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Immunotherapies in chronic adhesive arachnoiditis - A case series and literature review

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ABSTRACT

Chronic spinal adhesive arachnoiditis (CSAA) is a rare condition with limited therapeutic options. Surgical treatment proves effective in approximately 60% of cases. Conservative treatment options have not been extensively investigated. Here, we report the course of the disease, analyze the effect of immune treatments in patients with CSAA who were treated in the University Hospital Essen between 2015 and 2020, and conduct a literature review. Three out of four patients showed no improvement after treatment with corticosteroids, methotrexate, or plasmapheresis. All non-responders suffered from CSAA for several years, while one patient who had a disease duration of less than one month fully recovered. It is necessary to verify whether treatment at an early stage of the disease is better than treatment after chronic adhesion manifestation, as it interrupts the development of adhesions and all subsequent complications.

1. Introduction

Chronic spinal adhesive arachnoiditis (CSAA) is characterized by inflammation and subsequent adhesion of the arachnoid layer, which may lead to progressive myelopathy [1]. It is usually caused by spinal cord (SC) lesions such as subarachnoid hemorrhages or alterations of the natural anatomic structure of the spine such as from spinal surgery [2–6]. During the course of the disease, cysts and venous congestion may occur with subsequent edema [7–9], leading to central spinal or radicular symptoms [3,10,11]. Symptoms depend on the affected nerve root or SC level. The diagnosis is based on the patient's history, clinical presentation, and magnetic resonance imaging (MRI). CSAA is a very rare condition with fewer than 1000 cases since its first description in the literature [1,12]. Therefore, no therapeutic trials have been carried out so far. CSAA remains a disease with limited efficacious options. While in a few cases surgical intervention led to an improvement by cyst fenestration or shunt operation to reduce compression and restore the flow of cerebrospinal fluid and/or venous drainage, in several cases, a progression of adhesions and edema was described [3,6]. CSAA is mainly treated symptomatically with analgesics or physical therapy. To date, corticosteroids have not been extensively investigated, with only a few single case reports describing the effect of corticosteroids [11,13–16].

Here, we report the course of the disease and analyze the effect of immune treatments on patients who were diagnosed and treated with CSAA in the University Hospital Essen, Germany between 2015 and 2020 and conduct a literature review.

2. Methods

By a structured search in our medical database according to ICD-10 (International Statistical Classification of Diseases and Related Health Problems) diagnoses (G03.x), we identified four cases of CSAA who were diagnosed in the University Hospital Essen, Germany between 2015 and 2020. The following data were selected and evaluated: diagnosis, year of birth, age at diagnosis, age at manifestation, sex, cause, symptoms, neurological examination, course of disease, diagnostic procedures (MRI, laboratory values, cerebrospinal fluid (CSF) findings, evoked

Abbreviations: CSAA, Chronic spinal adhesive arachnoiditis; CSF, Cerebrospinal fluid; ICD, International Statistical Classification of Diseases and Related Health Problems; i.v., intravenous; MCTD, Mixed connective tissue disease; MRI, Magnetic resonance imaging; SC, Spinal cord; s.c., subcutaneous.

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potentials), therapeutic procedure, and clinical outcome parameters (Table 1). The ethical review board of the faculty of medicine of the University of Duisburg-Essen gave its approval for this study.

2.1. Case report #1

A 66-year-old female patient developed CSAA after several periradicular infiltrations for pain caused by scoliosis, resulting in the formation of an abscess and subsequent meningitis. Two years after infiltration therapy, she developed a progressive gait disorder with urinary and fecal incontinence. An MRI was performed 2.5 years later following a rapid deterioration of the gait disorder and fine motor skills showing confluent, intraspinal, arachnoid noninflammatory cystic formations (Fig. 1a). However, CSAA was not diagnosed until six months later, and a second MRI showed similar findings: arachnoid cystic formation with compression on the already partially atrophic thoracic myelon. Neurosurgical intervention was not considered advisable. The electrophysiologic studies were consistent with polyradicular damage. One gram of methylprednisolone i.v. was administered daily for five consecutive days followed by a gradual decrease orally. Three months later, the MRI scan result was similar to the prior findings. After further deterioration, symptomatic analgesic treatment with tapentadol and mirtazapine was started, with increasing dosages. Further treatment with corticosteroids and methotrexate was recommended, but the patient was lost to follow-up.

