Original Article

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# Qualitative Assessment of Cerebral Perfusion in Post Stroke Seizure Patients using Arterial Spin Labeling: A Case-control Study

Radiology Section

ANJALI B SUSAN[1], UTTAM B GEORGE[2], JEYARAJ D PANDIAN[3]



## ABSTRACT

**Introduction:** Arterial Spin Labeling-Perfusion Weighted Imaging (ASL-PWI) assesses cerebral blood flow using magnetically labeled inflowing arterial blood water protons as freely diffusible tracer without exogenous contrast agent. Seizures are one of the sequelae of stroke with its mechanism not well studied.

**Aim:** Qualitative assessment of cerebral perfusion in post stroke seizure patients using ASL-PWI and comparison of perfusion patterns in post stroke seizure patients and stroke patients.

**Materials and Methods:** This was a case-control study among 100 stroke patients who underwent Magnetic Resonance Imaging (MRI) of brain in Department of Radiodiagnosis, from Outpatient and Inpatient Department of Neurology, Christian Medical College, Ludhiana, Punjab, India, from 1st January 2018 till 1st January 2020. Total 50 post stroke seizure patients were included as cases and 50 age-matched post stroke patients without seizure were included as controls. Perfusion pattern was compared qualitatively using ASL-PWI. Qualitative variables were associated using Chi-square test/Fisher's-Exact test.

**Results:** The mean age of the study subjects were 56.38±16.98 years (age range from 19-87 years) and that of controls were 59.66±11.86 years (age range from 19-89 years) (p-value=0.265). Cerebral hypoperfusion was noted in 39 (78%) of total 50 post stroke seizure patients irrespective of type of stroke. Cortical and subcortical area of frontal and parietal lobes was predominantly involved in post stroke seizure patients. Out of 39, 23 (71.88%) early onset seizure and 16 (88.89%) late onset seizure patients had hypoperfusion. There was no significant association of perfusion abnormality with onset of seizure (p-value=0.241). Hypoperfusion was noted in cases and controls without any statistical difference.

**Conclusion:** Cortical and subcortical area of involvement was noted in post stroke seizure patients. Post stroke seizure patients showed hypoperfusion, irrespective of stroke type and onset of seizure. There was no difference seen in perfusion abnormality between post stroke seizure patients and stroke patients without seizure.

**Keywords:** Hypoperfusion, Magnetic resonance imaging, Neuroimaging

## INTRODUCTION

Stroke is one of the common causes of epilepsy in elderly population. Post stroke seizure can be early onset seizure which occurs within two weeks of stroke or can be late onset, where the seizure occurs after two weeks following the stroke. In seizure there is an increase in metabolic demand of the affected brain parenchyma. As a result there is a temporary increase in cerebral perfusion in the involved region [1]. So, analysing the perfusion has become widely accepted in evaluation of seizures. Various nuclear medicine perfusion techniques like Positron Emission Tomography (PET), Single Photon Emission Computed Tomography (SPECT) have showed increased cerebral perfusion during peri-ictal period and decreased perfusion during interictal period following seizure [2]. However, clinical utility of these techniques is limited as they are expensive and require multiple patient evaluation and use of radioactive substances. Perfusion computed tomography or dynamic susceptibility contrast Magnetic Resonance Imaging (MRI) scan can also assess cerebral perfusion. However, it require contrast material for the study.

Arterial Spin Labeling (ASL) is a non invasive Magnetic Resonance Imaging (MRI) technique, which measures cerebral blood flow using magnetically labelled blood water protons as endogenous tracer. It does not require any contrast material for assessment of perfusion making it suitable for perfusion studies in patients with renal insufficiency, paediatric population or those that need repetitive follow up scans. Combination of Radiofrequency (RF) pulses and gradients invert the longitudinal magnetisation of blood water protons in feeding arteries. After certain transit time, the inverted protons reach the brain parenchyma and generate a labelled image. The transit time depends on the individual's age and circulatory conditions. A second acquisition is made without prior inversion of water protons and this will generate a control image. Subtraction of labelled and controlled images will produce an absolute measurement of cerebral blood flow [3]. ASL can be used to assess perfusion in dementia, especially to determine hypoperfusion in patients with Alzheimer's disease [4]. Perfusion mismatch can also be determined through ASL MR perfusion imaging in cerebrovascular disease. A study has shown the added benefit of inclusion of ASL MR perfusion imaging to conventional MR imaging in stroke patients to predict outcome [5]. Another study has also shown the potential use of ASL in identification of arteriovenous malformation and Moyamoya disease [4]. The Arterial Spin Labeling -Perfusion Weighted Imaging (ASL-PWI) has been used to determine the perfusion abnormalities in seizure patients and differentiate them from post stroke seizures and seizure mimickers [2]. However, more studies on this topic are imperative to establish the role of ASL-PWI. The present study aimed for qualitative assessment of cerebral perfusion patterns in post stroke seizure patients using arterial spin labelling and comparison of cerebral perfusion patterns in post stroke seizure patients and stroke patients without seizure.

