. Previous SAH from a particular aneurysm

were more likely to be associated with rupture into the brain [23, 24].

Risk factors for intracerebral hemorrhage

Intracerebral Hemorrhage include primary (Hypertensive) intra cerebral hemorrhage [17], ruptured saccular aneurysm, ruptured arterio-venous-malformations (AVM), hemorrhagic disorders (leukemia, aplastic anemia, thrombocytopenic purpura, anticoagulant therapy), septic embolism, mycotic aneurysm, hemorrhagic infarction, arterial or venous, inflammatory disease of arteries and veins, arterial amyloidosis, neoplasm, vascular hamartoma [17] (Telangiectasis, Cavernous angioma, and Arterio venous hamartoma), advanced age increase the risk [1], heavy alcohol consumption increase the risk [1], and cocaine use is one of the most important cause in young [1].

Primary (hypertensive) intra cerebral hemorrhage

Hypertensive intra cerebral hemorrhage results from spontaneous rupture of a small penetrating artery deep in the brain. The most common sites are the basal ganglia (putamen, thalamus and adjacent deep white matter) deep cerebellum and pons. Most hypertensive hemorrhage develops over 30 – 90 minutes and, within 48 hours macrophages begin to phagocytise the hemorrhage at its outer surface. After 1 to 6 months the hemorrhage is generally resolved to a slit like orange cavity lined with glial scar and hemosiderin laden macrophages [1]. The causative abnormality must be present in relation to one or more causes of the circulating blood, the vessel wall, and the surrounding parenchyma [25]. Cerebral arteries are weaker than most other arteries in that the amount of medial muscle is slight, there is no external elastic lamina and but little adventitia; with a damaged internal elastic lamina, hemorrhage may occur first into the Virchow – Robin space to form a miliary aneurysm and later into the cerebral tissue [25]. This is the common well known "Spontaneous" brain hemorrhage, although it occurs rarely with levels of blood pressure in the

normal range and sometimes with levels of only 150/90 to 170/90 mm of Hg. In most cases the levels are much higher. If the hemorrhage is large midline structures are displaced to the opposite side and vital centers are compressed, leading to coma and death. Nature of vascular lesion leads to arterial rupture is not fully known. But segmental lipohyalinosis and the false aneurysm of Charcot-Bouchard may be responsible [17].

Haemorrhagic disorders
Leukemia, aplastic anemia, thrombocytopenia and anticoagulant therapy associated with intracranial hemorrhages. ICH due to anticoagulant therapy can occur at any location, they are often lobar or sub dural and this hemorrhage may evolve slowly over 24 to 48 hours. Coagulopathy should be reversed with fresh-frozen plasma and vit-k to limit the volume of hemorrhage. When ICH is associated with thrombocytopenia (platelet count <50,000/ul) transfusion of fresh platelets is indicated. ICH associated with hematologic disorders (leukemia, aplastic anemia, thrombocytopenic purpura) can occur at any site and may present as multiple intra cerebral hemorrhages [1].

Neoplasm
Hemorrhage into a brain tumour may be the first manifestation of neoplasm. Choriocarcinoma, malignant melanoma, renal cell carcinoma, and bronchogenic carcinoma are among the most common metastatic tumours associated with ICH. Glioblastoma multiforme in adults and medullo blastoma in children may also have areas of ICH [1].

Advanced age and heavy alcohol consumption
Advanced age and heavy alcohol consumption increase the risk. Excessive use of alcohol increases the risk of ICH by impairing coagulation and directly affecting the integrity of cerebral vessels [2, 26, 27].

Cocaine induced stroke
It is an important cause of stroke, particularly in patients <40 years of age. ICH, ischemic stroke and SAH are all associated with cocaine use. Angiographic findings vary from completely normal arteries to large vessel occlusion or stenosis, vasospasm or changes consistent with vasculitis. Mechanism of cocaine related stroke is not known but cocaine enhances sympathetic activity causing acute sometimes severe hypertension and this may lead to hemorrhage. Slightly more than half of cocaine related ICH are intra cerebral and the rest are SAH [1].

Inflammatory diseases of arteries and veins
Inflammatory diseases of arteries and veins especially polyarteritis nodosa and lupus erythematosus are sometimes associated with hemorrhage into the nervous system. In these disorders rupture of a vessel may occur on the basis of HTN or local vascular disease [17].

Vascular hamartomas
Vascular hamartomas like telangiectasis, cavernous angioma and arterio-venous hamartoma associated with ICH. The hamartomas consist of a collection of abnormal arterial and venous channels and the hemorrhage is probably caused by rupture of a thin walled dilated venous channel. Russell (1954), Crawford and Russell (1956) described 'Cryptic' hamartomatous lesions of the brain. In Russell's (1954) series out of 461 ICH cases 20 were due to AV hamartomas and other cause (total 21) was due to multiple cavernous hemangiomas [23, 24].

Arterial amyloidosis
Cerebral amyloid angiopathy is a disease of the elderly in which arteriolar degeneration occurs and amyloid is deposited in the walls of the cerebral arteries but not elsewhere. Amyloid angiopathy causes both single and recurrent lobar hemorrhages and is probably the most common cause of lobar hemorrhage in the elderly. This disorder is suspected in patients who present with multiple hemorrhages and infarcts over several months to years but is definitively diagnosed by demonstration of Congo-red staining of amyloid in cerebral vessels [1].

Aneurysm

The word aneurysm means a dilatation of an artery. In relation to the central nervous system we are concerned with [25], Congenital saccular aneurysms, Mycotic aneurysms, Atherosclerotic fusiform aneurysms, Spontaneous and post traumatic aneurysms between the internal carotid artery and the cavernous sinus.

Congenital saccular aneurysms

Although aneurysms have been recorded at the extremes of life, they most commonly cause death during the 5th decade. Almost all Saccular aneurysms arise at or very close to the point of division of arteries. The commonest site is the MCA at its first or second point of branching in the Sylvian fissure (30%); next common site is the junction of an ACA and anterior communicating artery (25%); next terminal portion of the ICA and its branches (20%) and basilar artery and its branches. The blood from a ruptured saccular aneurysm may pass [25] into the SAH, into the overlying brain, but more often ploughing through the substance of the brain to enter and fill the ventricular system, much less commonly into sub dural space.

