

Prevalence of sacral spinal (Tarlov) cysts in Persistent Genital Arousal Disorder

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Abstract

Introduction:

Neither consistent etiology nor treatment have been established for Persistent Genital Arousal Disorder (PGAD), which is characterized by uninvited, unwelcome, and distressing genital sensation. Sacral (Tarlov) cysts, which form on dorsal (sensory) roots, most commonly of S2 and S3 in the sacral spine, are reported to produce genital symptoms that bear similarities to those described for PGAD.

Aims:

The present study ascertained the incidence of Tarlov cysts in the sacral spine of women with PGAD symptoms.

Methods:

Women in a PGAD internet support group were asked to submit MRIs of their sacral region to the investigators, who evaluated the MRIs for the presence or absence of Tarlov cysts.

Main Outcome Measures:

The presence or absence of Tarlov cysts at the level of the sacral spine.

Results:

Tarlov cysts were present in 12 of the first 18 (66.7%) MRIs submitted to the investigators by women who suffer from PGAD symptoms. By contrast to this incidence, that of Tarlov cysts reported in the literature for large samples of the population observed for various disorders (e.g., lumbosacral pain) is 1.2% - 9.0%.

Conclusion:

Tarlov cysts have been described in the literature as producing paresthesias and genital sensory disturbances. Hence, at least some cases of PGAD might be considered to be a Tarlov cyst-induced paresthesia. Based on the relatively high occurrence of Tarlov cysts currently observed in women who suffer from PGAD symptoms, it would seem advisable to suspect Tarlov cysts as a possible organic etiological factor underlying PGAD.

Introduction

Origin of a clue: This study began serendipitously. A colleague mentioned to one of us (BRK) that his wife, diagnosed with Persistent Genital Arousal Disorder, showed an incidental MRI finding of Tarlov cysts, and asked if they might produce pelvic vasocongestion and hence her discomfort. Sacral cysts, first described by I.M. Tarlov in 1938 [1] are occasionally found incidentally in the course of radiological examination of the lumbosacral spine for various disorders. Review of literature on Tarlov cysts (also termed, "sacral cysts" [2] indicated that they form characteristically at the S2 and S3 dorsal root ganglia, ballooning out as a result of filling with cerebrospinal fluid, contain dorsal root nerve fibers in the wall and/or internal portion of the cyst, and generate paresthesias. Because the S2 and S3 dorsal roots convey the sensory pudendal and pelvic nerves, which innervate the external and internal genitalia (for review, [3]), could they generate the abnormal sensations characteristic of PGAD?

What is Persistent Genital Arousal Disorder?: PGAD is a complex pathology with multiple etiologies and with variably effective therapies. It is a perplexing condition characterized by high levels of genital arousal occurring in the absence of subjective interest or desire. PGAD sufferers are women who experience intrusive, unsolicited and seemingly spontaneous genital arousal that can be unrelenting. This arousal can persist for hours, days or even longer, despite attempts to relieve it with sexual activity or orgasm, which at best provide only brief relief from the symptoms. Attempts to quell the genital arousal by engaging in masturbation or sexual activity may lead to brief relief, no relief, or even more arousal and activation. First characterized and termed Persistent Sexual Arousal Syndrome (PSAS) by Leiblum and Nathan [4, 5], the condition was later renamed PGAD by Leiblum [6] to clarify that the disorder was a problem of genital, rather than sexual, arousal.

The following five diagnostic features of the disorder were characterized by Leiblum and Nathan [4, 5]:

- 1) The physiological responses characteristic of sexual arousal persist for an extended period (hours to days) and do not completely subside on their own;
- 2) The signs of physiological arousal do not resolve with ordinary orgasmic experience, and may require multiple orgasms over hours or days to remit;
- 3) These physiological signs of arousal are usually experienced as unrelated to any subjective sense of sexual excitement or desire;
- 4) The persistent sexual arousal may be triggered not only by sexual activity, but also by seemingly nonsexual stimuli or by no apparent stimulus at all; and
- 5) The physiological signs of persistent arousal are experienced as uninvited, intrusive and unwanted.

