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Review Article

Perspective: Lumbar adhesive arachnoiditis (AA)/ Chronic AA (CAA) are clinical diagnoses that do not require radiographic confirmation

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ABSTRACT

Background: Our hypothesis was that lumbar adhesive arachnoiditis (AA)/chronic lumbar AA (CAA) are clinical diagnoses that do not require radiographic confirmation. Therefore, patients with these syndromes do not necessarily have to demonstrate significant radiographic abnormalities on myelograms, Myelo-CT studies, and/or MR examinations. When present, typical AA/CAA findings may include; central or peripheral nerve root/cauda equina thickening/clumping (i.e. latter empty sac sign), arachnoid cysts, soft tissue masses in the subarachnoid space, and/or failure of nerve roots to migrate ventrally on prone MR/Myelo-CT studies.

Methods: We reviewed 3 articles and 7 clinical series that involved a total of 253 patients with AA/CAA to determine whether there was a significant correlation between these clinical syndromes, and myelographic, Myelo-CT, and/or MR imaging pathology.

Results: We determined that patients with the clinical diagnoses of AA/CAA do not necessarily exhibit associated radiographic abnormalities. However, a subset of patients with AA/CAA may show the classical AA/CAA findings of; central or peripheral nerve root/cauda equina thickening/clumping (empty sac sign), arachnoid cysts, soft tissue masses in the subarachnoid space, and/or failure of nerve roots to migrate ventrally on prone MR/ Myelo-CT studies.

Conclusion: Patients with clinical diagnoses of AA/CAA do not necessary show associated neuroradiagnostic abnormalities on myelograms, Myelo-CT studies, or MR. Rather, the clinical syndromes of AA/CAA may exist alone without the requirement for radiologographic confirmation.

Keywords: Adhesive Arachnoiditis (AA), Chronic Adhesive Arachnoiditis (CAA), Clinical Syndrome, Diagnosis, Lumbar, Magnetic Resonance Imaging (MR), Myelo-CT Scans, Myelography, Mild-Moderate, Severe

INTRODUCTION

Patients with lumbar adhesive arachnoiditis (AA)/chronic AA (CAA) have clinical syndromes characterized by symptoms of pain, paresthesias, and varied motor, sensory, and/or sphincteric deficits. Our hypothesis was that patients with these syndromes do not have to demonstrate any significant radiographic confirmatory pathology on myelograms, Myelo-CT studies, or

MR examinations.[8] To better assess this, we reviewed 3 articles and 7 patient-based studies (i.e. total 253 patients) to determine whether AA/CAA patients did or did not demonstrate central or peripheral nerve root/cauda equina thickening/clumping (i.e. latter empty sac sign), arachnoid cysts, soft tissue masses in the subarachnoid space, and/or failure of nerve roots to migrate ventrally on prone MR, and Myelo-CT studies [Table 1].[1-5,8-10]

History of AA

In 1909, Sir Victor Horsely presented 21 cases where he anticipated finding spinal tumors. However, at surgery he encountered AA variously labeled as; chronic spinal meningitis/arachnoiditis, acute myelitis, adhesive spinal arachnoiditis, and meningitis serosa circumscripta spinalis [Table 1].[7] Later, in 2020, Tsuchida *et al*. similarly

Table 1: Summary of arachnoiditis literature.