Etiological factor in the development of these aneurysms is aplasia or hypoplasia of the muscle coat at the point of branching or junction of the arteries. Charcot and Bouchard (1868) investigated cerebral hemorrhage and described saccular and fusiform aneurysms on small cerebral arteries probably 250 - 400μm in diameter [23]. Micro aneurysms were also present in 10 out of 35 brains from normotensives [23]. Apart from SAH intra cerebral hematoma is an important consequence of rupture of a berry aneurysm. Arterial cerebral aneurysms more often produce coma and ICH than aneurysms elsewhere.

Mycotic Aneurysm

A term introduced by Osler in 1875, an aneurysm caused by localised bacterial or fungal inflammation of an artery [25]. A mycotic aneurysm forms when the wall of an artery is weakened from within by pyogenic bacteria. The bacteria usually reach the affected portion of the wall in an infected embolus. The commonest source of such emboli is aortic valves in infective endocarditis less commonly pulmonary suppuration and pyaemia. The aneurysms are usually found on the branches of the MCA; especially in the lateral fissure [25]. When the elastica and the media are sufficiently damaged, the wall of the artery shows a localised dilatation, which may progress to rupture and hemorrhage into the sub arachnoid space (SAS) or into the nervous tissue. Mycotic aneurysms are usually small and rarely heal; and there will be a varying amount of ischemic necrosis of nervous tissue in the field of distribution of the artery.

Ruptured saccular aneurysm is the fourth most frequent cerebro vascular disease (CVD) following atherothrombosis, embolism and hypertensive ICH. Saccular aneurysms vary in size from 2 mm to 2 or 3 cms in diameter, averaging 8 to 10 mm [25].

Unruptured aneurysms

The international study of un ruptured aneurysm (ISUA) [28, 29], enables the identification of the factors in aneurysmal rupture which are three in number: Earlier hemorrhage, Size of hemorrhage, Age of the patient.

The risk of bleeding is directly correlated to the size of the aneurysm: The location of the aneurysm is also predictive factor for rupture, aneurysms of the carotid ending; the arteria communicants posterior and the arteria basilaris have a higher risk of rupture than other aneurismal locations. The hemorrhagic risk is inversely correlated to the age of the patient, there are two modalities of treatment one is Surgery. A recent Meta analysis centering on 2460 aneurysms, which were operated upon shows a mortality rate of 2.6% and morbidity rate of 10.9%. Clipping under sight control is presumed to be a guarantee for an anatomical cure. Endovascular treatment of intracranial aneurysms, it has recourse to Guglielmi detachable microcoils (GDC), the clinical and angiographic results reported and suggest that

this technique is associated with a lower rate of complications than that of surgery [28].

Ruptured aneurysms

The early treatment of ruptured intra cranial aneurysm is based on the increased risk of rebleed in the first few days of meningeal hemorrhage. The prognosis is fixed at the outset by the clinical score (Scale of measurement of the World Federation of NeuroSurgery-WFNS). As far as the aneurysm is concerned, three parameters are to be taken into account: The location of the aneurysm, The size of the sac and, The size of the neck of the aneurysm. The totality of the aneurysms of the posterior cavity comes under endovascular techniques, as it could involve aneurysms of the vertebral artery, originating from the PICA, the basilar artery or its branches, and the rate of complete occlusion is 94.6% at 7.2 months. As far as the aneurysm of anterior circulation concerned the results are more difficult to appreciate. Ruptured aneurysm of size less than 15 mm and those between 15 and 25 mm, have a complete occlusion rate of 70.8% for aneurysm in the first category and 33% in the second category [30].

Stable aneurysms

The complete exclusion or the unchanged aneurismal remnant in relation to the initial treatment represents 86.4%, which are controlled between the 6th and 65th post operative month [28].

Unstable aneurysms

There is a progression of the aneurysmal remnant at the neck or a re-expansion of the aneurysmal sac. Unstable aneurysms should be subjected to secondary treatment by embolisation or surgery. The annual risk of rebleed is 0.8% in the first year, 0.6% in the second year and 2.4% in the third year. Stable aneurysms or aneurysms that have benefited from a second operation must be followed up by angio-MRI at 3 years and at 5 years after the initial treatment [28, 31].

Giant aneurysms

Giant aneurysms do not respond to coiling techniques. The combination of different endovascular modalities, stenting and coiling, stenting and occlusion has been developed for treating some types of giant aneurysms [28].

Arteriovenous malformations of the brain

An arterio venous malformation (AVM) consists of a tangle of dilated vessels, which form an abnormal communication between the arterial and venous system. It is a developmental abnormality representing persistence of an embryonic pattern of blood vessels. An AVM was apparently recognised by the Egyptians as early as 1500 B.C. Steinfneil in 1895 and Isenschmid in 1912 were the first to make clinical diagnosis of a cerebral AVM [17]. AVMs occur in all parts of the brain, brainstem, and spinal cord, but the larger ones are more frequently found in the central part of the cerebral hemispheres. It is equally frequent in males and females. The natural history of AVMs has been studied by Crawford, et al. [17], from a group of 343 patients, 42% patients had hemorrhage. A systolic bruit heard over the carotid in the neck or over the mastoid process or eyeballs in a young adult is almost pathognomonic of AVM. The blood pressure is elevated or normal. Occurrence of intracranial hemorrhage with normal BP should raise the suspicion of an AVM, ruptured saccular aneurysm, a bleeding diathesis; cerebral vessel amyloidosis or hemorrhage into a tumour. Fully 95% of AVMs are disclosed by CT scan if a contrast study is done. Arteriography establishes the diagnosis with certainty and will demonstrate AVMs larger than 5 mm in diameter. The first hemorrhage due to AVM is fatal but in more than 90% of cases the bleeding stops and the patient survives. Hemorrhage obviously occurs when a vessel of an AVM bursts [17].

Symptomatology

Although clinical manifestations not particularly associated with exertion, intra cerebral hemorrhage almost always occurs while the patient is awake, and sometimes when stressed. The hemorrhage generally presents as the abrupt

onset of focal neurologic deficit. The focal deficit typically worsens steadily over 30 to 90 min and is associated with a diminishing level of consciousness and signs of increased intra cranial pressure (ICP), such as headache and vomiting. Clinical features mainly depend on the site of hemorrhage [32].