A diagnosis of PGAD is made, based upon the presence of all five of the above features, when distress is rated as moderate or severe (a score of 2 or 3 on a scale in which 0 = no distress and 3 = extreme distress) [7]. Women who complain of PGAD can be severely distressed, especially when the symptoms continue for extended

duration without relief. This property of PGAD predisposes some women to become severely depressed and even suicidal [8]. As characterized by Goldstein et al. [9], “In our experience, women affected with the condition are frequently suicidal, socially ostracized, isolated, frustrated, miserable, embarrassed, and extremely humiliated.” The shame and embarrassment attached to the symptoms has likely contributed to the phenomenon going previously unrecognized and underreported to health care providers, which could account, at least in part, for the absence of reliable data on the prevalence of PGAD.

By contrast with PGAD, some women experience persistent genital arousal as a normal, pleasant and even reassuring experience. However, those women differ from the PGAD sufferers in that their arousal is neither continuous nor unrelenting, and does not cause them distress [10, 11].

Sensations associated with PGAD: Complaints by PGAD sufferers have been described as clitoral tingling, irritation, vaginal congestion, vaginal contractions, throbbing, pressure, pain, and in some cases, spontaneous orgasms [12].

Sensations associated with Tarlov Cysts: Symptoms of Tarlov cysts include: pain in perineum, vagina, penis, buttock, leg, lower back, sacrum, or coccyx, dyspareunia, proctalgia, bladder dysfunction, urinary incontinence, micturition disorders, bowel incontinence, radicular pain (neuralgia), loss of sensibility, muscle weakness or paresis (partial paralysis), dysesthesias (abnormal sensations experienced in the absence of stimulation), paresthesias (burning, pricking sensations) of thigh or foot, retrograde ejaculation, and “impotence”, a description based on case reports and a limited sample size [13 -15]. Tarlov cysts smaller than 1.5cm diameter have been reported as asymptomatic. It seems plausible that despite differences in terminology, there is a degree of congruence between these two sets of symptomatology.

Characteristics of Tarlov Cysts: From the descriptions in the literature, Tarlov cysts evidently develop at the distal limits of the dural sheath, a thinned extension of the dura

mater, which encapsulates not only the brain and spinal cord, but also the dorsal and ventral nerve roots. The thinned dural sheath, which includes the epineurium, terminates just at, or distal to, the dorsal root ganglia. At that point, the perineurium, which is the distal continuation of the pia/arachnoid, balloons out under pressure of the cerebrospinal fluid (CSF), perhaps in part because of the absence of constraint by the proximally-overlying dura mater. The perineurial wall contains spinal nerve root fibers and ganglion cells [1, 16] and the fibers may be present in the cyst cavity itself [16]. It is described that the neck of this fluid-filled balloon structure is constricted, creating a one-way “ball-valve” structure that allows CSF to enter, but not leave readily. As the volume of CSF changes in response to postural and positional changes, e.g., from supine to sitting, or in response to the Valsalva maneuver, sensory nerve root fibers in the cyst wall can stretch or compress against adjacent bone or the nerve roots, thereby producing abnormal sensations [16]. As pointed out by Acosta et al. [16] “...a great deal of confusion continues to exist over the precise definition of Tarlov cysts and their distinction from other spinal cysts. Tarlov cysts have been referred to as perineurial cysts, nerve root diverticula, meningeal cysts, sacral cysts, arachnoid cysts, and arachnoid pouches.” For the present purpose, we refer to them as Tarlov or sacral cysts.

Present hypothesis: Tarlov cysts can generate PGAD symptoms: Missing from all the above considerations is that at least some cases of PGAD could be due to pathology of the dorsal spinal roots, which could generate abnormal, distressing genital sensations. Therapeutic procedures focused on pelvic organs or their innervation, e.g., clitoridectomy [17] or surgical relief of nerve entrapment or pudendal or pelvic nerve block [9, 18] would not provide relief if the symptoms are generated proximal to these interventions, e.g., at the dorsal spinal nerve roots near their entry to the spinal cord. That is, even if the clitoris is removed or anesthetic is administered as a nerve block to the distal portion of the nerve, direct Tarlov cyst-produced mechanical stimulation of the (unanesthetized portion of) the nerve where it enters the spine could still generate afferent action potentials that would be perceived as if originating in the genital region.

Based upon the above considerations, we hypothesized that Tarlov cysts can generate PGAD. We tested the hypothesis by contacting a specific PGAD internet support group, (Ms. Jeannie Allen, pers. comm.) asking the women in the group to send us MRIs of their lower back that they may have had taken for diagnostic purposes; we then evaluated the MRIs for existence of Tarlov cysts. In the general population, the incidence of Tarlov cysts has been reported as between 1.2% and 9.0%, based upon incidental findings of the cysts in thousands of cases and in a variety of conditions for which lumbosacral MRIs were obtained [2, 16, 18].

