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 necessarily show associated neuroradiographic abnormalities on myelograms, Myelo-CT studies, or MR. Rather, the clinical syndromes of AA/CAA may exist alone without the requirement for radiographic 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
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) | Study design | Clinical data | Causes of AA | Imaging Findings | Treatment/surgery conclusions |
|--------------|--------------|---------------|--------------|------------------|------------------------------|
| Horsley [7] | 21 Cases based on operative findings with AA | “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.” |
| Benoist et al. [2] | 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] | 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 Etiology of AA Epi or SAH Contrast Media Lead to CAA | Group 3 : Soft tissue mass replacing SA Space | MR accurate diagnosis Excellent Correlation -Myelo-CT+Myelogram Lumbar AA |
| Rice et al. [9] | Suggested OB EPI lea to CAA Clinical Criteria Back pain Leg Pain Neuro Deficits | “Because of the varied symptomatology, clinical diagnosis (CAA) is difficult” | “...radiological and pathological findings do not invariably correlate with the clinical features (of AA)” | The precise relationship between the pathological findings and symptomatology has not been defined” |

(Contd...)
| Author (Ref) | Study design | Clinical data | Causes of AA | Imaging Findings | Treatment/surgery conclusions |
|-------------|-------------|--------------|--------------|----------------|-----------------------------|
| Hampl et al.[6] Curr Opin Anesthesiol 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 Glucanate 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 7 Weakness 5 Gait Abnl 4 Sphincter 2 Paraplegia | Causes of AA TR 10 Prior OR 9 SAH 7 Inf 3 Myelo 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 MR Findings Cauda Equina Nerve root Thickened Adhesions Levels ENH Delamarter Group* | 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 | 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...)
corroborated that lumbar AA could be misinterpreted as; “...tumors of the spinal cord or meninges...” [Table 1].

**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]. 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. 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].

Benoist *et al.* (1980) attributed lumbar AA to prior lumbar surgery after reoperating on 38 patients [Table 1]. 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]. In 2014, Hempi *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]. 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. Further, using Chlorhexidine Gluconate for skin disinfection prior to spinal blocks

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**Table 1: (Continued).**

| Author [Ref] | Study design | Clinical data | Causes of AA | Imaging Findings | Treatment/surgery conclusions |
|--------------|--------------|---------------|--------------|------------------|------------------------------|
| Epstein et al. | 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 II 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 disectomy/fusion, TL: Thoracic and lumbar spine, L–LS: Lumbar and lumbosacral, Entire: Entire spinal levels, OB: Obstetric, EPI: Epidurals, TNS: Transient neurologic symptoms
increased the potential risk for neurotoxicity and adhesive arachnoiditis [Table 1].
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].

Parent et al. (2020) additionally attributed AA in 28 patients to prior surgery or to prior infections [Table 1].

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].

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].

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]. 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].

Typical clinical features of AA

Patients with AA may present with a wide array of neurological symptoms and signs [Table 1].

Anderson et al. (2017) 29 patients with severe CAA displayed significant radiculopathy, back pain, and/or sensory neuropathy [Table 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 hysthesia/sensory loss, with or without sphincter dysfunction [Table 1].

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.”

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].

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].

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.

Benoist et al. findings [Table 1]

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].

Delamarter et al. findings [Table 1]

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]. 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”.

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]

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]. 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].

Anderson et al. findings [Table 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].

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]. 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…”[9] 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…”[9] 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].[1,4,8] 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”[2] 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
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Conflicts of interest
There are no conflicts of interest.

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