



Address: 1 Kraljice Natalije Street, Belgrade 11000, Serbia ### +381 11 4092 776, Fax: +381 11 3348 653
E-mail: office@srpskiarhiv.rs, Web address: www.srpskiarhiv.rs

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## Case Report / Приказ болесника

Dušan Marić<sup>1,2,†</sup>, Vukadin Milankov<sup>1,2</sup>, Ivica Lalić<sup>2,3</sup>, Marko Bumbaširević<sup>4,5</sup>, Džihan Abazović<sup>6</sup>

# Calcification of cervical intervertebral disc in children – a case report and review of literature

Калцификација међупршљенског диска вратне кичме код деце – приказ случаја и преглед литературе

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### <sup>†</sup>Correspondence to:

Dušan MARIĆ

Institute for Children and Youth Health Care of Vojvodina, Hajduk Veljkova 10, 21000 Novi Sad, Serbia Email: dusan.maric@mf.uns.ac.rs

<sup>&</sup>lt;sup>1</sup>Institute for Children's and Youth Health Care of Vojvodina, Novi Sad, Serbia;

<sup>&</sup>lt;sup>2</sup>University of Novi Sad, Faculty of Medicine, Novi Sad, Serbia;

<sup>&</sup>lt;sup>3</sup>Clinical Centre of Vojvodina, Clinic for Orthopaedic Surgery and Traumatology, Novi Sad, Serbia;

<sup>&</sup>lt;sup>4</sup>University of Belgrade, Faculty of Medicine, Belgrade, Serbia;

<sup>&</sup>lt;sup>5</sup>Clinical Centre of Serbia, Clinic for Orthopaedic Surgery and Traumatology, Belgrade, Serbia;

<sup>&</sup>lt;sup>6</sup>Emergency Medicine Centre of Montenegro, Podgorica, Montenegro

# Calcification of cervical intervertebral disc in children – a case report and review of literature

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#### **SUMMARY**

**Introduction** We report a case about calcification of cervical intervertebral disk in children. This is a rare condition, and has been described in about 400 cases worldwide. Children affected by it present with onset of pain, muscle spasm and on radiography presence of calcification of intervertebral disk.

Our aim was to present a case of sudden onset of pain in the neck and torticollis.

Case outline In our case, condition was diagnosed after trauma, presented with neck pain and spasm of right sternocleidomastoid. Initial neck radiography was done, and after identifying the calcification in front of C4 and C5 vertebra body, CT analysis was conducted. When it was concluded that there is no compression on spine nerve roots, conservative course of treatment was followed. Child had full regression of symptoms after two weeks.

**Conclusion** Emergency personnel should bear in mind, even though radiographical finding of calcification shadow in front of the spine may raises concern, the nature of this disorder is benign in most cases, and responds excellent to conservative treatment.

**Keywords:** calcification; intervertebral disk; neck pain; torticollis

#### Сажетак

Увод Представљамо случај калцификације интервертебралног диска вратне кичме код деце. Ово ретко обољење је описано у око 400 случајева широм света. Оболела деца долазе код лекара са тегобама у виду болова у врату, мишићним спазмом и рентгенским снимком калцификације интервертебралног диска.

Наш циљ био је да прикажемо случај изненадне појаве бола у вратуи тортиколиса.

нашем Приказ болесника случају, пацијенткиња се јавила након трауме са тегобама у виду болова у врату и спазмом десног стерноклеидомастоидног мишића. Иницијално је направљен рентгенски снимак када је откривена калцификација испред Ц4и Ц5 вратног кичменог пршљена, а затим је начињен ЦТ снимак вратне кичме. Када је искључено постојање компресије коренова спиналних живаца, укључен конзервативни третман. Две недеље после дете је било без тегоба.

Закључак Иако постојање сенке са предње стране кичменог стуба изазива забринутост, доктори ургентне медицине треба да имају у виду да је ово обољење у већини случајева бенигне природе те одлично реагује на конзервативно лечење.