2.2. Case report #2

A 72-year-old female patient developed CSAA after several periradicular infiltrations were administered for the treatment of severe back pain caused by scoliosis. The diagnosis of a CSAA was not made until 1.5 years after the manifestation of mild paraparesis resulting in gait disorder and reduced walking distance, pallhypesthesia of the right leg, urinary incontinence, and spinal ataxia. Severe back pain, previously attributed to scoliosis, persisted. The MRI scan revealed extensive myelon edema and an arachnoid cyst in the proximal cervical spine (Fig. 1b). After CSAA diagnosis, the patient was initially given steroid treatment, with one gram of methylprednisolone administered daily over five consecutive days. However, her symptoms continued deteriorating and included fecal incontinence. A follow-up MRI showed no relevant changes. CSF analysis revealed elevated protein (105 mg/dl) without pleocytosis. Q_{Alb} (albumin quotient CSF/serum 18.0×10^{-3}) was also increased as an indication of disturbed CSF flow dynamics. During the subsequent neurological rehabilitation therapy, the patient's condition further deteriorated. Treatment with methotrexate 15 mg subcutaneously (s.c.) once a week was commenced. The findings of the MRI three months later were unchanged. Thus, treatment was discontinued, and neurosurgical intervention was discussed but not advisable. Due to increased spinal ataxia and newly developed neuropathic pain in the legs, further therapeutic options were discussed. Five courses of plasmapheresis were then performed, which did not alter symptoms. Disease progression was not preventable, and she became dependent on a rollator. The most recent MRI depicted slightly increased degeneration with the development of syringomyelia. Currently, the patient is treated only symptomatically.

2.3. Case report #3

A 72-year-old female patient developed CSAA three weeks after spinal hemorrhage at vertebral level L5/S1 under oral anticoagulation. After resolving the hemorrhage, the patient's symptoms persisted, including pain as well as pain-related impairment of walking. The MRI

Table 1
Overview of the CSAA cases: patient characteristics, suspected causes, symptoms, MRI findings, therapy, and outcome.

Case	1	2	3	4
Sex	Female	female	female	male
Age	66 years	72 years	72 years	41 years
Suspected cause	periradicular infiltrations resulting in an abscess and meningitis	periradicular infiltrations	spinal bleed	lumbar punctures due to meningitis
Time until manifestation	2 years	unknown	3 weeks	27 years
Time until diagnosis	3 years	1.5 years	1 week	2 years
Symptoms/ Neurological examination	gait disorder, mobile with rollator, slight proximal paraparesis, hyperreflexia left leg, impaired fine motor skills, neuropathic pain, bimalleolar pallanesthesia, hypoesthesia legs, sensory ataxia, urinary and fecal incontinence	gait disorder, mobile with rollator, mild proximal paraparesis, spinal ataxia, hyp- and dysesthesia right leg, pallhypesthesia right leg, urinary and fecal disturbances, back pain, neuropathic pain legs	back pain, lumboischialgia left, pain-related reduction of walking distance, no neurological focal deficits	gait disorder, mobile with rollator, falls, mild spastic paraparesis, hyperreflexia legs, Babinski positive left, hypesthesia legs, pallanesthesia bimalleolar, sensory ataxia, enuresis
MRI findings	confluent intraspinal lumbar arachnoidal cyst formation with partially encapsulated subarachnoidal space and swollen cauda fibers in a sagittal T2 sequence	extensive myelon edema, irregularly configurated spinal cord C6 - Th12 with hyperintensity C7 - Th11 compressed by arachnoidal cyst, cystic formations in neuroforamina right at Th 9/10 and Th 10/11 in a sagittal T2 sequence and CISS sequence, no contrast agent uptake	intraspinal lumbar hypointensities in a sagittal T2 sequence, intraspinal hyperintense lesion in a T1 sequence, cystic mass dorsally compressing the spinal cord at L5/S1, myelon edema, adhesions of cauda fibers and meningeal contrast agent uptake	in a sagittal T2 sequence myelon edema, cystic and partially isointense to CSF changes Th9–12, stenosis C4/5 and C5/6, no contrast agent uptake
Therapy	1. high-dose i.v. cortisone over 5 days and decrease orally 2. recommendation: methotrexate and oral cortisone	1. high-dose i.v. cortisone over 5 days 2. neurological rehab 3. methotrexate 15 mg s.c. per week 4. wait and observe 5. plasmapheresis five courses	1. high-dose i.v. cortisone over 5 days 2. high-dose i.v. cortisone over 5 days 3. high-dose i.v. cortisone over 5 days 4. high-dose i.v. cortisone over 5 days	1. high-dose i.v. cortisone over 5 days
Outcome	1. deterioration 2. lost to follow-up	1. no effect 2. deterioration 3. no effect 4. deterioration 5. deterioration	1. improvement 2. pain alleviated 3. full recovery 4. stable, no symptoms 5. stable, no symptoms	1. first improvement, then deterioration back to initial condition, new lumboischialigia left