Methods

Approval was obtained from the Institutional Review Boards for the Protection of Human Subjects in Research of Rutgers University and the University of Medicine and Dentistry of New Jersey (Newark) for evaluating existing MRI records on a confidential basis, and each of the women gave written permission for publication of their MRI images to be made anonymously. We requested of women in the Internet PGAD support group hosted by Ms. Jeannie Allen to send us, postage prepaid, diagnostic MRIs that they had obtained of their sacral spinal region. Of the more than 300 women in the internet support group, we received 18 such MRIs, each of which we evaluated for the presence or absence of Tarlov cysts, which occur predominantly at the S2-S3 levels of the spine, and because they are filled with cerebrospinal fluid, appear bright on T2-weighted images and dark on T1-weighted images.

Results

Of the 18 sets of MRIs of the spinal region that we received from women suffering from PGAD, Tarlov cysts were clearly evident in 12 (i.e., 66.7%) (Figures 1 and 2). The MRIs of five women show one cyst, those of six women show two cysts, and those of one woman shows three cysts. Consistent with the classical location of Tarlov cysts, all of the 20 cysts in the 12 women were located at the S2 and/or S3 spinal levels; two of which (Figures a and j) were at the S1 and/or S2 level. The spheroidal-shaped cysts ranged in maximum diameter from 3mm to 20mm with a group mean maximum

diameter of 9.6mm +/- 5.1 (s.d). In the 13th woman, no Tarlov cyst was evident, but she had a spondylolisthesis, i.e., the L5 vertebra slid forward over the S1 vertebra by 25%, distorting the spinal nerve roots of the cauda equina. In the 14th woman, there was a severe stenosis at L2-3 compressing the cauda equina. No pathology was evident in the MRIs of the other 4 women.

Discussion

Limitations of the present methodology

We recognize that the validity of conclusions based on the present findings is tempered by the unconventional means by which the data were obtained. There was a self-selection process in which of more than 300 women in the PGAD support group who were notified via the internet of our request for MRI images of the lower back, only 18 women sent us their MRIs that included images of their sacral spinal region. Thus, we could not evaluate the MRI images by a blind procedure. We have no formal control group, but rather for comparison with our “convenience sample” of 18 women manifesting a 66.7% incidence of Tarlov cysts, we rely on the literature that reports a much lower incidence of Tarlov cysts (1.2%-9%) in the general population of thousands of patients with MRIs analyzed for lower back problems. As to whether small sacral cysts might have been overlooked in those studies, a coauthor of the present study (H-JL) was also a coauthor on one of the above-mentioned studies [18] and counted even small sacral cysts, finding only 7 cysts among 584 MRIs (1.2%). Despite the above limitations, we believe that the present findings indicate a sufficiently plausible etiological basis for PGAD to warrant further investigation.

PGAD not previously linked to Tarlov Cysts: A review of the literature on Tarlov cysts reveals no mention whatsoever of PGAD, despite multiple papers published on properties and surgical treatment of the cysts since 2001, when Leiblum and Nathan first characterized PGAD. On the other hand, a review of the literature on PGAD reveals no mention whatsoever of Tarlov cysts, even though physiological, as well as psychological, etiologies have been proposed.

Although multiple clinical reports claim that Tarlov cysts are asymptomatic unless they are at least 1.5cm in diameter, perhaps a less stringent criterion is required in the case of PGAD symptoms, which may be paresthetic rather than painful, and because of the potential under-reporting factor related to social factors. Leiblum et al [10] pointed out that PGAD patients often feel ashamed and embarrassed about revealing their symptoms. Embarrassment has been emphasized as a major factor in underreporting of sexual dysfunctions [19]. Tarlov cysts are frequently reported as just incidental findings, for they can be asymptomatic. However, if they produce PGAD, women may under-report the symptoms due to embarrassment, thus rendering the cysts only apparently asymptomatic. This could account, at least in part, for the lack of previous complaints of women diagnosed with Tarlov cysts reporting symptoms of PGAD, by contrast with the current finding that women who report PGAD symptoms have a relatively high incidence of previously undiagnosed Tarlov cysts. Thus, it should not be assumed, a priori, that a Tarlov cyst less than 1.5cm diameter would be too small to generate PGAD symptoms. At present, we cannot relate size of the cysts to individual women's symptoms because we have only reviewed existing MRI records. Future research will evaluate the possible relationship between specific spectra of PGAD symptoms and the presence/size of Tarlov cysts.