## MATERIALS AND METHODS

This was a case-control observational study conducted among 100 stroke patients in Christian Medical college, Ludhiana, Punjab, India, from 1st January 2018 till 1st January 2020. The study included Outpatients and Inpatients from Department of Neurology who underwent imaging in Department of Radiology. Patients with stroke undergoing MRI study of the brain and who were willing to participate in the present study, were enrolled after obtaining

an informed voluntary consent. Institutional Ethics approval was obtained for the study (Ref:2017-Dec/CMCL-IEC-450). Depending on the inclusion and exclusion criteria, patients were categorised into cases and controls. Total 50 cases and 50 age matched controls were included for the study.

**Sample size calculation:** On the basis of pilot study, 100% of controls had hyperperfusion/hypoperfusion abnormality and 80% of cases had hyperperfusion/hypoperfusion abnormality. Taking these values as reference, the minimum required sample size with 90% power of study and 5% level of significance was 42 patients in each study group. To reduce margin of error, total sample size taken was 100 (50 patients per group).

Formula used

$n=\{(pc\times(1-pc)+pe\times(1-pe)\}\times(Z_\alpha+Z_\beta)^2)/(pc-pe)^2$

Where, pc=hyperperfusion/hypoperfusion in controls

pe=hyperperfusion/hypoperfusion in cases.

$Z_\alpha$ was value of Z at two sided alpha error of 5%

$Z_\beta$ was value of Z at power of 90%.

**Inclusion criteria:**

For control:

- Stroke patients with seizure.
- Age >18 years.
- Computed tomography/magnetic resonance imaging diagnosis of ischaemic/haemorrhagic stroke/cerebral venous thrombosis.

For cases:

- Stroke patients without seizure.
- Age >18 years.
- Computed tomography/magnetic resonance imaging diagnosis of ischaemic/haemorrhagic stroke/cerebral venous thrombosis.

**Exclusion criteria:** Patients with known epilepsy prior to stroke event and patients with general contraindications for MRI such as pacemaker, aneurysmal clips, metal implants and claustrophobia were excluded from the study.

**Following definitions were used in the study:**

- Seizure: Transient occurrence of signs or symptoms due to excessive or synchronous neuronal activity in the brain [6].
- Epilepsy:

1. At least two unprovoked (or reflex) seizures occurring >24 hour apart.
2. One unprovoked (or reflex) and a probability of further seizures similar to the general recurrence risk (atleast 60 %) after two unprovoked seizures, occurring over the next 10 years.
3. Diagnosis of epilepsy syndrome [7].

- Early post stroke seizure: These are seizures, which occur within the second week following stroke [8].
- Late post stroke seizure: These are seizures that begin after a latent period of variable duration following stroke, usually more than two weeks to years [8].

### Magnetic Resonance Imaging (MRI) Protocols

The MRI was performed on 3 Tesla MRI Scanner (Magnetom Spectra Siemens) using 16-channel head coil. The imaging protocol included axial Fluid Attenuated Inversion Recovery (FLAIR) Diffusion Weighted Imaging (DWI) and Arterial Spin Labeling-Perfusion weighted Imaging (ASL-PWI) with image acquisition protocol as given in [Table/Fig-1]. The ASL-PWI was performed using pseudocontinous labeling with labeling duration as 1800 ms and post labeling delay as 2000 ms. The 3D T1 weighted images were used for registration and segmentation purpose.

