

# Transient splenial lesion of corpus callosum: A window to many diagnoses



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## ABSTRACT

Transient splenial lesions (TSL) are not of frequent occurrence and are usually observed with other diseases. The mechanism of TSL development still unclear despite of various theories put forward. These are secondary lesions and their diagnosis is of importance to associate them with clinical conditions. Magnetic resonance imaging is the modality of diagnosing TSL and 3T MRI was used in this study. The study includes 10 cases of TSL with varied disease etiologies like migraine, trauma, infection, demyelination etc. All the cases had follow-up imaging which showed resolution of the lesions after varied time intervals. An attempt to correlate the various theories with each type of disease is done in this study.

**Key words:** Corpus callosum; Craniocerebral trauma; Cytotoxic edema; Magnetic resonance imaging; Meningoencephalitis

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## INTRODUCTION

Transient splenial lesions (TSL) are uncommon radiological findings and are associated with various diseases.<sup>1</sup> As they are secondary lesions, it is important to identify them and associate them with the clinical/ radiological diagnosis.<sup>2</sup> Transient signal alteration in the splenium of corpus callosum (SCC) on magnetic resonance imaging (MRI) has been reported in a variety of neurological and non-neurological conditions.<sup>3,4</sup> These lesions can be of different shapes, but they are usually homogenous, non-hemorrhagic and non-enhancing lesions and are observed in the central region of the SCC. The lesions display hyperintense signal on T2 / fluid attenuated inversion recovery (FLAIR) images and are slightly hypointense on T1 weighted images. They show restricted diffusion and apparent diffusion coefficient (ADC) reversal which is classical of the same.<sup>1</sup>

TSL resolve completely after a certain period of time without any residual effects even though the associated conditions may show progression or regression. Few studies hypothesize that the possible pathophysiology of TSL is the reversible intramyelinic edema/ myelin vacuolization or inflow of macromolecules and inflammatory cells.<sup>5-7</sup>

The patterns of splenial lesions can be of four types: small round or oval (type 1), lesion in splenium but extending through callosal fibres into the adjacent white matter (type 2), boomerang sign (type 3) and lesion in the posterior aspect but extending in to the anterior aspect of corpus callosum (type 4).<sup>2,8,9</sup>

Imaging findings of cases with TSL are presented in this study. The MRI was done using 3 Tesla (GE Healthcare, 3

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Tesla Signa Pioneer). Associated conditions of all the ten TSL cases are discussed below.

## CASE SERIES

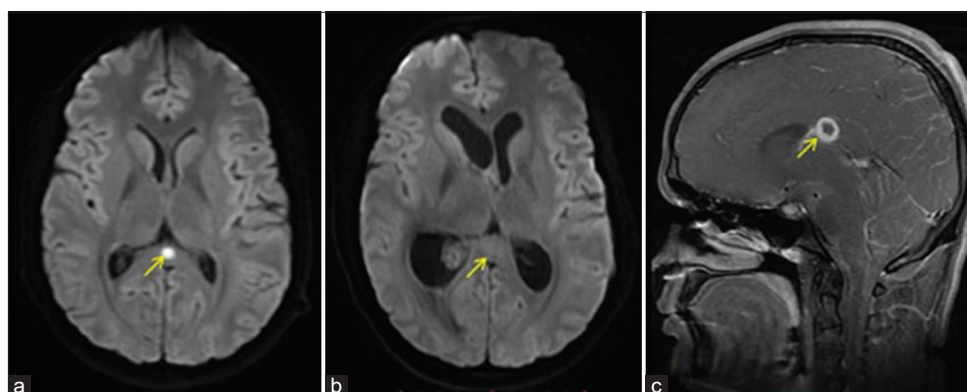
### 1. Infection associated TSL

There is an increase in the level of cytokines in the cerebrospinal fluid (CSF) during infections. The cytokines lead to cytotoxic edema of the glial cells leading to development of TSL.<sup>2,10</sup> We hereby report four cases of TSLs associated with infections.