Clinical types
Cortical and sub cortical
Effusions confined to cortical grey matter are extremely rare, the presence of blood on, in, or just under the cortex, especially if at, or near, the motor region, is likely to cause fits and is accompanied by early, persisting rigidity.

Capsular
The danger zone comprises the capsule and basal ganglia, where bleeding is very common. Symptoms depend on whether anterior, geniculate, or posterior division of the capsule is concerned. Contra lateral hemi paresis is the sentinel sign. The face sags on one side over 5 to 30 min, speech becomes slurred, arm and leg gradually weaken, and the eyes deviate away from the side of the hemi paresis. When the hemorrhages are large, drowsiness gives way to stupor as signs of upper brainstem compression appear. Coma ensues accompanied by deep, irregular, or intermittent respiration, a dilated and fixed ipsilateral pupil, bilateral Babinski signs, and decerebrate rigidity [32].

Thalamic hemorrhages
Also produce a contra lateral hemiplegia or hemiparesis from pressure on, or dissection into the adjacent internal capsule. Sensory deficit involving all modalities is usually present. Aphasia with preserved verbal repetition may occur after hemorrhage into the dominant thalamus and apractognosia or mutism occurs in some cases of non dominant hemorrhage. Homonymous visual field defect and several typical ocular disturbances occur. These include deviation of the eyes downward and inward, unequal pupil with absence of light reaction, skew deviation with the eye opposite the hemorrhage displaced downward and medially,

ipsilateral Horner's syndrome, absence of convergence paralysis of vertical gaze and retraction nystagmus. Patients may later develop a chronic contra lateral pain syndrome [32].

Brainstem hemorrhages
Usually crossed hemiplegia occurs, ipsilateral cranial nerve with opposite side hemiplegia. In pontine hemorrhages deep coma with quadriplegia usually occurs over a few minutes. Prominent decerebrate rigidity and pin-point (1 mm) pupils that react to light, impairment of reflex horizontal eye movements evoked by head turning (doll's head or occulocephalic maneuver), hypercapnia, sever hypertension, and hyperhidrosis are common. Death usually occurs within a few hours but there are occasional survivors [32].

Cerebellar hemorrhages
Usually develop over several hours and are characterised by occipital headache, repeated vomiting, and ataxia of gait. Dizziness or vertigo may be prominent there is a paresis of conjugate lateral gaze toward the side or an ipsilateral sixth nerve palsy. Blepharospasm, involuntary closure of one eye, ocular drooping, and skew deviation, dysarthria and dysphagia may occur. No Babinski sign until hemorrhage dissects into the ventral brainstem, patient may become comatose from brainstem compression [32].

Lobar hemorrhages
Clinical features of lobar hemorrhage depend on the lobes involved.
Frontal lobe: Disinhibition, lack of initiation, antisocial behaviour, impaired memory, expressive dysphasia, and incontinence, impaired smell, contra lateral hemi paresis are common.
Dominant parietal lobe: Dysphasia, dyscalculia, apraxia, agnosia. Associated physical signs: Contra lateral hemi sensory loss, astereognosis, contra lateral homonymous lower quadrantanopia, focal seizures [33].
Non-dominant parietal lobe: Neglect on non dominant side spatial disorientation, constructional apraxia, dressing apraxia, associated physical signs: Contra lateral hemi

sensory loss, astereognosis, contra lateral homonymous lower quadrantanopia, focal seizures.

Dominant temporal lobe: Receptive aphasia, dyslexia, impaired verbal memory; associated physical sign is contralateral homonymous upper quadrantanopia, complex hallucinations.

Non dominant temporal lobe: Impaired non-verbal memory, impaired musical skill. Associated physical sign is contra lateral homonymous upper quadrantanopia, complex hallucinations [33]. **Occipital lobe:** Visual inattention; visual loss; visual agnosia; Associated physical sign is homonymous hemianopia, simple visual hallucinations.

Arteriovenous malformations

Risk of bleeding from AVM's is approximately 2.4% per year. First symptoms include headache, seizures, sudden neurological problem, vision problem, weakness and inability to move a limb or a side of the body, lack of sensations in part of the body or abnormal sensations. If the AVM bleed once the risk is greater that it will bleed again in the future. ICH and SAH are most common first symptom of cerebral AVM's. Symptoms also develop because of loss of nerve cells in the brain caused by mechanical and ischemic factors. Cerebral AVM's occur in 3 out of 10,000 people although the lesion is present at the time of birth, symptoms may occur at any time, 2/3 of cases occur before age 40 [17].

Aneurysms

Un ruptured intracranial aneurysms: The only clinical feature of significant, relative to rupture, was aneurysm size. Wiebers et al observed 65 patients with one or more un ruptured aneurysms for at least 5 years after their detection. In the co-operative study of intracranial aneurysms (reported by Locksley) none of the aneurysms <7 mm in diameter had further trouble [17].

Ruptured intracranial aneurysms: When rupture occurs, blood under high pressure is forced into the sub arachnoid space and the resulting clinical events assume one of three patterns. There is an excruciating generalised headache and fall unconscious almost immediately. Rarely, unconsciousness may be lost quickly without any preceding complaint. Decerebrate rigidity may occur at the onset of hemorrhage in association with unconsciousness. If the hemorrhage is massive death may ensue within minutes. Gross lateralising signs in the form of hemiplegia, hemi paresis, homonymous hemianopia, or aphasia are absent in the majority of cases: Large aneurysms can compress the adjacent cranial nerves, there by signs and symptoms of cranial nerve lesion can be present [17].

Diagnosis

Intra cerebral hematomas are of two kinds: Traumatic, Non-traumatic. Traumatic intra cerebral hemorrhage diagnosis may be easy or most difficult, depending on its location and size. ICH following a stab or bullet wound can be identified by X-ray evidence of small skull depression, a broken off knife blade, or bullet fragments, but intra cerebral hemorrhage following a contrecoup cerebral injury is difficult [21].