| Parameters | 2D FLAIR | DWI | ASL-PWI (pseudocontinous) |
|---|---|---|---|
| Repetition time (msec) | 9000 | 5040 | 4000 |
| Echo time (msec) | 77 | 70 | 19.82 |
| Flip angle (degree) | 150 | 180 | 180 |
| Slice thickness (mm) | 5 | 4 | 3 |
| Slice gap% | 30 | 30 | 50 |
| Field of view (mm) | 220×220 | 235×235 | 192×192 |
| Matrix | 320×182 | 140×140 | 64×63 |
| No. of sections | 22 | 30 | 1 |

**[Table/Fig-1]:** MR imaging protocol.

### Qualitative Image Analysis

The ASL-PWI images were generated through automated colour maps. The images were visually analysed by two independent radiologists of three years and 18 years of experience for the presence or absence of the following parameters.

i. Diffusion restriction on diffusion weighted imaging (DWI)
ii. Hyperintensity on Fluid attenuated inversion (FLAIR)
iii. Perfusion abnormality on ASL-PWI

Perfusion abnormalities when detected will be further analysed in terms of the following:

i. Pattern (hyperperfusion or hypoperfusion compared to gray matter of normal contralateral parenchyma at the same slice).
ii. Multifocality (focal, multifocal or hemispheric).
iii. Atypical distribution against vascular territories (territorial, if perfusion abnormality corresponded to one or more vascular territories or non territorial, if not).

## STATISTICAL ANALYSIS

Data were entered into Microsoft Excel Sheet. Cases and controls were matched according to age. ASL perfusion parameters were analysed in two groups of the study. Categorical variables were presented in number and percentage (%) and continuous variables were presented as mean±SD and median. Normality of data was tested by Kolmogorov-Smirnov test. Non parametric test was used when the normality was rejected. Quantitative variables were compared using Independent t-test (as the data sets were normally distributed) between the two groups. Association between qualitative variables was evaluated using Chi-square test/Fisher's-Exact test. The determined p-value was considered statistically significant for values <0.05 for all the statistics. Data analysis was performed in Statistical Package for Social Sciences (SPSS) version 21.0.

## RESULTS

**Demographic profile:** The mean age of the study subjects were 56.38±16.98 years (age range from 19-87 years) and that of controls were 59.66±11.86 years (age range from 19-89 years) with p value of 0.265. The study included 41 (82%) males as cases and 35 (70%) males as controls. The mean duration between stroke and seizure was 18.36±10.98 hours and 28 (56%) post stroke patients had seizures within 24 hours of stroke. Majority of patients had ischaemic stroke in post stroke seizure patient group as well as in control group, followed by haemorrhagic stroke and few had cerebral venous thrombosis (p-value=0.042) [Table/Fig-2].

**Imaging findings:** Cortical and subcortical area of involvement had statistically significant association with post stroke seizure (p-value=0.050) [Table/Fig-2]. Statistically significant association with T2 FLAIR signal changes and onset of seizure was present (p-value=0.041) [Table/Fig-3]. There was also statistically significant T2 FLAIR signal changes in frontal and parietal lobe. There was no significant restricted diffusion involving temporal lobe in post stroke seizure patients (p-value >0.05) [Table/Fig-4]. Hypoperfusion was noted in 39 (78%) post stroke seizure patients. Arterial Spin Labeling (ASL)

| Parameters | | Cases (n, %) | Control (n, %) | p-value |
|---|---|---|---|---|
| Type of stroke | Ischaemic stroke | 26 (52%) | 38 (76%) | 0.042* |
| | Haemorrhagic stroke | 19 (38%) | 10 (20%) | |
| | Cerebral venous thrombosis | 5 (10%) | 2 (4%) | |
| Area of involvement | Cortical | 9 (18%) | 16 (32%) | 0.050* |
| | Subcortical | 3 (6%) | 17 (34%) | |
| | Cortical and subcortical | 28 (56%) | 4 (8%) | |
| | Deep gray matter | 10 (20%) | 13 (26%) | |
| Multifocality | Focal | 23 (45.65%) | 32 (64.44%) | 0.177† |
| | Multifocal | 18 (36.96%) | 14 (26.67%) | |
| | Hemispheric | 9 (17.39%) | 4 (8.89%) | |
| Atypical distribution against vascular territories | Territorial | 10 (21.74%) | 11 (22.22%) | 0.956† |
| | Non territorial | 40 (78.26%) | 39 (77.78%) | |