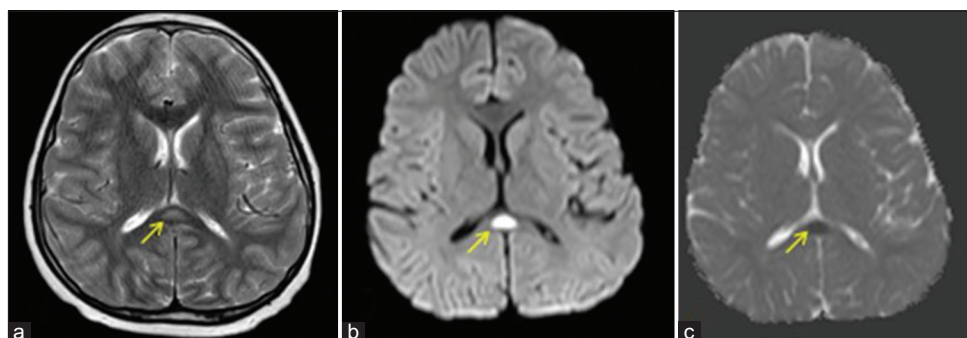
Case of tubercular meningitis with TSL is presented in Figure 1. The 23 year old patient was admitted with complaints of fever and headache. MRI showed multiple tuberculomas, features of meningitis and SCC showed a small restricting focus at its central region. Repeat MRI on 14<sup>th</sup> day of admission showed complete resolution of TSL with progression of the primary disease. A similar case was reported by Wu Jr-Wei et al where a case of CNS TB had a lesion in SCC and a follow-up MRI on day 11 showed disappearance of the lesion. The authors hypothesized the possible mechanism of transient lesion as intramyelinic edema and inflammatory cell infiltration.<sup>11</sup>

We encountered two cases of encephalitis aged nine and 16 years, with classical features of TSL. MRI showed multiple patchy areas of cortical and subcortical edema with an ovoid DWI restricting lesion in SCC which resolved spontaneously on MRI taken on 12<sup>th</sup> day and 14<sup>th</sup> day respectively for cases 1 and 2. (Figure 2) Similar findings were observed by Takatsu H et al in Japan where a high signal intensity lesion in the centre of the splenium on diffusion imaging disappeared after 14 days.<sup>12</sup> Similar views were put forth by Tada H where 15 patients with encephalitis/encephalopathy with reversible lesions in SCC were reviewed. The possible mechanisms suggested were intramyelinic edema and infiltration by inflammatory cells for the development of TSL in cases with encephalitis.<sup>7</sup>

Another 28 year old patient, a case of bacterial meningitis presented with symptoms of fever and neck stiffness was imaged with MRI. MRI showed an oval lesion on the right side of SCC which had completely resolved by the 10<sup>th</sup> day. (Figure 3) Similar case was reported by Tascilar N et al, where an isolated small lesion in SCC was observed in a case with features of meningitis. This lesion completely resolved during the follow-up visit after 5 weeks.<sup>13</sup> The lesions could also be due to vasogenic edema and also due



**Figure 1:** 3T MRI brain of a 23 year old male patient with TBM. (a) Axial DWI show restriction within the central part of the splenium. (b) Follow-up axial DWI show complete resolution of the TSL with primary disease progression and hydrocephalus. (c) Follow-up sagittal T1 weighted post contrast image show tuberculoma



**Figure 2:** 3T MRI brain of a nine year old male patient with encephalitis. (a) Axial T2 weighted image show hyperintensity within the central part of the splenium with (b) Restricted diffusion on axial DWI and (c) Reversal on ADC

to high density of drug and toxin receptors which could cause edema.<sup>14</sup>

## 2. Hypoglycemia associated TSL

Various mechanisms have been proposed for the association of SCC to hypoglycemia. One of the theories proposes that decrease in the glucose levels leading to severe energy failure in the brain results in reduction of ionic pump activity of the cell membrane. There is a shift of cerebral water into the intracellular space causing restriction of the molecules and thus showing diffusion restriction.<sup>15</sup> Another theory proposes that compact neuronal tissues as those found in the corpus callosum are more susceptible to hypoglycemic conditions.

Further, hypoglycemia might also disturb the cellular fluid mechanisms resulting in cytotoxic edema.<sup>16</sup>

We had a case of a one year old male child, who presented with drowsiness. (Figure 4) Random blood sugar was 43 mg/dl on admission. MRI showed the presence of TSL. Follow-up MRI on the 10<sup>th</sup> day showed complete resolution of the lesion.