Non-traumatic intra cerebral hemorrhage: There is no specific laboratory test for detection of intra cerebral hemorrhage, but imaging studies can detect the hemorrhage in the brain parenchyma. CT Scanning reliably detects even very small supratentorial hemorrhages, MRI is more sensitive for delineating associated abnormalities, such as aneurysm vascular malformation and neoplasm and is superior for imaging the posterior fossa and spinal column. If it is subarachnoid hemorrhage, CT and lumbar puncture are helpful.

AVM's: Clinical suspicion can be made as per history and symptoms and signs, in normotensive, non diabetic patients. CT scan brain can disclose AVM's over 90% if contrast study is done, arteriography establishes the diagnosis with certainty, and will demonstrate AVM's larger than 5 mm in diameter. Cranial MRI, magnetic resonance angiography can detect AVM's.

Aneurysms: Cerebral aneurysms usually diagnosed by the tests to determine the cause of bleeding with in the brain. CT scan brain: Can identify the bleeding and locate the aneurysm site. Cerebro spinal fluid: Confirm the bleeding when CT is non-diagnostic. MRI brain: alternate to CT, but not as sensitive to bleeding within the brain. Cerebral Angiography: is the most sensitive tool and pinpoints the location and size of aneurysm. EEG: may be performed, when seizures occur.

Cerebral amyloid angiopathy (CAA) [2]**:**Patient history, family history, clinical suspicion, CT and MRI identify the lobar hemorrhage as hemorrhage into this site is more common in amyloid angiopathy, Angiography not helpful in diagnosis of cerebral amyloid angiopathy but may need to exclude aneurysms.

Criteria for establishing the diagnosis
- Definite diagnosis: Requires confirmation at autopsy of severe CAA in the presence of lobar hemorrhage, with no evidence of another cause.
- Probable diagnosis: based on neuropathological findings in a biopsy specimen or tissue obtained during evacuation of hematoma and multiple lobar hemorrhages in a patient over the age of 60 years.
- Possible diagnosis: is usually documented if there is a solitary lobar intra cerebral hemorrhage and no other clear cause.

Immunohistochemical techniques have been developed that have made it easier to identify amyloid in tissue samples. Gradient echo MRI could provide an excellent surrogate outcome in future clinical trials, reducing the required sample size which detects sub clinical hemorrhage. Other risk factors for hemorrhagic stroke can be identified depending on history, laboratory tests, personal habits and diet [2].

Treatment

Cerebral hemorrhage, once considered untreatable, with 50% case fatality rate. Now the things have changed because of the advent of good drugs nimodipine, and accurate diagnosis with the advent of new imaging technologies CT scan and MRI.

Nifedepine and other Ca^{++} channel blockers have poor brain penetration. Nicardipine, Izrapidine and Nimodipine, have good brain penetration. Nimodipine, is the most extensively studied, has better penetration into the brain and brain vessels. Subarachnoid and cerebral hemorrhage causes sustained vasospasm. This vasospasm cause all the clinical signs and symptoms of ischemic neurological deficits. Nimodipine is a dihydropyridine ca^{2+} antagonist which dilates the vasospastic cerebral arterioles and increase cerebral blood flow. It is used in the prevention and treatment of delayed ischemic neurological deficits that frequently occurs in patients with sub arachnoid haemorrhage and cerebral haemorrhage as a result of sustained cerebral vasospasm..

Uncontrolled intra cellular influx of calcium is the current major culprit. Ischemia induces the release of excitatory amino acid neurotransmiter such as glutamate and glycine which promote calcium entry into neurons via such receptor-mediated membrane channels as the kainate the alpha – amino – 3hydroxy – 5 – methyl – 4 – isoxozole propionic – acid (AMPA), and the n-methyl d – aspartite (NMDA) channels. Later on a variety of enzymatic reactions follow, including those mediated by Calmodulin. Destruction of neurofilaments, disruption of cell membrane integrity and consequent cell death most likely result from the production of nitric oxide and subsequent formation of other free radicals. Ischemic penumbra impairs the function of the tissue (but potentially viable) around the hematoma.

The general medical management of the patients with large intra cerebral hemorrhages includes maintaining adequate ventilation, controlled hyper ventilation to a Pco_2 of 25 to 30 mm Hg, and tissue dehydration by the use of mannitol or furosemide (osmolality kept at 305 to 315 m.

osmol/lit and Na at 150 meq/lit) and limiting fluid intake to 1200 ml/day. There is no specific treatment for lobar hemorrhages, as they are mainly caused by cerebral amyloid angiopathy.

Surgical treatment of aneurysms include i.Procedures are either extra cranial (ligation of the CCA in the neck) or ii.Intracranial (Clipping or ligating the neck of the aneurysm, wrapping or tamponade of the aneurismal sac by muscle, fascia, plastic coating, or arterial graft, trapping the aneurysm, ligation of the main feeding vessel proximal to the aneurysm. iii.Endovascular treatment of intracranial aneurysm has recourse to Guglielmi Detachable micro Coils (GDC) till date more than 16,000 patients who have an intracranial ruptured or un ruptured aneurysm have been treated [28].

Ruptured aneurysm management must be as early as possible, aneurysm of the vertebro basilar system must be subjected to endovascular treatment in the first instance. For aneurysms of the anterior communicating artery, embolisation could be preferred to surgery if the anatomical characteristics are favourable by virtue of the less invasive nature of endovascular techniques. Giant aneurysms benefit from surgery when they are accessible [28].

Treatment of arteriovenous malformations include prevent complications by limiting bleeding, controlling seizures, if possible removing the AVM's by surgery.
Open brain surgery: Removal of AVM through opening made in skull.
Embolisation: Injecting glue like substance into abnormal vessels to stop aberrant blood flow into the AVM, is alternative if surgery is not feasible and stereo tactic radio surgery is another alternative.
Role of surgery: Evacuation of supratentorial hemorrhages does not improve the outcome. The STICH study shows, that in patients with spontaneous supratentorial intracerebral haemorrhage in neurosurgical units show no evidence of overall benefit with a policy of early surgery compared with initial conservative

medical treatment [55]. Clinical studies of surgery and imaging have so far failed to provide conclusive evidence, as reported in seven randomised trials [56-62], three metanalysis [63-65].

We expect an "**endoscopic surgery – stent with flow diversion**", a revolutionary technical progress will open new possibilities for treatment of intra cerebral hemorrhage, and in future, yield results different from that of international "STICH" study.