**[Table/Fig-2]:** Comparison of post stroke seizure patients (cases n=50) and controls (n=50).
*Fisher Exact test, †Chi-square test; p-value <0.05 was considered as statistically significant

| Parameters | | Early onset seizure (n=32) (n, %) | Late onset seizure (n=18) (n, %) | p-value |
|---|---|---|---|---|
| Type of seizure | Generalised seizure | 8 (25%) | 6 (33.3%) | 0.529† |
| | Focal seizure | 24 (75%) | 12 (66.6%) | |
| Type of stroke | Haemorrhagic | 12 (37.5%) | 7 (38.8%) | 0.195* |
| | Ischaemic | 15 (46.8%) | 11 (61.1%) | |
| | Cortical venous thrombosis | 5 (15.6%) | 0 | |
| DWI restricted diffusion | | 13 (40.63%) | 4 (22.2%) | 0.227* |
| T2 FLAIR hyperintensity | | 16 (50%) | 5 (27.78%) | 0.041† |
| Arterial spin labeling-perfusion abnormality | Hyperperfusion | 5 (15.63%) | 2 (11.11%) | 0.241* |
| | Hypoperfusion | 23 (71.88%) | 16 (88.89%) | |
| | No change | 4 (12.5%) | 0 | |

**[Table/Fig-3]:** MR imaging findings in post stroke seizure patients.
*Fisher Exact test, †Chi-square test; p-value <0.05 was considered as statistically significant

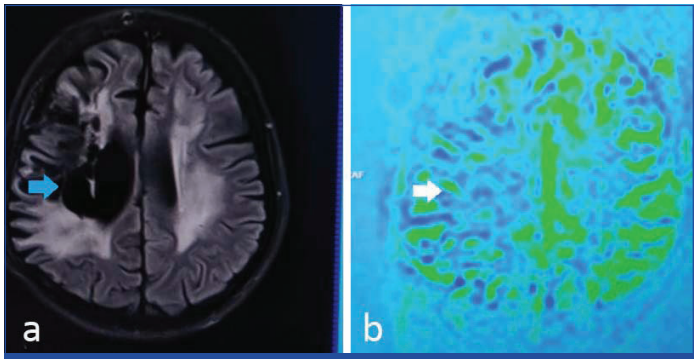
| Parameters | Cases (n, %) | Control (n, %) | p-value (Fischer's-Exact test) |
|---|---|---|---|
| **Diffusion restriction** | | | |
| Frontal lobe | 6 (12%) | 1 (2%) | 0.112 |
| Parietal lobe | 6 (12%) | 1 (2%) | 0.026 |
| Temporal lobe | 5 (10%) | 1 (2%) | 0.050 |
| Occipital lobe | 2 (4%) | 0 | 0.495 |
| Deep gray matter | 2 (4%) | 0 | 0.056 |
| **T2 FLAIR hyperintensity** | | | |
| Frontal lobe | 8 (16%) | 2 (4%) | 0.027 |
| Parietal lobe | 8 (16%) | 2 (4%) | 0.019 |
| Temporal lobe | 7 (14%) | 4 (8%) | 0.329 |
| Occipital lobe | 3 (6%) | 1 (2%) | 0.526 |
| Deep gray matter | 5 (10%) | 3 (6%) | 0.194 |
| **ASL perfusion abnormality** | | | |
| **Frontal lobe** | | | |
| Hypoperfusion | 24 (48%) | 14 (28%) | 0.080 |
| Hyperperfusion | 1 (2%) | 0 | |
| **Parietal lobe** | | | |
| Hypoperfusion | 20 (40%) | 14 (28%) | 0.235 |
| Hyperperfusion | 1 (2%) | 0 | |
| **Temporal lobe** | | | |
| Hypoperfusion | 20 (40%) | 13 (26%) | 0.109 |
| Hyperperfusion | 2 (4%) | 0 | |
| **Occipital lobe** | | | |
| Hypoperfusion | 6 (12%) | 8 (16%) | 0.252 |
| Hyperperfusion | 3 (6%) | 0 | |
| **Deep gray matter** | | | |
| Hypoperfusion | 9 (18%) | 13 (26%) | 0.358 |
| Hyperperfusion | 0 | 1 (2%) | |
| No change | 4 (8%) | 0 | |

**[Table/Fig-4]:** Comparison of imaging findings in post stroke seizure patients (cases, N=50) and controls (N=50).
p-value <0.05 was considered as statistically significant