Similar findings were reported by Miyakawa Y et al where they observed a TSL in the 17 year old male with poorly controlled type 1 diabetes mellitus which showed partial resolution on 22<sup>nd</sup> day post admission and complete resolution by 46<sup>th</sup> day.<sup>17</sup>

## 3. Epilepsy associated TSL

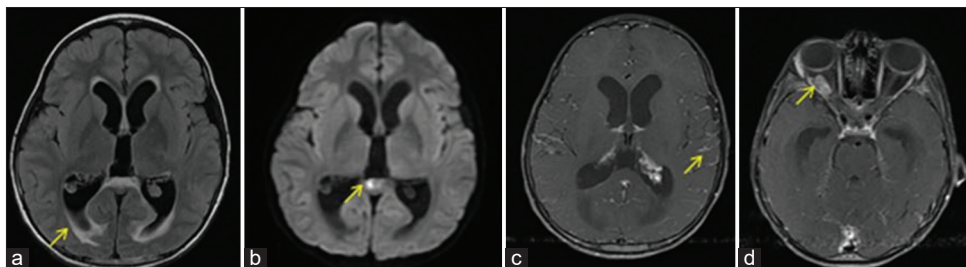
TSL has been shown to be present in a few cases of epilepsy. There are two possible mechanisms; first, the lesion can occur due to transient edema due to the effect on fluid-balance system by arginine-vasopressin (AVP) levels. Second, few anti-epileptic drugs (AED) can influence the fluid-balance system and cause edema. Further, the AED can interact with AVP, causing brain edema.<sup>18</sup> Figure 5 shows the MR image of a known case of seizure disorder aged 30 years on irregular medication, who presented in status epilepticus. MRI showed a TSL. Anti-epileptic drugs were titrated for the patient and repeat MRI on the day of discharge (day 14) showed complete resolution of the lesion.

Similar case was reported in a patient with tonic-clonic seizures on irregular treatment by Parikh NC et al which showed complete resolution after 4 weeks.<sup>19</sup>

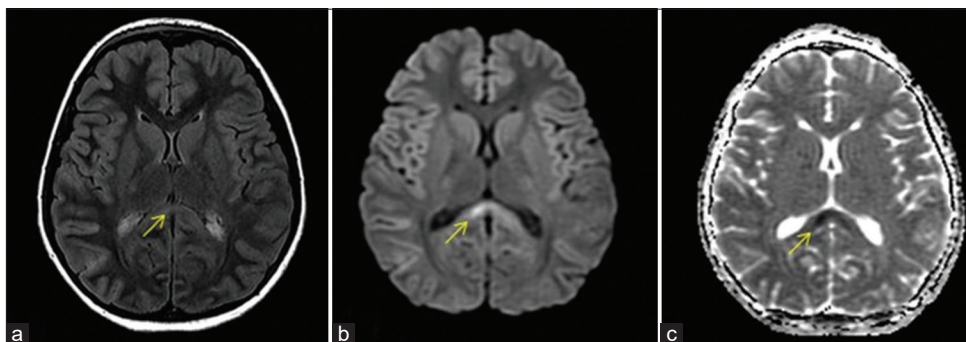
## 4. Trauma associated TSL

Damage to the white matter of the brain caused by shear strain results in diffuse axonal injury. As corpus callosum is entirely composed of white matter, it is susceptible to these kinds of injuries. In cases of head injury, transient, round/ovoid lesions in SCC can be observed and the cause is more likely to be cytotoxic edema rather than vasogenic edema.<sup>20</sup>

We had a patient aged 29 years with history of road traffic accident (RTA) and normal computed tomography



**Figure 3:** 3T MRI brain of a 28 year old male patient with bacterial meningitis. (a) Axial T2 FLAIR image show hydrocephalus with periventricular CSF seepage. (b) Axial DWI show restriction in the right side of splenium. (c) Axial T1 post contrast image show leptomeningeal enhancement. (d) Axial T1 weighted post contrast image at a lower level show an incidental orbital cavernoma



**Figure 4:** 3T MRI brain of a one year old male child with hypoglycemia and hypoxia. (a) Axial T2/FLAIR weighted image show hyperintensity in both sides of the splenium (boomerang sign) with (b) Restricted diffusion on Axial DWI and (c) Reversal on ADC