Hypotheses of PGAD etiology: Leiblum [6] postulated that there may be two subtypes of PGAD: one more closely related to physiological factors (e.g., neurovascular or neurochemical), and the other to psychological factors. In the literature review by Leiblum et al. [7] the authors concluded that there are multiple hypotheses, but no consensus, regarding the etiology of PGAD. They summarized the hypotheses as: central neurological changes (e.g., post-surgical, post-injury brain lesion, seizure disorder), peripheral neurological changes (e.g., pelvic nerve hypersensitivity or entrapment), vascular changes (e.g., pelvic congestion or dilatation, or vascular pathology associated with chronic fatigue syndrome [20], mechanical pressure against genital structures, medication-induced changes (e.g. upon either initiation or cessation of SSRI or mood stabilizer therapy), psychological factors, (e.g., severe stress, the

perception and persistence of symptoms, anxiety and obsessive vigilance about physical symptoms, overall lower levels of sexual satisfaction, lower desire and greater pain), beginning menopause, physical inactivity [7], some combination of these, and/or idiopathic factors.

Other types of physical etiology were suggested by Waldinger and Schweitzer [17]; they observed a close association of PGAD with “restless legs syndrome (RLS)”, and characterized PGAD as “...the expression of a nonsexually driven hyperexcitability of the genitals and subsequent attempts to overcome it by genital manipulations”, the latter of which are an “...imperative urge to suppress dysesthesias and paresthesias.”

Waldinger et al [21] related PGAD-like symptoms in two men to genital hypersensitivity, which they attributed to “small fiber sensory neuropathy” of the pudendal nerve. In light of the possible involvement of Tarlov cysts in producing PGAD symptoms, and the fact that the S1 and S2 spinal nerve roots control the posterior dermatomes of leg sensation and movement (e.g., [22]), the possibility should be considered that Tarlov cyst-induced irritation of the spinal nerve roots S1 and S2 could interact with, and contribute, at least in part, to the cerebral component of RLS. In a followup study, we plan to ascertain whether specific symptoms of PGAD, e.g., RLS, are correlated with the existence or absence of Tarlov cysts, at least in the women who comprise the sample in the current analysis.

Goldstein et al. [9] observed that “...persistent sexual arousal syndrome has much in common with stuttering or recurrent [clitoral] priapism” in women with idiopathic symptoms of the syndrome, and hypothesized an “unusual genital tissue biochemistry” as the etiology. But ultimately, they suggested that most patients with “persistent sexual arousal syndrome” should be classified into the category “idiopathic” because, “...for the most part, clearly recognized causes for the syndrome are limited” [9]. And in their own survey of 76 women who fulfilled all the criteria of PGAD, Leiblum et al [7] found “no evidence of specific major medical illness or pharmacological agents effects associated with the report of PGAD.” Thus, there appears to be no known unitary etiology for PGAD.

Current PGAD therapies: In a comprehensive literature review and recommendations for management of PGAD, Goldmeier et al [11, 23] emphasized taking: a) a psychiatric history, b) a full medical history including medications, c) a comprehensive genital and pelvic examination, and d) a pelvic ultrasound. They emphasized this approach in order to exclude local pathology, such as genital dermatosis and genital prolapse, and to exclude conditions such as pelvic or cerebral pathology. Regarding the latter, a speculation of a neurological epileptic basis for PGAD [9] was followed by a finding that epileptic activity was temporally associated with a patient's symptoms, and anti-epileptic treatment suppressed both her orgasmic seizures and symptoms of PGAD [24]. Using a different approach, repeated sessions of electroconvulsive shock were reported to ameliorate PGAD symptoms [25]. Waldinger et al [26] reported, in two clinical cases, good therapeutic effects against PGAD and restless legs symptoms by using TENS stimulation. Perhaps in Waldinger's two cases, the effectiveness against the symptoms was due to an effect originating at the same sacral region as that affected pathologically by Tarlov cysts.