Author [Ref] journal year	Study design	Clinical data	Causes of AA	Imaging Findings	Treatment/surgery conclusions
Horsley[7] Br Med J 1909	21 Cases based on operative findings with AA Diagnosed as: Chronic spinal meningitis Often diagnosed as Tumors	". I have seen a relatively large number of such cases on most of which I have performed laminectomy and subdural mercurial irrigation". Clinical symptoms pain Progressive loss of power in the legs Slight Kyphotic curvatures of spine	Develops ultimately progressive paraplegia "runs through the ordinary course and terminates in fatality" They are cases of chronic spinal meningitis "the causation of which has yet to be finally determined"	"They yield to surgical treatment when ordinary medicinal treatment has failed" "I do not mean to say that they all yield to surgical treatment.	I wish they did-but so many do that it is quite clear that earlier diagnosis would have saved in my opinion the majority if not all of the cases".
Benoist *et al*.[2]Spine 1980	Postop Lumbar Epiduro-Arachnoiditis Clinical Presentation LBP Sciatica Radiculopathy	38 Patients all prior surgery for Lumbar AA Diagnosis AA confirmed at repeat surgery massive scarring	Results of surgery: 13 gohod 8 Fair 17 Failure	Myelographic patterns did not disclose any correlation with the clinical symptoms	"Five myelograms were normal, while 6 others simulated a recurrent disc herniation.
Delamarter *et al*.[3] Spine 1990	Diagnosis of Lumbar AA by MR-Compared 24 Cases on MR vs. Myelo-CT+Myelogram	MR Abnormal Configuration Nerve Roots 3 Anatomic Groups	Group 1: Conglomerations of Adherent Nerve Roots Centrally in Thecal sac Group 2 :Nerve Roots Adherent Peripherally to Meninges Giving Empty Sac	Group 3 : Soft tissue mass replacing SA Space	MR accurate diagnosis Excellent Correlation -Myelo-CT+Myelogram Lumbar AA
Rice *et al*.[9] Br J of Anaes 2004	Suggested OB EPI lea to CAA Clinical Criteria Back pain Leg Pain Neuro Deficits	".Because of the varied symptomatology, clinical diagnosis (CAA) is difficult"	Etiology of AA Epi or SAH Contrast Media Lead to CAA	"…radiological and pathological findings do not invariably corelate with the clinical features (of AA)	The precise relationship between the pathological findings and symptomatology has not been defined"

(*Contd*...)

Table 1: (*Continued*).

Author [Ref] journal year	Study design	Clinical data	Causes of AA	Imaging Findings	Treatment/surgery conclusions
Hampl *et al.*[6] Curr Opin Anethesiol 2014	Spinal Anesthesia-Toxicity New and Old Drugs	Chloroprocaine Lower Risk TNS versus Lidocaine Also Smaller Neurotoxic Potential Ropivacaine versus. Levobupivacaine, Procaine, and Bupivacaine	Use of Chlorhexidine Gluconate Skin Prep Prior to Spinal Blocks Still Controversial Due to Potential Association with Neurotoxicity and AA (Case report Severe AA after obstetric Anesthesia)	High Safety Profile Intrathecal Drugs for Spinal Anesthesia	Small Neurotoxic Potential Intrathecal Drugs Study Focused on Increasing Safety of Spinal Anesthesia
Anderson *et al.*[1] AJR 2017	CAA Rare 29 Cases RR 1995-2013 Chronic Spinal Arachnoiditis Adhesive Spinal Arachnoiditis Meningitis Serosa Circumscripta Spinalis Chronic Spinal Meningitis Spinal level 12 TL Spine 9 L-LS Spine 5 Entire spine	29 MR 7 Myelo -CT Ages 23-96 Median 65 years old 11F 18 M Most Often Symptoms 12 Back pain 14 Radicular 11 Sensory Myelopathy 7 Weakness 5 Gait Abnl 4 Sphincter 2 Paraplegia	Causes of AA TR 10 Prior OR 9 SAH 7 Inf 3 Myelo 1 GB 1 AS 1 UNK 1 23LOC CSF 15 CLUMP ENH DISP 12 CSwell Hi T2 11 ArachS 6 CORDA 5 Syrinx 3 IntraCA Others Arachnoid Cysts Dural/Pial Enhancement	Surgery 10 patients 7 Laminectomy 5 Lysis of adhesions Additional procedures 4 cyst fenestration 4 Intradural Exploration 3 Syrinx Drainage 2 Syringopleural Shunt 2 Duraplasty 1 Myelotomy 1 Ventricular drain 1 ACDF	10 Surgery Only 1 Returned to work: 4 Brief improved/worse 3 no better 1 slow decline 1 mixed improved/worsening AA Clinical diagnosis Not Correlate with MR/Myelo-CT Findings
Parenti *et al.*[8] Clin Neurol Neurosurg 2020	Lumbar AA multiple etiologies 28 Patients Lumbar AA on MR 2012-2018 Retrospective	Determine if MR features of lumbar AA associate with clinical findings Majority Postop or Post-INF	MR Findings Cauda Equina Nerve root Thickened Adhesions Levels ENH Delamarter Group*	IN general MR findings did not associate with clinical features of lumbar AA MR findings in lumbar AA offer limited insight into the clinical presentation of the disorder	"No association exists between findings of arachnoiditis observe on MR and those assessed clinically"
Tsuchida[10] Pain Pract 2020	17 High Risk Lumbar AA versus. 18 no risk Patients chronic LBP	Focus: MR Intrathecal mobility of nerve roots MR supine and Prone	11 Axial T2 Images L2-L5S1 Assess low intensity areas dorsal half/ total low intensity area dural sac	Nerve Roots Lose Potential to Migrate (ventrally) in Dural Sac in Gravitational force direction (prone) on MR	AA "…lack of definitive diagnostic criteria Prone MR useful to document lack ventral root mobility prone MR

(*Contd...*)

Table 1: (*Continued*).