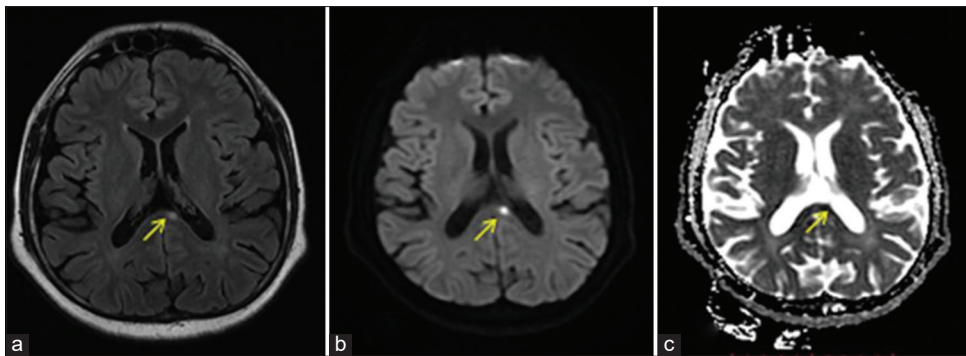
findings. Patient was further evaluated with MRI due to low GCS score. MRI showed multiple small hemorrhagic foci involving the subcortical locations of bilateral frontal and parietal lobes showing diffusion restriction with adjacent subarachnoid hemorrhage and a restricting lesion in the SCC. A repeat MRI on 11<sup>th</sup> day showed resolution of the TSL. (Figure 6).

A similar case was studied by Takayama H et al where a 24 year old patient with diffuse brain injury showed a lesion in body and SCC on 12<sup>th</sup> day post-injury which showed complete resolution in follow-up scan done after 6 months.<sup>20</sup>

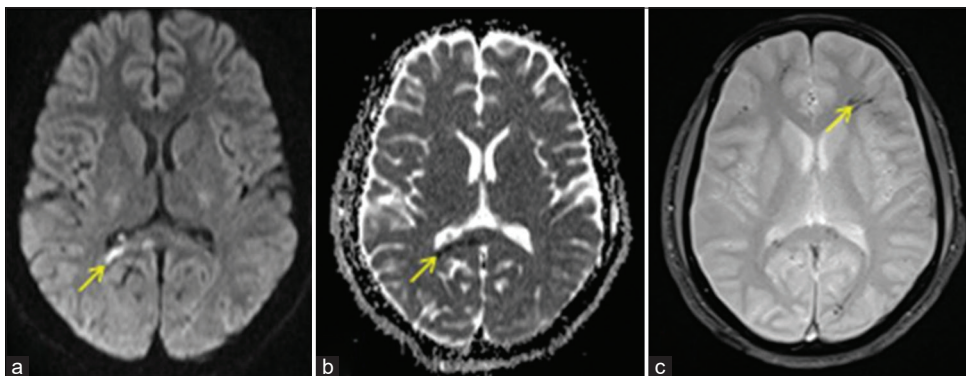
Another case of TSL associated with RTA was reported by Al Brashdi YH et al which resolved after 13 days.<sup>21</sup>

### 5. Demyelination associated TSL

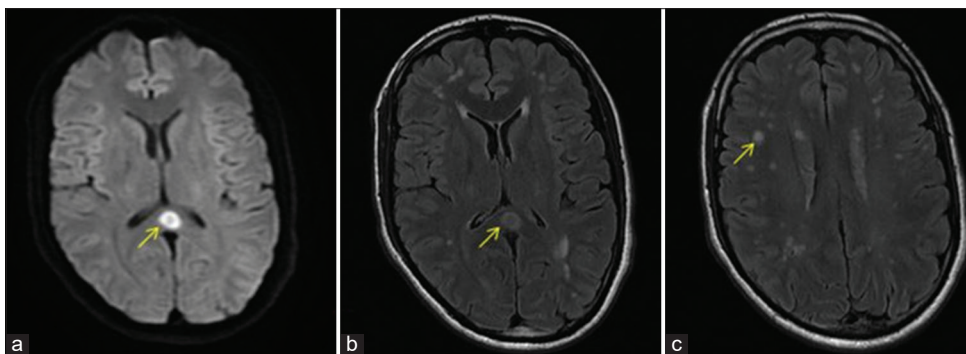
Few cases of demyelination are associated with TSL. We had a case of multiple sclerosis aged 48 years, with relapse showing a lesion in SCC which resolved on 14<sup>th</sup> day MRI. However, primary disease did not show resolution. (Figure 7) A case of TSL associated with ADEM (acute demyelinating encephalopathy) was reported by Saenz-Farret M et al.<sup>22</sup> To our knowledge very few cases of TSL associated with MS haven been documented so far.



