INTRODUCTION

Headache is defined as pain in the head; caused by dilation of cerebral arteries, muscle spasm, insufficient oxygen in the cerebral blood or due to any other reason. All headaches are considered primary headaches or secondary headaches. Primary headaches are not associated with other diseases. Examples of primary headaches are migraine headaches, tension headaches, and cluster headaches. Secondary headaches may result from innumerable conditions, ranging from life threatening ones such as brain tumors, strokes, meningitis, and subarachnoid hemorrhages (SAH).

During the study period, approximately 40% of patients with sub arachnoid hemorrhage die or rendered permanently disabled by the initial hemorrhage, the remainder admitted in the hospital in good condition, with the potential for a complete recovery. The decline in morbidity and mortality can be attributed to the early referral of patients to specialized centers, combined with aggressive management to minimize the risks of re-bleeding and vasospasm. Calcium antagonists, pharmacological agents for the removal of clotted blood and endovascular techniques have contributed to this decline. Post traumatic sub arachnoid hemorrhage may have infective, epileptic, post concussive syndromes and neurobehavioral disorders. Headache is one of the most common complaints in patients with traumatic brain injury. By definition, headache that develops within 1 wk after head & neck trauma (or within 1 wk after regaining consciousness) is referred to as posttraumatic headache (PTH). Acute post traumatic headache can have a tension type headache, migraine like headache & cluster headache.

Nimodipine is a dihydropyridine calcium channel blocker originally developed for the treatment of high blood pressure. It has shown good results in preventing major complication of subarachnoid hemorrhage associated vasospasm. Mortality rates did not differ between Nimodipine and placebo treated patients. Post-traumatic cerebral vasospasm is being increasingly recognized as a possible significant complication after head injury. The presence of blood in subarachnoid space in patients with severe head injury is associated with a risk of vasospasm, with poorer outcome. Patients treated with Nimodipine had a significantly less unfavorable outcome (death, vegetative survival, or
severe disability) at 6 months than placebo-treated patients.  

OBJECTIVE  
To study the effect of Nimodipine on headache associated with post traumatic Sub Arachnoid Hemorrhage

MATERIALS AND METHODS  
6 months prospective study conducted at the Department of Neurosurgery & General Surgery, District Headquarters Hospitals Rawalpindi

INCLUSION CRITERIA  
Patients from both genders who consented for the study were included in the study. All the patients who had evidence of Sub Arachnoid Hemorrhage on CT scan, GCS of 13-15 on initial presentation, Grade 1 and 2 on Hunt and Hess Grading for Sub Arachnoid hemorrhage, grade 1 or grade 2 score on Fischer Grading for Sub Arachnoid Hemorrhage on CT scan were included in the study. Poly Trauma patients, previous history of cerebral aneurysmal bleeds & history of Diabetes Mellitus and Hypertension were excluded from the study.

RESULTS  
The study was carried out at Rawalpindi Medical College after taking written informed consent from the patient or guardian and permission from hospital ethical committee. The data on each of the patient was collected using pre-designed proforma (Annex. I). All patients were given a code number and divided into 2 groups. All odd numbers patients were given Tab Nimodipine 30 mg 2 tablets 6 hourly. The patients were classified according to Hunt and Hess grading and Fischer grading for SAH. Only the patients falling under grade 1 and 2 under both categories were considered for the study. Patients having presentation of GCS 13-15 were enrolled (mild head injury). 60 Patients from both genders, undergoing hospital admissions that fulfill the inclusion criteria were included in the study. 12 patients were lost due to poor patient follow up. Of the remaining 48 patients, 29 (60.4%) were male and 19 (39.6%) were females. 16 patients were from low socioeconomic background, 28 were middle class, and 04 were from high socioeconomic class. 16 patients (33.3%) were students, 14 (29.2%) patients were businessmen, 2 were laborer (4.2%), 11 (22.9%) were housewives, 5 (10.4%) were unemployed. 29 (60.4%) patients suffered road side accidents, 12 (25%) had assaults and 7 (14.6%) had fall. The patients were classified according to Hunt and Hess and Fischer Grading. 34 patients had grade 1 and 14 had grade 2 injuries according to Hunt and Hess classification. 31 had grade 1 injuries and 17 had grade 2 injuries according to Fischer Grading. The mean age was 34.02 years with a range of 4 to 71. The mean interval since trauma & admission were 1.4 hour. Headache was scored on the basis of visual analog scale for pain. Score 0 will be considered no pain while score 10 will be considered severe most headache. Score in between showed the progressive increase in severity of headache in ascending order. Both group scores were noted and compared.