Other therapeutic PGAD interventions have focused on symptom management. "Psychoeducation" and social support impress on the patients that they are not alone in their experience with PGAD. Patients were trained to distract themselves from focusing on their genital sensations and to become aware of the triggers that worsen symptoms. Cognitive-behavioral interventions have been used to enhance coping skills and assist in interrupting the cycle of anxiety and catastrophizing of the symptoms [7]. Anesthetizing agents have been applied to numb the genital area. Neither clitoridectomy [27], local anesthesia, nor peripheral pudendal nerve block would be expected to alleviate PGAD symptoms if the symptoms were generated proximally at the spinal level, e.g., by Tarlov cysts. Pelvic massage or stretching exercises provided by a specialist in pelvic floor physical therapy have been applied to reduce or eliminate the pelvic tension and break up connective tissue strictures that might contribute to the condition [6, 8]. Medication has been applied by trial and error, as certain medications may be associated paradoxically with either alleviation or activation of the symptoms.

Mood stabilizing, antiseizure medications such as valproic acid, or SNRIs (Selective Norepinephrine Reuptake Inhibitors) have been tried [6, 8]. In light of the current findings, it would seem advisable to add a diagnostic sacral spinal MRI to the PGAD therapy armamentarium. To our knowledge there are no reports in the literature of amelioration of PGAD symptoms by treatment of Tarlov cysts.

Potential value of MRI to test for presence of Tarlov Cysts in cases of PGAD: The present finding that 66.7% of the 18 women with PGAD who sent us their spinal MRIs have one or more Tarlov cysts in their sacral spine far exceeds the population average, which has been reported to range from 1.2% to 9% [2, 6, 18, 28]. Langdown et al [2] found that 54 of 3,535 (1.5%) patients showed Tarlov cysts in MRI scans for lumbosacral symptoms; 70% were women. Similarly, Oaklander et al. [28] reported that 1.8% of 1,305 lumbosacral scans showed Tarlov cysts, of which 75% were in women. In an international survey of persons with Tarlov cysts, women outnumbered men by 9:1; the predominant age of all respondents was 40-60 [29, 30]. While PGAD is likely to have a variety of etiologies, the prevalence of Tarlov cysts in the present population indicates that in the absence of any other identified pathology related to a woman's PGAD symptoms, it would seem reasonable and advisable to test whether a Tarlov cyst is generating the PGAD symptoms.

Therapy for Tarlov Cysts

Probably the most convincing evidence as to whether a Tarlov cyst is generating the PGAD symptoms would be administration of an epidural anesthetic block at the sacral spinal level, which, if the Tarlov cyst is responsible for the PGAD symptoms, should eliminate the symptoms for the brief duration of the block, with return of the symptoms thereafter. If the PGAD symptoms are reversibly blocked by this epidural procedure, there are a number of long-term surgical procedures that could then be applied, although variable success has been claimed for surgical treatment of Tarlov cysts. As the most recent example, Murphy et al [31] surgically treated symptomatic Tarlov cysts by CT fluoroscopic-guided needle aspiration of the cerebrospinal fluid, followed by fibrin "glue" injection; they claim they have not had any case of aseptic meningitis as a

potential side effect of this procedure [31]. Other techniques for treating Tarlov cysts include subarachnoid drain, cyst fenestration and imbrication, muscle graft over the fenestrated cyst, cyst wall resection, and closure with myocutaneous flap reinforcement to prevent cyst recurrence or CSF leakage. A more drastic procedure is resection of the cyst and adjacent nerve roots, but this can result in neurological deficit [16]. For a concise summary of surgical treatments of Tarlov cysts, see Young [32]. A relatively recent procedure used in chronic pain patients is intrathecal catheterization with an implanted pump whose dose and timing of medication delivery is controlled by the patient [30]. This procedure has apparently not been reported to be used in the case of Tarlov cysts, but it would appear to be feasible if the cyst is situated superior to the filum terminale. There are risks associated with the procedure, e.g., movement of the catheter, CSF leakage, infection, etc. [30].

Characteristics of Tarlov Cysts and other spinal pathologies: Tarlov cysts are just one of several types of spinal cysts. While it is likely that the pathologies in the MRIs in Figures 1 and 2 are indeed Tarlov cysts, other, intraoperative and/or histological procedures, are necessary for differentiation of the several types of cyst. For example, Tarlov cysts show delayed filling on myelography because of their constricted, valve-like neck, which renders them non-compressible when tested intraoperatively [16]. They develop between the endoneurium and perineurium at, or distal to, the dorsal root ganglion. Their lining contains nerve fibers and /or ganglion cells. By contrast, meningeal diverticula freely communicate with the subarachnoid space, are situated proximal to the dorsal root ganglion, and possess a lining composed of arachnoid mater and dura mater devoid of neural tissue [16]. Histopathological examination of 8 specimens of Tarlov cysts demonstrated nerve fibers in 75%, ganglion cells in 25%, and evidence of old hemorrhage in 50% [13].