**Кључне речи**: калцификација; интервертебрални диск; вратни бол; тортиколис

### **INTRODUCTION**

Juvenile calcification of intervertebral disc is an infrequent condition, that is benign and self-limiting in character and it primarily affects nucleus pulposus. Luschka described in 1858 [1, 2] the first case of intervertebral disc calcification on anatomic dissection and Beneke demonstrated this condition first radiographically in 1897 [2, 3]. This disorder in children was first discovered by Baron in 1924 [4] and others in more than 400 cases to this day. Our aim was to present case of sudden onset of pain in the neck and torticollis.

### **CASE REPORT**

Patient, six year old girl came to the clinic because of sudden onset of pain in the neck and torticollis. After examining patient history we found that she fell from the bicycle two days earlier and was treated for sub-acute rhinitis for the last two weeks.

Clinical examination revealed tilting of the head to right side and upwards, consistent with muscle spasm of sternocleidomastoid muscles. There was also asymmetry of the shoulders and elevated tonus of posterior neck and shoulder muscles. Active range of motion for cervical part of the spine was significantly decreased: flexion 25°, extension 0°, right and left lateral flexion 10°, 60° right rotation, 40°left rotation. Postural dysfunction and thoracolumbar scoliosis was also noted. Pain was provoked by terminal movement and palpation of posterior side of the neck bilaterally. No motor neurological deficits were present and no sensomotoric neurological deficits were noted (Gross motor strength, Gross motor functions, Fine motor functions, Simple Sensory Skill test were performed)

Because of positive trauma heteroanamnesis, plain radiography of cervical spine was taken. We discovered some calcified lesion between C4 and C5 vertebrae, which we first attributed to trauma (Fig.1). Next day computerized tomography (CT) scan was done without the contrast (Fig.2). Results of analysis were: oval hyper dense lesion (density of calcium) in intervertebral space at the level of C4/C5 vertebrae, centrally positioned, approximately 5.5 x 5.5mm, with nearby punctiform calcifications. Oval lesion is situated in intervertebral disc, his anterior aspect and punctiform calcification is in lateral aspect of the disc. There was no penetration in spinal canal and no signs of compression on spinal nerves. Cervical part of spinal canal was consistent and with no pathological changes. There are signs of sclerosation of inferior plateau of C4 vertebral body and on superior plateau of C5 vertebral body.

Since there were no neurological symptoms and CT didn't show any pathologies related to spinal canal, magnetic resonance imaging (MRI) wasn't indicated.

Patient was treated conservatively, analgesics were prescribed, soft cervical collar (Schantz) was placed for seven days and after the initial management was referred to physiotherapist for further treatment. All symptoms were gone after two weeks.

## DISCUSSION

Juvenile calcification of intervertebral disc is uncommon childhood condition characterized by neck pain, torticollis and calcification of the intervertebral disk. Most of the patients were aged between 5 and 12 years, predominantly male (male:female=8:5), though some studies suggest different ration of male to female (1:1) [5,6]. These results should be taken carefully because studies were done on small series of patients [7].

Calcification could be found on any level of the spine and there may be multiple level involvements in 30% to 40% of cases [5,6,8-10]. Most affected part of the spine is cervical. The

nucleus pulposus and annulus fibrosus can be calcified, but in the majority of the cases it's the nucleus pulposus. When calcifications are found in cervical spine, its lower part is more frequently involved and almost all patients are symptomatic [7,11]. The onset is usually acute, and patients are often referred to emergency room for evaluation of possible more serious condition.

Muscle pain was the major symptom of the condition, and it is found in about 70% of patients, followed by sensorimotor disturbance (focal weakness, sensory loss, compressive myelopathy) and fever [7]. Dysphagia has been reported in patient with anterior disk protrusion [12-15] and is attributed to formation of retropharyngeal edema due to tissue irritation by herniated calcium salts and macrophage-mediated inflammatory reaction, similar to calcific tendinitis [15]. Our patient exhibited muscle pain, right torticollis and reactive thoracolumbar scoliosis. Fever in our patient was not detected, nor were sensorimotor disturbances.

Sonnabend et al [13] demonstrated that only disc herniation cannot explain the pain [16]. Some authors suggest that the clinical symptoms are related to inflammation process within the disc [17,18]. Muscle spasm could also be partly responsible for pain. Immobilization using head halter traction or cervical collar probably limits the loading of inflamed disc during the phase of acute inflammation and relieves the pain resulting from muscle spasm. Pain could be associated with a rise of intradiscal pressure, which partly explains the enlarged intervertebral space and adjacent decrease in vertebral body height [13,16] even though this was not the case with our patient.