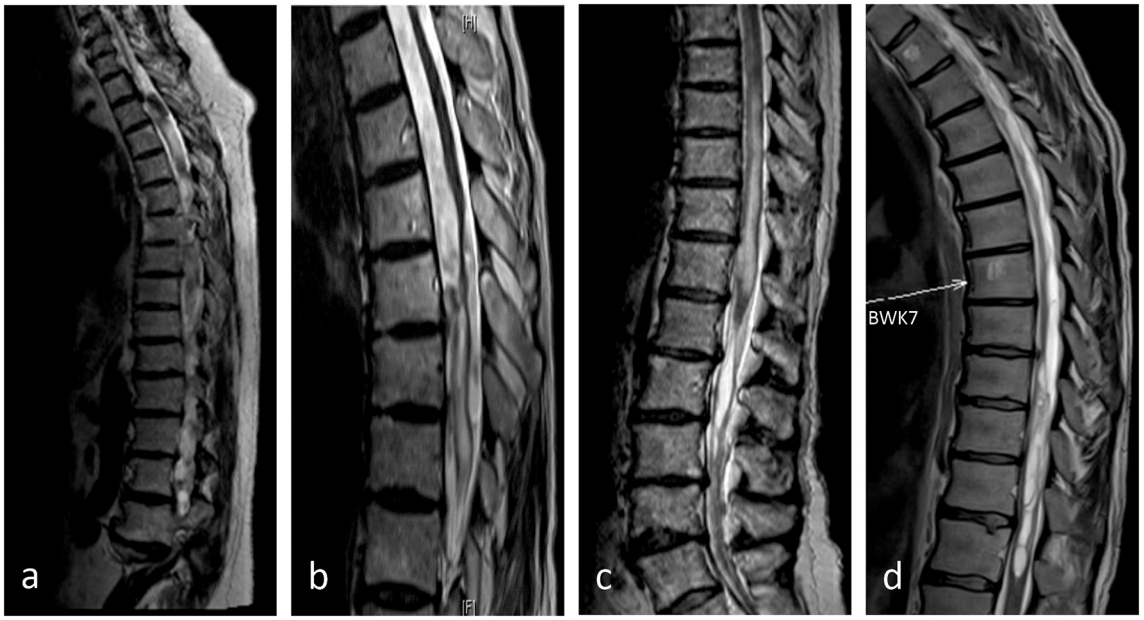

Fig. 1. Magnetic resonance imaging of the spine showing arachnoiditis. (a) Case 1: The sagittal T2-weighted image shows confluent intraspinal lumbar arachnoidal cyst formation with partially encapsulated subarachnoidal space and swollen cauda fibers. (b) Case 2: The sagittal T2-weighted image shows myelon edema and arachnoidal adhesions in the upper thoracic level. (c) Case 3: The sagittal T2-weighted image shows longitudinal myelon edema and adhesions of the cauda fibers. (d) Case 4: The sagittal T2-weighted image shows myelon edema, longitudinal cystic changes and arachnoidal adhesions in the cervical and thoracic spine.