**Figure 5:** 3T MRI brain of a 30 year old male patient with intractable seizures. (a) Axial T2/FLAIR weighted image show hyperintensity within the left side of the splenium with (b) Restricted diffusion on axial DWI and (c) Reversal on ADC



**Figure 6:** 3T MRI brain of a 29 year old male with traumatic brain injury. (a) Axial DWI show restriction in central part of the splenium with (b) Reversal on ADC. (c) Axial gradient image shows subarachnoid hemorrhage in the left frontal region



**Figure 7:** 3T MRI brain of a 48 year old male with multiple sclerosis. (a) Axial DWI show restriction in the central part of the splenium. (b) Axial T2/FLAIR image show hyperintensity in the same location. (c) Axial T2/FLAIR image shows multiple demyelinating foci in other regions

## 6. Migraine associated TSL

Few studies have reported the presence of TSL among patients suffering from migraine with aura.<sup>23</sup> The underlying pathology can be excite-toxic edema which is a form of cytotoxic edema. This is a condition that results from an increased amount of extracellular glutamate due to varied mechanisms.<sup>24</sup>

In our case, the patient aged 29 years was a known migraineur who came for MRI imaging during an acute attack. A solitary restricting lesion in the SCC was found. (Figure 8) The patient was treated with analgesics for migraine and follow-up MRI done after 15 days showed complete resolution of the lesion.

A similar case was reported by Unver O et al where a patient aged 13 years presented with severe headache, confusion, slurred speech, violent behavior and right hemiparesis and was having TSL on MRI.<sup>25</sup>

Another case was reported by Ling FU et al where a 33 year old patient having migraine with aura showed TSL on MRI.<sup>26</sup>

## 7. Alcohol associated TSL

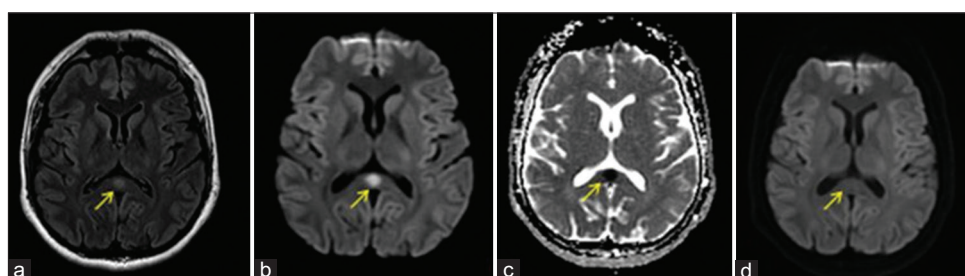
Alcoholism is reported to cause callosal abnormalities in the splenium and genu of CC. This is attributed to deficiency of vitamin B. Severe alcohol consumption causing

chronic dehydration and loss of electrolytes resulting in hyponatremia is also explained as a likely cause.<sup>27</sup>