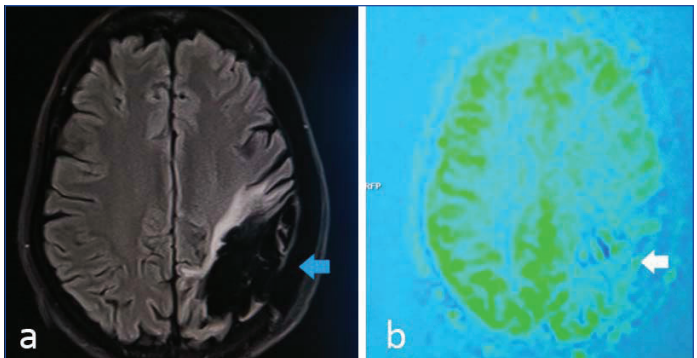
hyperperfusion was seen in 7 patients (14%); out of which most them had ischaemic type of stroke (p-value <0.001) [Table/Fig-5]. There was no statistically significant association of perfusion abnormality with onset of seizure was obtained. There was no statistically significant territorial distribution of perfusion abnormality in post stroke patients. The imaging details are summarised in [Table/Fig-4]. Representative MR images including ASL-PWI are shown in [Table/Fig-6-9].

| Parameters | Ischaemic stroke (n=26) (n, %) | Haemorrhagic stroke (n=19) (n, %) | Cortical venous thrombosis (n=5) (n, %) | p-value |
|---|---|---|---|---|
| T2 FLAIR hyperintensity | 13 (50%) | 7 (36.84%) | 1 (20%) | <0.001† |
| Diffusion restriction | 9 (34.62%) | 7 (36.84%) | 1 (20%) | 0.775† |
| Hypoperfusion | 21 (80.77%) | 18 (94.74%) | 0 | <0.0001* |
| Hyperperfusion | 5 (19.23%) | 1 (5.26%) | 1 (20%) | |
| No change in perfusion | 0 | 0 | 4 (80%) | |

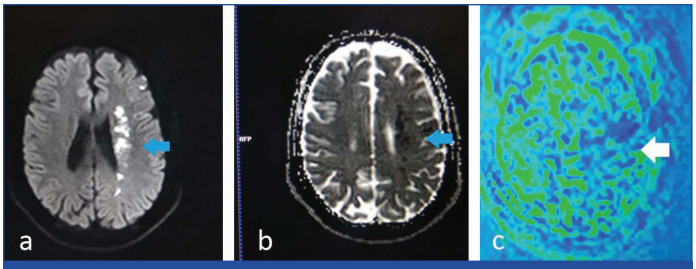
**[Table/Fig-5]:** Comparison of MRI findings in post stroke seizure patients with type of stroke.
*Fisher Exact test, †Chi-square test; p-value <0.05 was considered as statistically significant



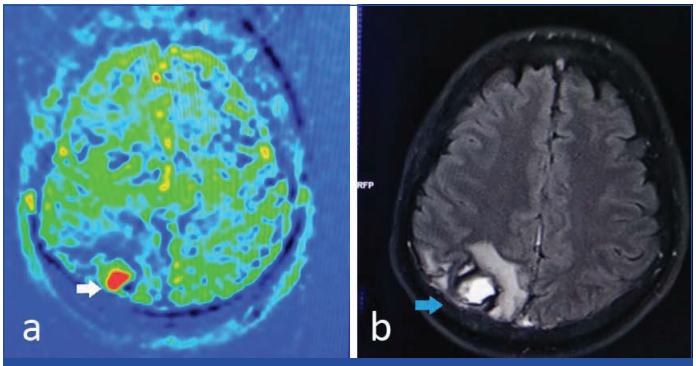

**[Table/Fig-6]:** A 58-year-old female with past history of ischaemic stroke involving right middle cerebral artery territory presented with late onset seizure; a) T2 FLAIR (axial) image demonstrating encephalomalacia in right frontoparietal parenchyma (blue arrow); b) ASL-PWI colour maps showing hypoperfusion in the corresponding area (white arrow).




**[Table/Fig-7]:** A 35-year-old female with past history of stroke and late onset seizure; a) Axial T2 FLAIR image showing hypointensity suggestive of encephalomalacia with surrounding gliosis in left frontoparietal lobe (blue arrow); b) ASL-PWI colour map shows hypoperfusion in left frontal and parietal lobe (white arrow).