The pain score on the visual analog pain scale before treatment had a mean of 4.46. The patient score on visual analog scale after treatment was 2.33 and P-value was calculated to be <0.001.

DISCUSSION  
There were not many studies available for comparison but most of leading neurosurgery & trauma centers around the world agreed upon the role of nimodipine in post traumatic headache. According to Ducros & Bousser, approximately 60% of cases are secondary, mainly postpartum and after exposure to vasoactive substances. In our study 60.4% suffered from road traffic accident. The major complications are localized cortical

![Visual analogue scale used to assess the severity of pain.](image-url)
subarachnoid hemorrhage (22%) and parenchyma ischemic or hemorrhagic strokes (7%) which may leave permanent squeal. Nimodipine seems to reduce thunderclap headaches within 48 h.

Another study shows the cause of SAH is a ruptured aneurysm in 85% of cases, non-aneurysmal perimesencephalic haemorrhage (with excellent prognosis) in 10%, and a variety of rare conditions in 5%. Measures of proven value in decreasing the risk of delayed cerebral ischemia are a liberal supply of fluids, avoidance of antihypertensive drugs and administration of nimodipine.

Our study shows 50% decrease in headache when treated with nimodipine. Similarly other centers showed that early treatment with nimodipine reduces the severity of neurological deficits resulting from vasospasm in subarachnoid hemorrhage (SAH) patients. In SAH, nimodipine reduced spasm-related deficits of all severities & nimodipine is a safe drug with an important place in pharmacotherapy and effective in reduction of severity of neurological deficits resulting from vasospasm in SAH patients. Another study done by Przewlocki & his colleagues showed beneficial effect of Nimodipine in continuous infusion given to patients in SAH after carotid artery stenting. There are other calcium channel blockers like Nicardipine and Nisoldipine and who had beneficial effects in headache due to SAH.

The coronary vasodilating properties of nisoldipine have led to the investigation of this agent for use in angina. Selectivity for the cerebrovascular bed makes nimodipine potentially useful in the treatment of subarachnoid hemorrhage, migraine headache, dementia, and stroke. All these studies showed that Calcium is very important in many physiological mechanisms and could be involved in neuronal damage following cerebral ischemia. Thus, calcium-channel blockers agents could be of interest in medical treatment.

### Table-I. Test of Significance used: Man Whitney U test

<table>
<thead>
<tr>
<th>Group of drug</th>
<th>N</th>
<th>Mean Rank</th>
<th>Sum of Ranks</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nimodipine</td>
<td>24</td>
<td>16.08</td>
<td>386.00</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Control/placebo</td>
<td>24</td>
<td>32.92</td>
<td>790.00</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>48</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

### Table-II. Showing Hunt and Hess grading scale for Sub Arachnoid Hemorrhage

<table>
<thead>
<tr>
<th>Grade</th>
<th>Clinical Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Asymptomatic or minimal headache and slight nuchal rigidity</td>
</tr>
<tr>
<td>2</td>
<td>Moderate to severe headache, nuchal rigidity, and no neurological deficit other than cranial nerve palsy</td>
</tr>
<tr>
<td>3</td>
<td>Drowsiness and confusion or mild focal deficit</td>
</tr>
<tr>
<td>4</td>
<td>Stupor, moderate to severe hemiparesis, and possibly early decerebrate rigidity and vegetative disturbances</td>
</tr>
<tr>
<td>5</td>
<td>Deep coma, decerebrate rigidity, and moribund appearance</td>
</tr>
</tbody>
</table>

### Table-III. Showing Fischer Grading for Sub Arachnoid Hemorrhage

<table>
<thead>
<tr>
<th>Grade</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>No blood</td>
</tr>
<tr>
<td>2</td>
<td>Diffuse deposition or thin layer, no clots greater than 3mm, thick or vertical layer greater than 1mm thick</td>
</tr>
<tr>
<td>3</td>
<td>Dense collection of blood greater than 1mm thick in the vertical plane (inter hemispheric fissure, insular cistern or ambient cistern), or 5 x 3mm in longitudinal or transverse dimension in the horizontal plane (stem of Sylvian fissure, Sylvian cistern, or interpeduncular cistern)</td>
</tr>
<tr>
<td>4</td>
<td>Intracerebral or intraventricular clots but with only diffuse or no blood in basal cistern</td>
</tr>
</tbody>
</table>

of cerebrovascular diseases, headache and subarachnoid hemorrhage.

CONCLUSIONS
Our results signify that Nimodipine should be prescribed to patients with Grade 1 and 2 post traumatic Sub Arachanoid Hemorrhage. Headache was considerably reduced. The results are statistically significant.

REFERENCES
PREVIOUS RELATED STUDIES