Endovascular embolisation is the treatment of choice for ruptured and unruptured intracranial aneurysms. Technology is moving fast,better coils, better stents, more experienced operators. Flow diversion is poised to be a revolutionary technological advancement in the field.

Prognosis
Prognosis of the intra parenchymal hemorrhage is mainly depends on the underlying cause, and the site of the hemorrhage within the brain parenchyma. Hypertensive hemorrhages within basal ganglia, thalamic and pontine hemorrhages when evacuation done through an open craniotomy is obscured by the neuronal damage. Recent attempts at early craniotomy did not alter the outcome and were usually associated with increased risk of recurrent bleeding. Regarding the cerebellar hematomas morbidity and mortality are related to compression of the brainstem and are decreased by timely decompression. The lobar intra cerebral hemorrhages incidence is increased markedly with age. Patients with CAA and intra cerebral hemorrhage have a lower mortality and a greater risk of recurrence than patients with other types of ICH. Recurrence is uniformly associated with greater disability and an increased mortality rate. In a case of aneurismal hemorrhage the immediate mortality rate of the first hemorrhage is approximately 35 percent and an additional 15 percent die from a secondary rupture within the next few weeks or months. Death may occur instantly with in an hour when a large aneurysm ruptures. Most of the AVM's are clinically silent

for a long time, but sooner or later they bleed. The first hemorrhage may be fatal, but in more than 90 percent of cases the bleeding stops and the patient survives. The rate of rebleeding is about 6 percent in the first year if no treatment is given. There is no recurrence of hemorrhage in up to 5 years in those patients whose AVM has disappeared [11].

Future directions

New treatments need to be developed that prevent the deterioration of neurologic function after an intra cerebral hemorrhage. Studies of genetic factors that may indicate the risk of intra cerebral hemorrhage should be persuaded. For example study of the association between genes for certain apolipoproteins (E_2 and E_4) and intra cerebral hemorrhage may provide valuable insight into the pathogenesis of degenerative changes that lead to vessel rupture and possibly a means of preventing these changes. The development of therapies that reduce cerebral edema and neuronal damage may require a more complete understanding of the injury and of the sequence and mediators of pathophysiologic events that produce secondary injuries [2].

Aim

To study the incidence, identify etiological factors, assess mortality, assess prognostic factors, clinical comparison, of intra cerebral hemorrhage in normotensive cases and hypertensive cases.

Materials and methods

All cases of cerebro vascular accident (CVA) presented to Gandhi Hospital, Musheerabad, were considered, and the cases of CVA with intra cerebral hemorrhage evidenced by lumbar puncture and CT scan brain were prospectively studied at Department of Medicine, Gandhi Hospital Muheerabad, between May 2002 and April 2004.

Exclusion criteria
- CVA with intra cranial hemorrhage of surgical emergencies were excluded.

- Intra cerebral hemorrhage with hypertension and other associated risk factors for intra cerebral hemorrhage were excluded, but they were studied for comparison with intra cerebral hemorrhage of normotensive cases.

Inclusion criteria
- Only intra cerebral hemorrhage cases were included.
- Intra cerebral hemorrhage without risk factors for ICH were included.
- Intra cerebral hemorrhage with all age group, both males and females were included.
- Only normotensives were included.

After fulfillment of both exclusion and inclusion criteria, total number of 63 cases were considered for study. Out of 63 cases, 22 cases are with normotensive, non diabetic and non-risk factors; and 41 cases are with only hypertension; two groups of cases were studied separately and the comparison was made. Risk factors for intra cerebral hemorrhage were excluded like hypertension, smoking, alcohol consumption, cocaine use, bleeding diathesis, head injury, etc. by history, clinical examination, and laboratory investigations. Neurological assessment was done by Glasgow Coma Scale Score (GCSS) and hematoma volume was measured on CT scan, in this method, the estimated volume of the hematoma is half the product of **A, B, and C**, where **A.** is the greatest diameter of the hemorrhage on the CT scan, **B.** is the diameter perpendicular to A, and **C.** is the number of slices showing hematoma multiplied by the slice.

Results

Total number of 254 Cerebro Vascular Accident (CVA) cases presented to Gandhi Hospital, Musheerabad. Cerebral Thrombosis were 171, Cerebral Embolism were 20, and Intra Cerebral Hemorrhage were 63. Out of 63 Intra cerebral hemorrhage cases, 41 were hypertensive and 22 were normotensive. Out of 23 deaths in 17 cases hematoma volume was more than 60 ml (**Table –**

1). Out of 26 deaths, in 18 patients Glasgow coma scale score was < 9 (**Table – 2**).

Table – 1: Hematoma Volume and Number of deaths.

Hematoma Volume (ml)	Number of deaths
21 – 30	0
31 – 40	4
41 – 50	2
51 – 60	3
61 – 70	11
71 – 80	4
81 and over	2

Table – 2: GCSS and Number of deaths.

Glasgow Common Scale Score	Number of deaths
3	1
4	1
5	0
6	6
7	0
8	10
9	6
10	2

Out of 63 ICH cases 41 were hypertensive and 22 were normotensive, out of 41 hypertensive cases (**Table - 3**), death occurred in 23 cases and 18 patients survived; out of 22 normotensive cases, deaths reported for 3 cases and 19 cases survived (**Table - 6**). Out of 63 cases studied 42 were males; and 21 were females; male: Female (2:1), normotensive 22, and hypertensive 41. Out of 22 normotensive cases 16 were males and 6 were females (**Table - 3**), 18 were conscious, and 4 were unconscious, out of 18 conscious patients, 1 death has occurred and 17 cases recovered with conservative management, out of 4 unconscious patients, deaths occurred in 2 cases and 2 cases recovered by conservative management. In hypertensive cases 13 were conscious, and 28 were unconscious; out of 13 conscious patients 2 deaths occurred and 11 patients recovered with anti hypertensive and Nimodipine management; out of 28 unconscious patients 21 deaths occurred and 7 cases recovered with anti