**[Table/Fig-8]:** A 60-year-old male with acute ischaemic stroke and early onset seizure (<24 hours); a,b) Show restricted diffusion with corresponding ADC drop in left corona radiata suggestive of acute infarct (blue arrow); c) ASL-PWI colour map demonstrates hypoperfusion in left frontal and parietal lobe (white arrow).




**[Table/Fig-9]:** A 62-year-old male with haemorrhagic stroke presented with early onset seizure (<24 hours); a) shows ASL-PWI colour maps indicating focal area of hyperperfusion in right parietal lobe (white arrow); b) shows corresponding T2 FLAIR image with T2 hyperintensity and peripheral hemosiderin rim and perilesional edema suggestive of intracranial haemorrhage (blue arrow).

## DISCUSSION

The present study showed significant association of cortical and subcortical area of involvement with seizure in post stroke patients. Majority of post stroke seizure patients had ischaemic stroke. Most of patients with early onset seizure showed T2 FLAIR hyperintensity in the affected region while patients with late onset seizure showed T2 FLAIR hypointensity. There was no significant involvement of temporal lobe in post stroke seizure patients. Majority of post stroke seizure patients showed hypoperfusion on ASL-PWI irrespective of type of stroke. There was no significant association of perfusion abnormality with onset of seizure seen in the present study. The study also did not show any statistical difference in perfusion abnormality between stroke and post stroke seizure patients.

In the present study, it was noted that 52% of post stroke seizure patients had ischaemic stroke, 38% had haemorrhagic and lastly 10% had cortical venous thrombosis. In a study conducted among 66 post stroke patients by Proccacianti G et al., 87% of patients had ischaemic stroke as compared to 12% having haemorrhagic stroke [9]. According to Zhao Y et al., 10-20% of stroke patients developed seizure after haemorrhagic stroke when compared with 2-14% patients developing seizure after ischaemic stroke [10]. In study by Beghi E et al., that enrolled 714 post stroke patients, reported Odds ratio of 7.2 for haemorrhagic stroke to develop post stroke seizure [11]. There are many variations in the type of stroke among post stroke seizure patients. The present study showed post stroke seizure to be seen following predominantly in ischaemic stroke rather than in haemorrhagic stroke which was similar to study by Proccacianti G et al., [9]. It could be because of greater extent of cortical involvement in ischaemic stroke as compared to haemorrhagic stroke as the former has vascular territorial distribution. Moreover, haemorrhagic stroke may involve deep gray matter and subcortical area as in cases of hypertensive bleed with lesser area of involvement. So, type of stroke cannot be taken as sole risk factor for post stroke seizure, other factors, like area of involvement and even duration of follow-up, should also be considered.

A cohort study by Conrad J et al., in that included 593 patients reported that location of stroke, whether in cortical or subcortical region did not influence the risk of seizure [12]. But in study that enrolled 714 post stroke patients by Beghi E et al., reported that 41% of post stroke seizure patients had cortical involvement [11]. This study showed that majority of seizure patients had involvement of both cortical and subcortical area which was inconsistent with other studies. These differences could be because of the larger extent of involvement in post stroke seizure rather than the precise area of involvement. In haemorrhagic stroke, haemorrhage volume and blood products could have triggered the onset of seizure rather than its location. There were also few patients with isolated subcortical white matter or deep gray matter involvement with post stroke seizure in the present study. Such cases could be due to release of glutamate from axonal terminals arising from affected thalamocortical neurons [13].

As per study by Cheung CM et al., anterior circulation and partial anterior circulation stroke had significant association with post stroke seizure [14]. Graham NSN et al., reported that temporal lobe followed by frontal lobe was mostly affected in post stroke seizures [15]. However, in the present study most patients did not show significant temporal lobe involvement as observed through absence of restricted diffusion or T2 FLAIR signal changes involving temporal lobe. Most patients in the present study had frontal lobe and parietal lobe involvement as observed through T2 FLAIR signal changes and cerebral hypoperfusion. This could be because of larger area of involvement of cortex when frontal and parietal lobe is involved as few studies had shown larger cortical area being a risk factor for post stroke seizure [11,15].