scan three weeks after the hemorrhage showed an intraspinal hyperintense lesion in the T1-weighted sequences and a cystic mass dorsally compressing the spinal cord consistent with CSAA (Fig. 1c). One gram of methylprednisolone was administered daily over five consecutive days. As a consequence, symptoms improved and supportive therapy with gabapentin was no longer required. However, continuous adhesions of the cauda fibers were visible in the next MRI. Another cycle of one gram methylprednisolone daily was then administered for five days, which reduced the pain. The walking distance was no longer restricted, and follow-up MRI showed a decrease in meningeal gadolinium enhancement. In view of these positive effects, two three-day cycles of methylprednisolone three months apart were recommended, and further follow-ups are planned.

2.4. Case report #4

A 41-year-old patient was diagnosed with CSAA after developing rapidly progressive gait disorder and hypesthesia of the legs over the past two years. The suspected causes of CSAA were several lumbar punctures performed due to bacterial meningitis 27 years ago as well as recurring falls. The neurological examinations revealed mild central sensory motor paraparesis with pyramidal signs, bimalleolar pallanesthesia, and spastic atactic gait disorder requiring the help of a rollator to walk. The MRI showed chronic arachnopathy, and cystic changes were partially isointense to CSF (Th9–12) (Fig. 1d). There was no gadolinium uptake. Surgical intervention was not performed due to known tracheal stenosis. One gram of methylprednisolone was administered daily over five consecutive days, leading to a transient slight improvement. The MRI scan revealed no changes. Six months later, the patient complained about lumboischialgia, which could not be ascribed to a nerve root because of diffuse pain symptoms. Physical and pain therapy did not resolve symptoms.

3. Discussion

Here, we report four cases of CSAA that all received immunosuppressive treatment, including high-dose i.v. steroids, two patients received methotrexate additionally, and one patient received plasmapheresis. Among them, one patient who received steroids at a very early stage of the disease showed improvement after treatment, while the other patients did not benefit from immune treatment.

We performed a literature search on PubMed using the term “adhesive arachnoiditis”. We found 690 results. In the last 20 years, 71 case reports and case series with 165 cumulative patients were published.

CSAA as its own disease entity was first recognized in 1909 [12]. CSAA affects most patients over 50 years (57%), with a mean age of 52 ± 14 years. At risk are also young women who receive epidural anesthesia during childbirth [15,17]. In general, women (60%) are diagnosed with CSAA more often than men. Different causes for CSAA have been described, although it is important to note that the time between cause and manifestation varies greatly, with reports of up to 50 years [18]; therefore, only assumptions can be made. Furthermore, sporadic cases with no remarkable history explaining CSAA have been reported (3.6%) [4,19,20]. Historically, infections [5,18,21–24], mostly tuberculous meningitis [5,25–29], or myelographies due to the application of an oil-based contrast agent [2,30–35] played a major role as the cause of CSAA. Spinal surgery [6,11,22,36–40] and subarachnoid hemorrhages [3] continue to play a key role in the development of CSAA. In these cases, CSAA often does not occur until years after the event, whereas after epidural blood patches [10,13] or injection of chemical substances [11,15,16,35,41], CSAA manifests earlier. In recent years, spinal epidural and spinal block anesthesias [42], radicular infiltrations [14] and intrathecal injections with steroids [43] have become increasingly causal for CSAA development.

To date, the pathomechanisms are not well understood, but several observations indicate that immunological processes are responsible for developing CSAA. An animal model by da Silva et al. [44] in 2019 showed that lumbar punctures through tattooed skin lead to acute inflammatory changes such as perivascular lymphocytic infiltrates after 30 days in rabbits. After 360 days, 50% of the rabbits had histologically developed arachnoiditis. These findings are supported by Bilello et al. [45], who analyzed the concentrations of the inflammatory factors alpha-1-antitrysin and myeloperoxidase in the serum of patients with CSAA. Compared with healthy subjects, the concentrations were higher in patients with CSAA.