It is important to note that Tarlov cysts can occur in association with other spinal degenerative pathologies that could produce the symptoms, such as intervertebral disc prolapse or a stenosis that compresses the nerve root, sacral bone erosion which can produce an “insufficiency fracture,” spondylolisthesis (vertebral displacement), etc. [2].

Etiology and mechanism of action of Tarlov Cysts: No definitive etiology or mechanism of action has been established for Tarlov cysts; it has been suggested that they form as a result of congenital weakness of the meninges (perhaps more fundamentally related to connective tissue abnormalities) combined with subsequent acute physical trauma [33]. Trauma to the sacrum in the form of a skiing or motor vehicle accident, or heavy lifting or straining, were reported in the history of 5 of 10 patients who developed Tarlov cysts [13]. Perhaps the combination of the congenital and physical trauma factors can create a torsion defect specifically in the unique “weak-spot” just where the distal extension of the dura mater, i.e., the dural sheath, ends at the dorsal root ganglion, thus allowing the Tarlov cyst to form.

We are not aware of any direct evidence as to the mechanism(s) by which Tarlov cysts produce their symptoms. It would seem plausible that their sensory and/or motor (including autonomic, thus perhaps circulatory and/or vascular) effects could be produced by the mechanical irritation of sensory and/or motor nerve roots and/or the ganglion cells in the wall and/or the interior of the cyst, and/or by the cyst wall exerting force on the surrounding nerve roots, bone, or other tissue.

Conclusion: The lack of clear understanding of the etiology and mechanisms underlying PGAD has prevented the application of a rationale-based therapy. While PGAD may well be a complex pathology with multiple etiologies, the present findings provide a clue as to a plausible organic pathology – sacral (Tarlov) cysts -- that has evidently not been reported in the literature previously. It is too early to know whether, and if so which, symptoms of PGAD can be accounted for by these aberrant nerve fiber-containing cysts on the genital and leg sensory nerve roots in the sacral spine, but in the present study, the unusually high (66.7%) incidence of the cysts in women suffering from PGAD, much higher than the incidence of these cysts in the general population (up to 9%), strongly suggests that it would be worthwhile to test their possible role. In seeking the etiology and treatment for PGAD for a sufferer, it would seem advisable to test for – at the very least to rule out -- Tarlov cysts.

Figure Legend

Figures 1 & 2: MRI images submitted by 12 different women (a-l) showing evidence of sacral (Tarlov) cysts, out of 18 women with PGAD symptoms who submitted their sacral MRIs to us. All the images except (g) are T2-weighted; T1-weighted images are (also in some cases) shown in (g, h, i, and k). The arrows point to the cysts: Image a) sagittal view of three different cysts at the S1 and S2 levels ; b) sagittal and transaxial views of two different cysts at the S2 and S3 levels; c) sagittal and coronal views of one cyst at the lower S2 level; d) sagittal and transaxial views of one cyst at the S2-S3 junction; e) sagittal and transaxial view of a double cyst at the S2 and S3 levels; f) sagittal and coronal views of a pair of cysts at the lower S2 and upper S3 levels; g) T1-weighted images of sagittal and coronal views of three cysts. The T2-weighted images that were submitted did not include these sacral levels, so are not shown. The first two panels on the left show one cyst at the S2-S3 junction. The three panels on the left show two additional cysts, one (left side) at lower S2 and the other (right side) at upper S3; h) sagittal T1 and T2-weighted images of the same cyst at S2 and a T2-weighted transaxial view of this cyst (right panel); i) T1- and T2-weighted images of a large cyst at S2 (upper panels) and transaxial T1- and T2-weighted images of this cyst (lower panels); j) axial T2-weighted image of a cyst at the S1-2 level; not visible in sagittal view k) T1- and T2-weighted images of a cyst at the S2 level (upper panels) and coronal S2 and “STIR” images of the two cysts in this woman (bottom panels); l) sagittal T2-weighted image of a cyst at the S2 level.

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