Most of early onset seizure patients showed T2 FLAIR hyperintense signal intensity and majority of late onset seizure patients showed T2 FLAIR signal hypointensity. Majority of early onset seizure patients had seizure within 24 hours of stroke. Hence, the T2 FLAIR hyperintensity could be due to vasogenic oedema or good collateral formation in acute phase. As in case of haemorrhagic stroke, T2 FLAIR signal intensities alter with time. Acute onset of stroke within 48 hours showed T2 FLAIR hyperintensity mainly attributed to oxyhaemoglobin and later on degradation occurs and deoxyhaemoglobin and methaemoglobin contents in blood cause variable signal intensities. Patients with T2 FLAIR hypointensity had encephalomalacia which may have caused late onset post stroke seizure. Study by Yoo RE et al., reported that early onset post stroke seizure patients particularly that occurred within 24 hours had hyperintense T2 FLAIR signal changes which was consistent with the present study findings [2].

In the present study, hypoperfusion of affected area was the predominant finding in both cases and control group irrespective of type of stroke. Hypoperfusion may be due to ischaemic penumbra or areas of encephalomalacia in the affected region. Yoo RE et al., reported combination pattern of hypoperfusion and hyperperfusion was observed in early onset post stroke seizure patients [2]. Study also reported territorial distribution of hypoperfusion in post stroke seizure patients. These were inconsistent with the present study findings. As per study by Miyaji Y et al., on late post stroke seizure, focal hyperperfusion was noted in patients during peri-ictal phase of seizure [16]. In this study, there was no statistically significant difference in perfusion pattern neither in between post stroke seizure patients and stroke patients without stroke nor in between early onset seizure and late onset seizure patients. These differences could be due to differences in study design and time difference in MRI acquisition following seizure. White matter has delayed arterial transit time and cerebral blood flow compared to gray matter and hence ASL is less sensitive to white matter lesions [3]. Susceptibility artefacts from air/bone of base of skull hinders the qualitative perfusion assessment in posterior cranial fossa structures.

The present study has some noteworthy strengths. There are limited studies in the literature which have assessed the perfusion parameters in post stroke seizure patients using ASL, as discussed above. Also, since no exogenous contrast agent was required for the study, it enabled inclusion of patients with renal impairment and had added advantage of lack of risk of contrast induced reactions or development of nephrogenic systemic fibrosis to patients.

### Limitation(s)

There were several limitations in the study, having been conducted as a single-centre study in a tertiary care hospital. Uniformity in time interval between stroke and scan between cases and control group could not be obtained due to limited number of patients with the disease process. Data regarding follow up of stroke patients were unavailable.

## CONCLUSION(S)

Cortical and subcortical area of involvement was noted in post stroke seizure patients. Post stroke seizure patients showed hypoperfusion irrespective of stroke type and onset of seizure. There was no difference seen in perfusion abnormality between post stroke seizure patients and stroke patients without seizure.

Although the risk factors of development of post stroke seizures are being researched, not much emphasis on the perfusion abnormality and its potential of being risk factor is being given. The ASL perfusion studies are also upcoming imaging modalities, whose clinical applications are still being researched. The present study may help in further research studies with better study design including quantitative analysis with cerebral blood flow parameters in near future.

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**PARTICULARS OF CONTRIBUTORS:**
1. Assistant Professor, Department of Radiology, Christian Medical College, Ludhiana, Punjab, India.
2. Professor, Department of Radiology, Christian Medical College, Ludhiana, Punjab, India.
3. Professor, Department of Neurology, Christian Medical College, Ludhiana, Punjab, India.

**NAME, ADDRESS, E-MAIL ID OF THE CORRESPONDING AUTHOR:**
Dr. Anjali B Susan,
House No. 3, Gateway Terrace, Christian Medical College and Hospital,
Ludhiana, Punjab, India.
E-mail: anjalisusan26@gmail.com

**PLAGIARISM CHECKING METHODS:** [Jain H et al.]
- Plagiarism X-checker: Feb 01, 2022
- Manual Googling: Apr 02, 2022
- iThenticate Software: Apr 04, 2022 (11%)

**ETYMOLOGY:** Author Origin

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- Financial or Other Competing Interests: None
- Was Ethics Committee Approval obtained for this study? Yes
- Was informed consent obtained from the subjects involved in the study? Yes
- For any images presented appropriate consent has been obtained from the subjects. Yes

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