Author [Ref] journal year	Study design	Clinical data	Causes of AA	Imaging Findings	Treatment/surgery conclusions
El Homsi *et al.*[4] JMRI 2021	Prior imaging studies of lumbar AA Based on small sample-inconsistent results 2009–2018 "…demonstrated lack of consensus and clarity in the classification system of lumbar AA"	96 AA Patients average age 61.3 43 F 53 M MR Findings 49 Postop 29 Deg 6 VFx, 3 EPI/SAH 1 INF 1 Other	sagittal/axial t2 1.5 and 3 t mr reviewed by 2 msk and 3 neurology radiologists blinded to design/no clinical data	Used Delamarter's 3 Groups Added 4th group: "Non-specified" indeterminate imaging: Not fall into 3 classical groups	Post-surgical 51% Deg changes 30% Most common About 7–55% Classified group 4 Poor classification agreement between readers
Epstein[5] Surg Neurol Int 2021	Intraop DT/CSF fistulas 8.7–9.5% primary lumbar OR Critical detect early and Reoperative 3–4 Weeks to avoid AA	Recurrent CSF leaks 8.1–17% of cases Diagnose CSF Leaks; MR/Myelo-CT If Needed	Findings (delamarter criteria) MR/Myelo CT: Roots aggregated centrally, Roots along peripheral/empty sac, soft tissue Masses in SA Space Interventions rarely useful EBP Inject FG/FS	Optimal direct surgical occlusion Microscope adequate exposure 7–0 gore-tex sutures muscle dural patch graft suture anchors microfibrillar collagen FS/FG	Conclude Lumbar AA common result failure diagnose and treat postop CSF leaks Best treatment; diagnose CSF fistula early and repair

CAA: Chronic adhesive arachnoiditis, RR: Retrospective review, F: Females, M: Males, TR: Trauma, Prior OR: Prior Surgery, SAH: Non Traumatic subarachnoid hemorrhage, INF: Infect, Myelo: Myelography with iophendylate, GB: Guillain-Barre syndrome, AS: Ankylosing spondylitis, UNK: Unknown causes, LOC: Loculated, CSF: Cerebrospinal fluid, CLUMP: Nerve root clumping, ENH: Enhancement, DISP: Displacement, CSwell: Cord Swelling Hi T2: High Cord signa on T2 Images, ArachS: Arachnoid septations, CordA: Cord atrophy, IntrathCA: Intrathecal calcifications, OR: Surgery, MR: Magnetic resonance imaging, Myelo-CT: Myelogram-CT findings, LBP: Low back pain, Delamarter Group: 3 Defined findings (see paper), SA: Subarachnoid (Space), JMRI: Journal magnetic resonance imaging, VFx: Vertebral Fractures, Deg: Degenerative, EPI: Epidural, MSK: Musculoskeletal, DT: Dural tears, EBP: Epidural blood patch, FG: Fibrin glue, FS: Fibrin sealant, SNI: Surgical neurology international, LAM: Laminectomy, No-InstrF: Non instrumented fusion, Fus: Fusion, Abnl: Abnormality, OYL: Ossification yellow ligament, ESI: Epidural steroid injection, ID: Intradural, Br Med J: British medical Journal, ACDF: Anterior cervical diskectomy/fusion, TL: Thoracic and lumbar spine, L-LS: Lumbar and lumbosacral, Entire: Entire spinal levels, OB: Obstetric, EPI: Epidurals, TNS: Transient neurologic symptoms

corroborated that lumbar AA could be misinterpreted as; "… tumors of the spinal cord or meninges…" [Table 1].[7,10]

Etiology of adhesive arachnoiditis (AA)/chronic adhesive arachnoiditis (CAA)