hypertensive and Nimodipine treatment (**Table - 6**). In all 63 cases headache, vomiting and neurological deficit were commonly identified, other signs also identified depending on the site of the lesions. Out of 22 normotensive cases, 12 cases had lobar as a common site (**Figure - 1 to 8**) and 10 had other than lobar in their location. Extension of intracerebral hemorrhage was as per **Table – 4**. Site of lesion was as per **Table – 5**. Out of 22 normotensive cases cause was identified in 5 patients, 4 cases due to rupture aneurysm and 1 case due to rupture arteriovenous malformation (AVM) and cause was not identified in 17 cases (**Table - 7**). Mortality was as per **Table – 8**. Out of 4 aneurismal cases 1 death occurred, and 4 cases are under follow up and 1 case with AVM also under follow up. Out of 41 hypertensive cases 13 were conscious and 28 were unconscious, out of 13 conscious patients death occurred in 2 cases, 11 cases recovered. Out of 28 unconscious cases death occurred in 21 cases and 7 cases recovered (**Table - 6**). In all 63 cases majority of cases were in the 5th, 6th and 7th decades (**Table - 3**). All Cases were given broad spectrum antibiotic (inj. Ampicillin 500 mg IV 6th hourly, Inj. Flagyl 500 mg IV 8th hourly and Inj. Gentamycin 80mg IV 12th hourly), Inj. Mannitol 100 mg IV 8th hourly, tablet Nimodipin 30 mg, 2 tablets, 8th hourly, proper care and physiotherapy.

Table – 3: Sex and age incidence.

	Hypertensive	Normotensive
Total Number of cases 63	41 (65.07%)	22 (34.92%)
Sex incidence		
Males	26 (63.41%)	16 (72.72%)
Females	15 (36.58%)	06 (27.27%)
Age incidence		
20-29	01 (2.43%)	00
30-39	01 (2.43%)	03 (13.63%)
40-49	08 (19.51%)	01 (4.54%)
50-59	08 (19.51%)	05 (22.72%)
60-69	13 (31.70%)	09 (40.90%)
70-79	09 (21.95%)	04 (18.18%)
80 and over	01 (2.43%)	00

Table – 4: Intra cerebral hemorrhage – extension.

ICH extension	Hypertensive	Normotensive
Ventricular extension	11 (26.82%)	05 (22.72%)
Subarachnoid extension	01 (2.43%)	01 (4.54%)
No Ventricular extension	29 (70.73%)	16 (72.73%)

Table – 5: Site of lesion.

Site of Hemorrhage	Hypertensive	Normotensive
Frontal	Nil	03 (13.63%)
Frontoparietal	01 (2.43%)	04 (18.18%)
Temporal	Nil	01 (4.54%)
Temporo-parietal	05 (12.19%)	03 (13.63%)
Capsulo-ganglionic	14 (34.14%)	05 (22.72%)
Thalamic	06 (14.63%)	02 (9.09%)
Thalamo-ganglionic	06 (14.63%)	02 (9.09%)
Bil. Capsulo-ganglionic	02 (4.87%)	01 (4.54%)
Brain Stem	05 (12.19%)	nil
Cerebellum	02 (4.87%)	nil

Discussion

Cerebral hemorrhage is a less frequent cause of stroke, than cerebral thrombosis and it is more often thought fatal. It is usually attributed to small vessel disease and the most common sites of hemorrhage are the basal ganglia, internal capsule, cerebellum and pons [11]. Cerebral hemorrhage is mainly of two types A.Primary intra cerebral hemorrhage: where hypertension is common cause and amyloid angiopathy common in normotensives. B.Secondary intra cerebral hemorrhage: where vascular malformations and other risk factors for intra cerebral hemorrhage are common [11, 48].

Although hypertensive intra cerebral hemorrhage remains the most common form of intra cerebral hemorrhage, underlying vascular abnormalities should always be considered in appropriate circumstances, because of high risk of recurrent hemorrhage and available treatment options [2, 5, 6].

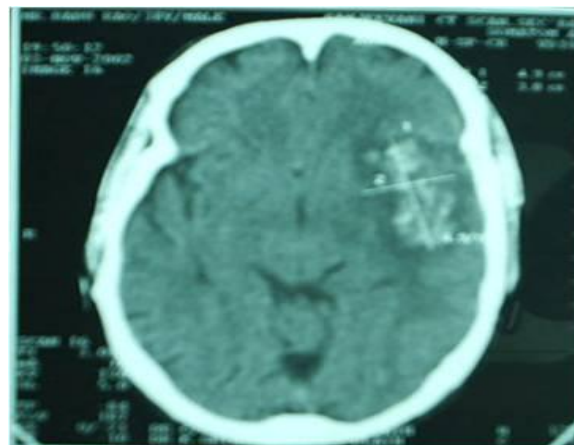**Figure – 1:** Left Tempero Parietal Hemorrhage.
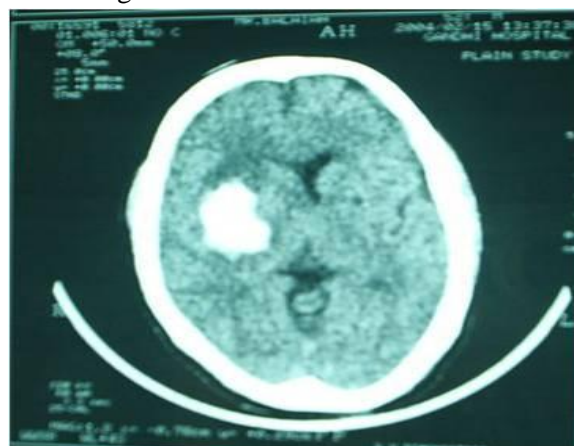

Figure – 2: Right capsuloganglionic hemorrhage.
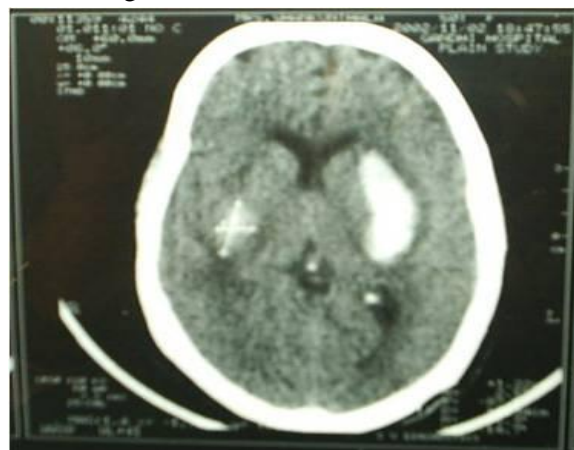