Before MRI scanners became available, CSAA was often diagnosed

using myelography [6,19] and later CT myelography [40,46,47]. In many cases, the diagnosis was made during surgery [6]. Today, MRI scans are considered the gold standard when diagnosing arachnoiditis [3,11,37,48]. Anderson et al. [49] described the great variety of MRI findings in 29 cases and CT myelography in seven cases. Features noted on CT myelography included partial or complete myelographic block, arachnoid septations and cysts, cord displacement, thickened or tethered nerve roots, intrathecal calcifications, cord atrophy or expansion, nerve root nonfilling, and soft-tissue mass in the subarachnoid space. MRI findings included loculated CSF collection, nerve root clumping, enhancement, and displacement, cord swelling with increased T2 signal, arachnoid septations, cord atrophy, syrinx, and intrathecal calcifications. Arachnoid cyst formation was the most common MRI finding and was present in 79% of the patients. A syrinx was present in five of the 29 cases. This does not reflect the majority of reported cases. Most of the authors report cases with arachnoiditis and syringomyelia [23,25,28,47], therefore cases of arachnoiditis without these findings were underrepresented. This is important to consider when treating patients with arachnoiditis. In this series no enhancement of the spinal cord was seen in any patient. In our case series and other case reports [11] gadolinium enhancement of the meninges and spinal nerve roots as an indication of a possibly acute inflammatory process was seen. By the way Parenti et al. showed no association between imaging and clinical features [50]. In our case series there is also no correlation. No preference towards a certain level of the spinal column was observed either. Tsuchida et al. [51] searched for new MRI diagnostic criteria that could make it possible to diagnose CSAA in earlier and asymptomatic stages of the disease. Reduced nerve root mobility has been suggested as an early marker for the development of CSAA. MRI images in supine and prone positions were compared, and the proportion of the low-intensity area in the dorsal half to the total low-intensity area in the dural sac for each axial view in T2-weighted images between the vertebral L2 and L5/S1 levels was calculated. It was shown that in patients with a high risk of developing CSAA, the low-intensity area in the dorsal half of the dural sac was relatively larger than that in the no-risk patients.

In the literature there is hardly any information on CSF changes in arachnoiditis patients. Since the diagnosis is often made by MRI or surgically, a CSF examination is not carried out or the findings are not considered to be reportable. An increased protein content without pleocytosis and/or an increased albumin quotient are conceivable as an indication of a disturbed CSF flow, as in our 2nd case report. However, a mild, lymphocytic pleocytosis is also conceivable as an expression of an intraspinal inflammatory reaction not caused by pathogens.

Most cases described are being treated surgically, and only a single case series specifically describes conservative treatment and the outcome. Approximately 60% of patients initially profited from surgical treatment. Maulucci et al. [38] reviewed 72 cases of arachnoiditis ossificans in 2014, in which 50% of the patients profited from surgical treatment. Different surgical techniques have been described in the literature [6,36,52–58], but no technique has proven superior to the others, possibly because there are so few cases. Most commonly, laminectomy and lysis of adhesions were performed. Additional surgical procedures include cyst fenestration, intradural exploration, syrinx drainage, shunt placement, duraplasty, myelotomy, intraventricular drain placement, discectomy, and anterior fusion. In the field of shunt placement, various types of shunts have been implanted, i.e., cystoperitoneal cystopleural, cystosubarachnoid, ventriculoperitoneal, etc. [24,58]. Surgical treatment of adhesive arachnoiditis seems to be effective in the short term, but the long-term outcome proved unsatisfactory. Johnston et al. [53] described the short- and long-term outcomes of 28 arachnoiditis patients treated with microscopic adhesiolysis. While initially the patients reported an improvement, on the long run, symptoms worsened, and the adhesions recurred. The follow-up time was up to six years. In regard to syringomyelia, different outcomes due to patient characteristics and surgical techniques are reported. Koyanagi et al. [24] described the outcome of 15 patients that were treated with SP, SS or VP shunts. Eight patients required further shunting operation two months to 12 years after first surgery. Neurologic improvement was obtained in nine patients. One patient remained stable, and one patient showed gradual deterioration. All in all, the outcome was positive with approx. 50% of the patients. This is supported by Sgouros et al. [59] who observed clinical stability 10 years after shunting operation in just 50% of his patients. As an alternative to shunting Klekamp et al. [60] postulates better results with microsurgical dissection of the arachnoid scar and decompression of the subarachnoid space with a fascia lata graft.