The major etiology of AA/CAA typically includes activation of a "…subarachnoid inflammatory cascade…" [Table 1].[1-10] Factors precipitating this cascade include; trauma, myelography, infection/inflammatory reactions, epidural steroid injections/anesthesia, blood in the subarachnoid space (i.e. traumatic/atraumatic), intrathecal catheter placement, and/or prior surgery.[1-10] Horsely could not define a specific etiology for his 21 operated cases: "They are cases of chronic spinal meningitis the causation of which has yet to be finally determined [Table 1].[7] Benoist *et al.* (1980) attributed lumbar AA to prior lumbar surgery after reoperating on 38 patients [Table 1].[2] Rice *et al.* (2004) attributed severe CAA to epidural injections or the subarachnoid administration of contrast media; "Back pain with or without leg symptoms (e.g. pain, paresthesias or weakness) is typical but a wide range of neurological abnormalities have been associated with CAA" [Table 1].[9] In 2014, Hempl *et al.* further reviewed the toxicity of "… new and old drugs and compounds…" for spinal anesthesia as contributors to the clinical syndrome of AA [Table 1].[6] Specifically, they found that Chloroprocaine resulted in a reduced risk of neurotoxicity vs. Lidocaine, while Ropivacaine exhibited a "…smaller neurotoxic potential…" when compared with Levobupivacaine, Procaine, and Bupivacaine."[6] Further, using Chlorhexidine Gluconate for skin disinfection prior to spinal blocks

increased the potential risk for neurotoxicity and adhesive arachnoiditis [Table 1].[6] Anderson *et al*. (2017), in 29 (14%) patients with severe CAA taken from a total series of 209 AA patients, found that the majority of cases of CAA could be attributed to; trauma (10 patients), prior surgery (9 patients), and non-traumatic subarachnoid hemorrhage (7 patients) [Table 1].[1] Parent *et al*. (2020) additionally attributed AA in 28 patients to prior surgery or to prior infections [Table 1].[8] Tsuchida *et al*. (2020) further identified 17 "high risk" patients for lumbar AA (i.e. 15 with prior surgery, and 2 after oil-based myelography) who demonstrated: "… fibrous tissue outgrowth into the subarachnoid space… result (ing) in a fibrous tangle, entrapping the lumbar, and sacral nerve roots and cauda equina…"[Table 1]."[10] In El Homsi *et al*. (2021) series of 96 patients, lumbar AA was largely attributed to; "… postsurgical findings (49 patients), and/or degenerative disease (29 patients)…" [Table 1].[4] In 2021, Epstein emphasized the most common reason for AA was prior surgery, the likelihood of which increased with an intraoperative cerebrospinal fluid (CSF) leak (i.e. incidence primary CSF leaks 8.7–9.5%, incidence for subsequent CSF fistulas 8.1–17%) [Table1].[5] Further, the risk for developing AA was also markedly increased if such dural leaks went undiagnosed and untreated for over 3–4 postoperative weeks [Table 1].[5]

Typical clinical features of AA

Patients with AA may present with a wide array of neurological symptoms and signs [Table 1].[1,5,8,10] Anderson *et al*. (2017) 29 patients with severe CAA displayed significant radiculopathy, back pain, and/or sensory neuropathy [Table 1].[1] Tsuchida *et al*. (2020) 17 patients at "high risk" for lumbar AA exhibited; leg pain, neurogenic claudication, motor/sensory deficits including paraparesis/paralysis and hypesthesia/sensory loss, with or without sphincter dysfunction [Table 1].[10] Parenti *et al*. (2020) 28 patients with lumbar AA displayed;"… heterogenous (*symptoms/signs*) with patients reporting a range of lower extremity neurological symptoms with varying levels of severity".[8] Epstein further summarized the neurological findings for AA as including; "…low back pain, lower extremity weakness, cauda equina syndrome, progressive paraparesis, sensory loss/dysesthesias, and sphincter function loss" [Table 1"].[5]

MR and myelo-CT/plain film myelography (PFM) findings for AA/CAA

Myelograms, Myelo-CT studies, and MR examinations cited multiple imaging findings that may be associated with the clinical syndromes of AA/CAA [Table 1].[1-5,8-10] These findings often utilized by Delamarter *et al*. included classical radiological descriptions for AA (i.e. clumping (centrally, peripherally-empty sac sign), and/or soft tissue subarachnoid masses), while other authors like Tsuchida *et al*. newly added the failure of nerve roots or the cauda equina to migrate ventrally on prone MR studies. [1-5, 8-10]