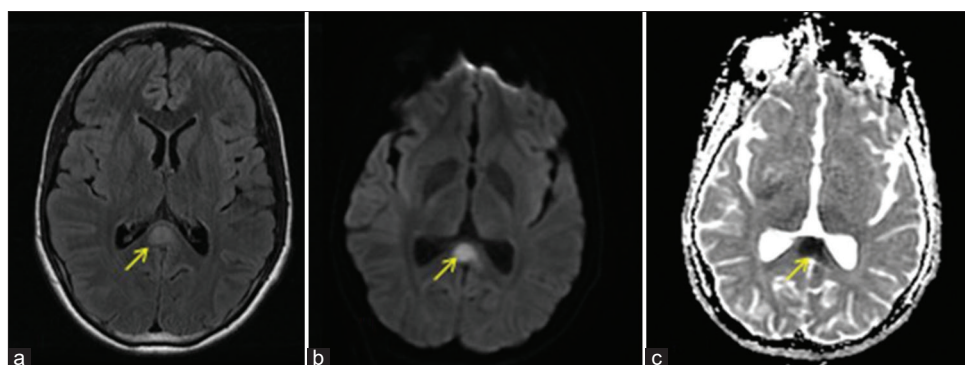
Our case was a 40 year old unresponsive patient after binge drinking who was imaged on MRI and was found to have TSL. (Figure 9) Similar lesion was isolated by Kakkar C et al where a chronic alcoholic patient in unconscious state showed TSL on MRI.<sup>28</sup> Dong X et al reported TSL/CLOOC in CC with varied shapes in patients with Marchiafava–Bignami disease.<sup>29</sup>

## DISCUSSION

Without follow-up imaging, the above mentioned lesions cannot be termed as TSL. Few other terminologies that are in use are mild encephalopathy with reversible splenic lesions (MERS), reversible splenic lesion syndrome (RESLES) reversible splenic lesions or clinically silent lesions in the splenium of the corpus callosum, transient focal lesions in the splenium of the corpus callosum, and cytotoxic lesions of the corpus callosum (CLOCCs).<sup>2,27,30</sup> However, in our study all the 10 cases had follow-up imaging which helped us to come to a conclusion that all of them were just transient lesions, even if the primary condition persisted or disappeared. There are various theories for development of transient lesions put forward.



**Figure 8:** 3T MRI brain of a 29 year old male patient with migraine. (a) Axial T2/FLAIR weighted image shows hyperintensity within the central part of the splenium with (b) Restricted diffusion on axial DWI and (c) Reversal on ADC. (d) Follow-up DWMRI after 15 days shows complete resolution of the TSL



**Figure 9:** 3T MRI brain of a 40 year old male patient with episode of binge drinking. (a) Axial T2/FLAIR weighted image show hyperintensity within the splenium (b) Restricted diffusion on axial DWI and (c) Reversal on ADC

The theory based on anatomy of CC states that, the development of the SCC differs from other parts of corpus callosum and it precedes the hippocampal commissure. Both the anterior and posterior circulations contribute for its perfusion. SCC is made up of axonal fibers with different calibers and also has the most number of callosal glial cells. This may contribute to its affinity for development of TSL.<sup>27,31,32</sup>

The arginine-vasopressin (AVP)/Antidiuretic hormone (ADH) levels also appear to play a role in development of TSL. In CNS infections and other relevant conditions, there can be increased production of ADH, thus causing hyponatremia. Hyponatremia when developed can lead to acute expansion of the extracellular fluid volume, causing diffusion restriction at the SCC acting as one of the major factors for development of TSL.<sup>33</sup>

Events like trauma, infections and inflammations activate the macrophages and lead to release of inflammatory cytokines. This results in leaky endothelial cells with breaking of blood-brain barrier and production of tumor necrosis factor-  $\alpha$  (TNF- $\alpha$ ).<sup>10,34,35</sup> Massive amounts of glutamate is released in to the extracellular space and water is trapped within the cells leading to intracellular edema which is termed as cytotoxic edema.<sup>2</sup>

## CONCLUSION

Transient splenial lesions of the corpus callosum are uncommon secondary conditions that have association with various diseases like infections, demyelination, migraine with aura, epilepsy, diffuse axonal injury, alcohol abuse, metabolic conditions etc. Theories like anatomical uniqueness of SCC, development of cytotoxic edema and role of AVP levels have described the predilection of the SCC for development of TSL. TSLs are usually small, round/ovoid shaped lesions, predominantly located in the central portion of the SCC; however, variants are also well known. All 10 cases reported by us had follow-up and hence we opine that the terminology transient splenial lesions is appropriate for these lesions rather than various other terminologies. Not many case series have reported with follow-up MR imaging showing complete resolution of TSL. However the duration at which the follow-up MRI imaging is advocated is still not clear.

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**FHM and AKM**-Concept and design, preparation, revision of manuscript and reporting radiologists; **ARV**-Preparation and revision of manuscript and reporting radiologist; **VUS, DA and RG**-Editing of manuscript and reporting radiologists.

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