In the only study by Grahame et al. [61] where conservative treatment was analyzed, 26 patients were treated with D-penicillamine. The study was randomized, double-blind with a six-month crossover design; switch from D-penicillamine 500 mg/d to placebo and vice versa. The three-month follow-up showed no statistically significant effect. There are several case reports in which, unfortunately, conservative treatment is mentioned but without specifically mentioning the treatments used [2,14,30,39,62,63]. From 2015 to 2020, seven cases were reported in which patients were treated with corticosteroids. In six patients, treatment was ineffective [11,13–16]. Clark et al. [48] described one patient who was treated with corticosteroids because he was initially misdiagnosed with neurosarcoidosis. This treatment initially led to an improvement but over the next 4 years the patient deteriorated. Then she was treated with oral corticosteroids and infliximab. This led to no improvement, after which the correct diagnosis arachnoiditis was made.

The observations in our patients are in line with these findings. Three out of four patients showed no improvement after corticosteroid treatment. Methotrexate and plasmapheresis also had no positive effects. Patients suffering from CSAA for several years did not respond to immune therapy, while one patient fully recovered after four cycles of steroid treatment. This patient had a rather short disease duration, and his/her MRI findings were less pronounced than that of the other patients.

This leads to the hypothesis, that a high-dose cortisone treatment is most effective in early stages of the disease because it intervenes in the pathomechanism and therefore interrupts the development of the adhesions and all following complications. To make early diagnosis possible, it is necessary to discover diagnostic criteria for the early stages of the disease, such as the reduced mobility of the nerve roots in high-risk patients [51].

One of the limitations of our case series is that the patients were only treated conservatively. There were no patients who received combination treatment. We do not know whether surgical treatment would have improved our patients. In this regard, however, it should be noted that in our case series no patient showed a syringomyelia at the time of diagnosis and most of the cases described in the literature related to the surgical treatment of a syringomyelia. One of our patients developed a syringomylia during the course of the disease, so surgical treatment was discussed.

However, an isolated conservative therapy cannot be recommended for most patients, especially not for patients with a prolonged disease duration. Our experience shows that expanding immunosuppressive therapy does not lead to any improvement if corticosteroids are ineffective. Methotrexate and even plasmapheresis had no effect on disease progression in those patients who got worse on corticosteroids. All of this leads to the question of whether combination therapy should be preferred. Both combinations, corticosteroid therapy before or after an operation, or even both, are conceivable. It can be assumed that immunosuppressive therapy, especially after an operation, could prevent the formation of new adhesions.

To support the hypothesis that immunotherapies are effective in interrupting the development of the disease, further studies are needed.

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Ethics approval

This research study was conducted retrospectively from data obtained for clinical purposes. The ethical review board of the faculty of medicine of the University of Duisburg-Essen gave its approval for this study. The study have been performed in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki and its later amendments.

Consent to participate

Not applicable.

Consent for publication

Not applicable.

Availability of data and material

The datasets used and analyzed for the current short report are available from the corresponding author upon request.

Authors' contributions

All authors contributed to the study conception and design. Material preparation, data collection and analysis were performed by Jana Hackert, Louisa Maßmann and Tim Hagenacker. The first draft of the manuscript was written by Jana Hackert and Louisa Maßmann and all authors commented on previous versions of the manuscript. All authors read and approved the final manuscript.

Declaration of Competing Interest

None.

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