Benoist *et al*. findings [Table 1][2]

In Benoist *et al*. (1980), 38 patients were diagnosed with lumbar AA attributed to prior lumbar surgery; all findings were confirmed at reoperations. Nevertheless, the authors found no specific correlation between the clinical syndrome of lumbar AA and any specific radiographic abnormalities; "Myelographic patterns did not disclose any correlation with the clinical symptoms…" [Table 1].[2]

Delamarter *et al*. findings [Table 1][3]

In 1990, Delamarter *et al*., based on a review of 24 MR and 20 Myelo-CT/plain film myelographic (PFM) examinations, divided classical findings for AA into 3 Groups; Group I "… conglomeration of adherent nerve roots resting centrally within the thecal sac; Group II "… nerve roots adherent peripherally to the meninges giving rise to an 'empty sac' appearance"; and Group III "…showed a soft tissue mass replacing the subarachnoid space" [Table 1].[3] They also emphasized the close correlation/relationship between MR and Myelo-CT/PFM findings; "MR imaging resulted in accurate diagnosis and had excellent correlation with CT myelography and plain film myelographic findings in the diagnosis of lumbar arachnoiditis".[3] Their emphasis on this close correlation between MR and Myelo-CT studies for arachnoiditis remains critical to this day for those attempting to correlate patients with clinical AA/CAA syndromes and neurodiagnostic images.

Rice *et al*. (2004): Epidural anesthesia for delivery does not lead to CAA [Table 1][9]

Rice *et al*. determined that patients who received epidural anesthesia for labor/delivery were not more susceptible to developing CAA as had been previously thought [Table 1].[9] They further noted that the: "…radiological and pathological findings (i.e. *imaging for CAA*) do not invariably correlate with the clinical features (of CAA)" [Table 1].[9]

Anderson *et al*. findings [Table 1][1]

Anderson *et al*. (2017) observed significant imaging findings (i.e. MR 29 patients: Myelo-CT 7 patients) in their 29 patients specifically selected for severe CAA [Table 1].[1] MR findings included; loculated CSF collections, root clumping (central), peripheralization/empty sac sign, subarachnoid soft tissue masses/pial dural enhancement, cord swelling/high T2 cord signal, distortion/tethering of the cord, syrinx, arachnoid cysts/webs/septations [Table 1].[1] Myelo-CT

studies demonstrated; arachnoid cysts/septations, thickened/clumped roots, intrathecal ossification, atrophic cord changes/enlargement, root lack of filling, other subarachnoid (partial/total block) filling defects, and/or soft tissue lesions throughout dural sac [Table 1].[1] Notably, the majority of nerve root/cauda equina adhesions occurred in the dorsal spinal canal, except for those with the most severe disease, whose adhesions could appear "circumferentially".[1]

Parenti *et al*. (2020) findings [Table 1][8]

Parenti *et al*. (2020) noted variable MR findings for their 28 patients with AA; "…cauda equina nerve root clumping/thickening, adhesion location/levels, enhancement, and (*the*) Delamarter group (*criteria*)" [Table 1].[8] They concluded; "MRI findings in lumbar arachnoiditis offer limited insight into the clinical presentation of the disorder". Further; "In general, MRI findings did not associate with the clinical features of lumbar arachnoiditis" [Table 1].

Tsuchida *et al*. findings (2020)[10]

Tsuchida *et al*. (2020) compared the MR scans performed in 17 "high-risk" AA vs. 18 "no risk" AA patients looking for failure of nerve roots/cauda equina to migrate ventrally on prone vs. supine studies [Table 1].[10] Indeed, they found;"… the intrathecal mobility of nerve roots and the cauda equina is reduced in patients at high risk for lumbar AA." Further, "... conventional MRI which is usually conducted only in the supine position, cannot detect this immobility..." In short, they concluded that many previous studies failed to accurately diagnose AA as they did not include prone MR examinations.