Figure – 3: Bi-lateral Capsuloganglionic Hemorrhage.
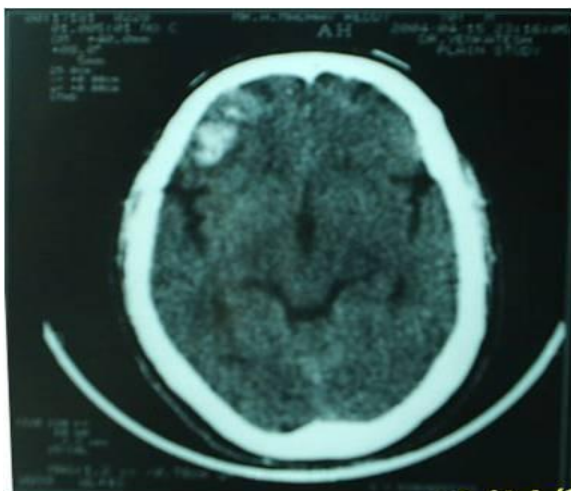
Figure – 4: Right frontal lobe hemorrhage.

Figure – 5: Left thalamoganglionic hemorrhage with ventricular extension.

Figure – 6: Left capsuloganglionic hemorrhage with ventricular extension.

Figure – 7: carotid angiogram showing giant internal carotid artery aneurysm.
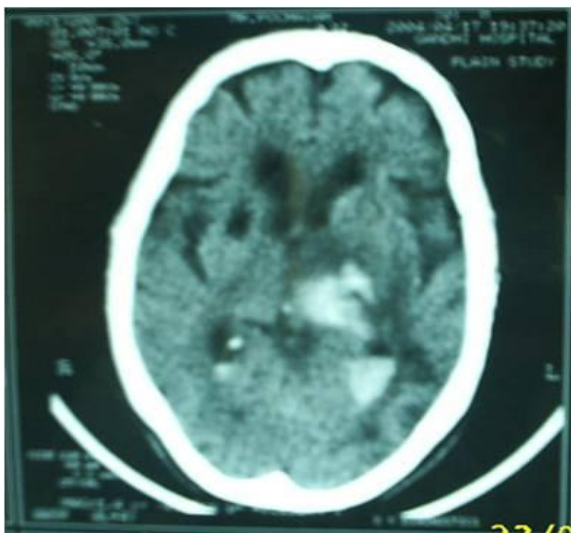
Figure – 8: Left temporal lobe hemorrhage.

In our study, 34.92% of cases were normotensives. A study by Dorothy Russell's (1954) [23] out of 461 cases half the cases (229) were not associated with hypertension. Since 1954, other causes and associations of intra cerebral hemorrhage have become apparent. Our study showed male predominance and was similar to other studies shown in **Table - 2**. We also noticed 5th and 6th decade was the commonest age group distribution and was compared with other authors shown in **Table – 9 to Table - 11**.

Although basal ganglia is the common site to be affected in hypertensive hemorrhages, some patients with intra cerebral hemorrhage, however the hemorrhage is lobar in location, such as in the frontal, parietal, temporal or occipital cortex

and such patients often do not have hypertension [12]. In our study we found lobar site was the commonest site to be effected.

Table – 6: Level of consciousness vs deaths.

Level of consciousness	Hypertensive		Normotensive	
	No. cases	No. of Deaths	No. cases	No. of Deaths
Conscious	13	2	18	1
Unconscious	28	21	4	2

In our study we studied 22 cases of normotensive intra cerebral hemorrhages, the cause is identified in 5 cases and in 17 cases no cause for the hemorrhage could be demonstrated. Out of 5 cases, 4 cases were due to aneurismal rupture, and one case was due to rupture of arteriovenous malformation. In a series of 22 patients reported by Kase, et al. [17], 55 percent were normotensive. Metastatic tumours, AVM's and blood dyscrasias were noted in 14, 9 and 5 percent of the patients respectively.

Table – 7: Cause of intra cerebral hemorrhage in normotensives.

Cause	No. of patients	Percentage
Aneurysm	4	18.18%
AVM's	1	4.54%
Unknown	17	77.27%

Table – 8: Mortality.

Mortality	Hypertensive	Non-Hypertensive
Total	41	22
No. of Deaths	23	3
No. of Survival	18	19

If the hemorrhage is large, midline structures are displaced to the opposite side and vital centers are compromised, leading to coma and death. Rupture or seepage into the ventricular system usually occurs and the CSF becomes bloody in more than 90 percent of cases. In our study we did lumbar puncture for all 63 cases, CSF suggestive of intra cerebral hemorrhage was identified in 84% of the cases and similar findings are noted in normotensive cases.

The presence of blood in the ventricles is associated with a high mortality rate. This effect may be related to development of obstructive hydrocephalus or a direct mass effect of the ventricular blood or periventricular structures, which is associated with global hypoperfusion of overlying cortex [2, 9].

In our study out of 63 cases, ventricular extension was found in 18 patients and mortality was high. 12 patients died with intra cerebral hemorrhage with ventricular extension (65%). The presence of blood in the ventricular system is associated with high mortality (**Table - 4**).

Abnormalities indicating higher level cortical dysfunction, including aphasia, neglect, gaze deviation, and hemianopia, may occur as a result of the disruption of connecting fibers in the subcortical white matter and functional suppression of overlying cortex as diasclisis [2].

It will be noted that in the localisation of intra cerebral hemorrhage, ocular signs are important. In putaminal hemorrhage deviation to side opposite the paralysis, thalamic lesion downward deviation, in pontine lesion pupils fixed, reactive, and in cerebellar lesion deviation is laterally to the side opposite the lesion. In our study also same presentation was observed.

Although the rapid onset of abnormalities and a decreased level of consciousness suggest the diagnosis of intra cerebral hemorrhage,

distinguishing definitively between cerebral infarction and intra cerebral hemorrhage requires imaging of the brain. On the initial CT Scan, the location and size of the hematoma, the presence of ventricular blood, and the occurrence of hydrocephalus should be noted. Selected patients should undergo angiography to look for the secondary causes of intra cerebral hemorrhage such as aneurysms, arterio venous malformations and vasculitis [2, 52].