El Homsi *et al*. findings (2021)[4]

In 2021, El Homsi *et al*. diagnosed 96 patients with lumbar AA using Delamarter's 3 Groups [Table 1].[4] To these, however, they uniquely added a 4th "*Non-Specified*" *Group* that was: "…proposed for indeterminate imaging findings that did not fall into the classical groups." They found that between 7-55% of MR studies were classified into the new 4th Group, and that there was a "… very poor classification agreement between readers…"

Epstein (2021) findings [Table 1][5]

Epstein (2021) reviewed Delamarter *et al*. classic 3 Groups based on MR and Myelo-CT studies for AA [Table 1].[5] They showed; "…nerve root clumping, enhancement/displacement, cord swelling/atrophy, and or syrinx formation." [Table 1].[5] Findings for Myelograms/Myelo-CT studies included; Type I: "…empty thecal sac sign (nerve roots adherent to peripheral dura), and Type II: local/diffuse filling defects throughout the thecal sac". Epstein further recommended incorporating Tsuchida *et al*. prone MR findings of a; "… a lack of ventral root mobility with prone MR positioning…", to the analyses of Myelo-CT examinations.

Lack of association between clinical and radiographic AA [Table 1]

Multiple studies have shown that the clinical diagnoses of AA/CAA are not necessarily corroborated by or associated with "diagnostic" radiographic AA abnormalities [Table 1].[1,4,8,9,10] Rice *et al*. (2004) found that "The precise relationship between the pathological (*radiological)* findings and symptomatology has not been defined" [Table 1].[9] Anderson *et al*. (2017) observed: "CAA has a variable imaging appearance on both myelography and MRI. CAA does not have a characteristic distribution…"[1] Further they noted the; "…poorly defined and varying imaging characteristics *(of AA)* may result in a missed or delayed diagnosis". Tsuchida *et al*. concluded; "… it is difficult to diagnose (AA) owing to the lack of definitive diagnostic criteria" [Table 1].[10] They also observed; "… the precise relationship between the complex clinical symptomatology and pathological imaging findings has not yet been defined and validated." Further; "Some MR findings (i.e. clumped nerve roots, empty sac appearance, and deformities of the dural sac) have been proposed as the typical presentation of lumbar AA. However these signs variedly emerge in patients with lumbar AA; therefore they are not established and valid as useful diagnostic symptoms". Parenti *et al*. (2020) noted that lumbar AA has "… multiple etiologies and a spectrum of imaging and clinical characteristics", and that: "In general, MR findings did not associate with clinical features of lumbar AA …"[8] They. concluded: "MR findings in lumbar AA offer limited insight into the clinical presentation of the disorder," and that; "…the extensive array of clinical and imaging findings exhibited in lumbar AA have been purported to lack association by multiple authors" [Table 1]. Also when El Homsi *et al*. (2021) evaluated the MR findings for patients with AA, they observed that previous imaging studies … reported, at best, "…inconsistent results."[4]

Surgery ineffective for lumbar AA [Table 1]

Surgical intervention typically does not result in sustained neurological improvement for patients with AA/CAA [Table 1].[1,5-8] Horsely in 1909 observed; " I have seen a relatively large number of such cases, on most of which I have performed a laminectomy and subdural mercurial irrigation".[7] He goes on to state: "I do not mean to say that they all yield to surgical treatment-I wish they did but so many do that it is quite clear that earlier diagnosis would have saved, in my opinion, the majority of not all of the cases". In Anderson *et al*. (2017) series, 10 of 29 CAA patients had surgery; the results were poor as 4 transiently improved

then worsened, 3 did not improve, 1 continued to decline, 1 was at first minimally improved and then worsened, and only 1 had improvement allowing him to return to work [Table 1].[1] They concluded; "Patients who have improvement after surgery often have relapses and progressive symptoms occur later, and their long-term prognosis after surgery remains poor". Parenti *et al.* (2020) noted that medical or surgical treatment modalities were typically ineffective for lumbar AA: "Despite surgical and medical advancements, therapeutic options are limited and mainly include microsurgical lysis of adhesions, corticosteroid therapy, and chronic pain control".[8] Epstein (2021) summarized the poor results of surgery for AA: "… the majority of clinical studies acknowledge that postoperative lumbar AA is not a surgically remediable lesion." The best way, therefore to avoid lumbar AA, was for: ". early recognition and repair of such persistent postoperative recurrent CSF fistulas/DT…"[5]

CONCLUSION

The clinical diagnoses of AA/CAA do not require radiographic confirmation on myelographic, Myelo-CT, and/or MR studies [Table 1].[1-5,8-10]

Declaration of patient consent

Patients' consent not required as patients' identities were not disclosed or compromised.

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Conflicts of interest

There are no conflicts of interest.

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