Table – 9: Other studies - age distribution.

Authors	Years								
	10-19	20-29	30-39	40-49	50-59	60-69	70-79	80-89	Total
Stehbens(1963) [44]	2	6	27	74	119	107	38	7	380
Freytag (1968) [37]			44	88	117	79	48	17	393
Present study*		1	4	9	13	22	13	1	63

Table – 10: Other studies - sex distribution (%).

Authors	Number of males	Number of females	Total
Aring and Merritt (1935) [46]	53%	47%	116
Mutlu, et al. (1963) [49]	43%	58	135
Stehbens (1963) [44]	77	116	380
Freytag (1968) [37]	214	149	393
Present study*	42	21	63

We advised angiography for 22 normotensive cases, 4 of them showed aneurysms, one with arterio venous malformation, and cause was not found in 17 cases. Zhu, et al. [53] reported abnormalities on angiography in 49 percent of patients with lobar hemorrhage and 65% of patients with isolated intraventricular hemorrhage. These authors also reported that 48% of the patients who were normotensives and 45 years of age or younger had abnormalities on angiography, where as hypertensive patients older than 45 years of age have no underlying vascular abnormalities.

On the basis of this evidence, patients with lobar or primary intra ventricular hemorrhage should undergo angiography regardless of age or the presence or absence of hypertension. Aneurismal rupture into the brain tissue without leakage into the sub-arachnoid space is unlikely, so that the diagnosis of ruptured aneurysm should never be made unless blood is present in the cerebro spinal fluid. We did lumbar puncture for all of our cases and majority we observed cerebro spinal fluid finding's suggestive of intracerebral hemorrhage.

Among the age and sex distribution it was equally reported in hypertensive hemorrhage and normotensive hemorrhage, but regarding site of the lesion basal ganglia, internal capsule were common sites in hypertensive hemorrhage and lobar site common in normotensive hemorrhages (**Table – 3** and **Table - 5**).

Factors predictive of high mortality rate are 1.A low score on the Glasgow coma scale 2.A large volume of the hematoma and 3.Presence of ventricular blood on the initial CT scan brain [2, 54]. Broderick, et al. [54] found in their study that the Glasgow coma scale of less than 9 and hematoma volume of more than 60 ml had a mortality rate of 90%. In our study similar results were observed (**Table – 1** and **Table - 2**).

Inspite of proper care and conservative management mortality is high in unconscious patients. It was also found that high mortality

among hypertensive hemorrhages, but mortality is less in normotensive cases, where lobar is the common site with affected patients are usually over the age of 60 years and in majority of them

cause for intra cerebral hemorrhage was not identified, in these cases cause can be a cerebral amyloid angiopathy or mucoid vasculopathy or others.

Table – 11: Other studies - distribution of intra cerebral hemorrhage.

Authors	Basalganglia + thalamus	Elsewhere in cerebrum	Brain stem	Cerebellum	Total
Hudson & Hayland(1958) [50]	23		7	1	31
Mutlu, Berry & Alpers (1963) [49]	94	33	8	-	135
Stehbens (1972) [36]	274	27	53	26	380
Dinsdale(1964) [51]	429		30	52	511
Freytag (1968) [37]	225	45	63	47	380
Present Study (2004)*	56		5	2	63

Conclusion

63 cases with intra cerebral hemorrhage of 254 cerebrovascular accidents were studied. An attempt has been made to identify the etiological factors in non hypertensive cases, though the hypertension is the common cause for primary intra cerebral hemorrhage. Clinical presentation and mortality was assessed in normotensive cases, hypertensive cases and the comparison was made. For all cases it is useful to score on the Glasgow coma scale, as Glasgow coma scale score of 14 or higher (good neurological status) have a good recovery or only mild disability. It is also useful to assess hematoma volume. As volume less than 40 ml (good neurological status) have a good recovery or only mild disability with conservative management. All the cases were confirmed as intra cerebral hemorrhage, evidenced by CT scan brain. Carotid angiography was done to identify the secondary causes of intra cerebral hemorrhage in normotensives. There is no significant difference regarding age and sex distribution in hypertensives as well as normotensives intra cerebral hemorrhage cases. The results of this study suggest that, it is useful to identify the risk factors for intra cerebral hemorrhage in normotensives, because the cause was identified

in 5 cases, 4 cases with aneurysm, one case with arterio venous malformations (AVM's) and most of the cases who belong to 5th and 6th decade, the cause for intra cerebral hemorrhage was not known. Cerebral amyloid angiopathy apolipoprotein genotype E_2 and E_4 should be studied further, as there is a correlation of risk for intra cerebral hemorrhage, which is not done in this study due to non availability of the test at this centre. Genetic studies in relatives of normotensive hemorrhage cases may provide some clues in future and may provide basis for prevention of intra cerebral hemorrhage. The arterial biopsy in intra cerebral hemorrhage should be done routinely to know the mucoid vasculopathy or amyloid angiopathy. Study of the association between genes for certain apolipoproteins and intra cerebral hemorrhage may provide valuable insight into the pathogenesis of degenerative changes that lead to vessel rupture, and possibly a mass of preventing these changes.

Nimodipine is the most extensively studied, has better penetration into the brain and brain vessels. Subarachnoid and cerebral hemorrhage causes sustained vasospasm. This vasospasm cause all the clinical signs and symptoms of

ischemic neurological deficits. Nimodipine is a dihydropyridine ca^{2+} antagonist which dilates the vasospastic cerebral arterioles and increase cerebral blood flow. Nimodipin was used in all the cases as initial medical treatment,with good results,when compared to 50% mortality in other studies where Nimodipin was not given.

The STICH study shows, that in patients with spontaneous supratentorial intracerebral haemorrhage in neurosurgical units show no evidence of overall benefit with a policy of early surgery compared with initial conservative medical treatment [55]. No initial surgery was contemplated in all the cases in this study.

We expect an "endoscopic surgery - stent with flow diversion", revolutionary technological advancement in the field will open new possibilities for treatment of intra cerebral hemorrhage, and in future, yield good results different from that of international "STICH" study.

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