Half of the patients, who underwent blood examination, exhibited abnormal levels of inflammatory indicators. Erythrocyte sedimentation rate was the most sensitive indicator, elevated in more than 90% of patients. In contrast, WBC count and CRP showed positive reactions in only about a third of patients. We found only one study in which thyroid function tests, parathyroid hormone level assay, serum calcium, serum phosphate, serum alkaline phosphatase, peripheral blood film, and urine complete examination were done, and all the result were within normal limits [5].

For determining the presence and to evaluate the progress of condition, frontal and lateral radiographs are sufficient. Computed tomography (CT) can confirm dense calcification, show edema and reveal an eventual herniation of nucleus pulposus, which could be found in up to 38% of patient [13], its migration into neural foramen and consequences on the spinal cord [19]. However, Ginalskiet al. considered in 1992 that CT represents unnecessary irradiation and should only be indicated when disk calcifications are associated with neurological symptoms [20]. MRI should be done to exclude root, spinal cord or vertebral artery compression [16]. In case of symptoms such are headache, syncope, vertigo, tinnitus, ataxia, dysarthria, visual disturbance, Horner's syndrome, vomiting or dysphagia should alert the physician about the possibility of vertebral artery insufficiency, possibly due to herniation of disk through foramen transversarium, some author recommend doing magnetic

resonance angiography (MRA)[16]. Additional investigations (CT, MRI and MRA) should be recommended only for patients with sensorimotor disturbances [8,21].

When calcification is clearly visible on plain films, in the form of oval, round shape that could be fragmented, the diagnosis is straightforward. However, if calcification of the discs has not yet developed the findings are more subtle consisting primarily of bulging of the involved intervertebral disc into the adjacent vertebral bodies [11]. On MRI study in patients with disc calcification, there is loss of signal on T1- and T2-weighted images. In some patients, loss of signal in an adjacent vertebral body without loss of vertebral height had been found [22, 23]. These changes could be evidence that would support the concept of the vertebral body sustaining the initial insult and that disc involvement may be secondary [22]. In our study, CT scan showed sclerosation of adjacent vertebral bodies without loss of height. This could be indicator of later stage of mentioned process.

There are several hypotheses, some focus on the changes in the disc as primary part of process, while others place vertebral body in the central role.

Signs of low-grade fever, leukocytosis, elevated erythrocyte sedimentation, mild pleocytosis and elevated protein in the cerebrospinal fluid support the theory of an inflammatory process during disc development or during the symptomatic interval of the disease [24]. Smith et al. [6] who performed anterior discectomy in a 12 year old boy at the C4/C5 level, demonstrated an inflammatory response of severe reactive fibroblastic proliferation associated with multinucleated foreign body type giant cells and pleomorphic histiocytes in the disc material, while the herniated fragment of the nucleus pulposus appeared to be relatively normal. Contrary to these reports, Garlach et al. [24] found neither evidence of inflammatory or reactive changes, nor neovascularization.

Swischuk et al. [11] focus on disruption of blood supply to the intervertebral disk with resulting disk swelling and necrosis. In children, the discs are supplied by small blood vessels through the cartilaginous vertebral end plates. However, approximately at the age of 8 vessels begin to obliterate. By 20 to 30 years, the blood vessels for the most part disappear. After this sequence of events, disc nutrition becomes nonvascular and nucleus pulposus starts getting nutrients by osmotic passage from the vertebral end plate. This way of transition occurs effortlessly and it appears that the process is relatively smooth. It's noted that during this transition, there is a tenuous vascular connection between the cartilaginous end plate of the vertebral body and the juxtaposed layer of the intervertebral disc. It is possible that in some patients during the critical period of nutrition transfer, some external factor or combination of could result in premature interruption of blood/nutrition to the disc/vertebral body complex. It may be that vertebral body becomes involved first, but in most cases escapes unharmed. It could also be that vertebral body involvement could lead to interruption of the tenuous blood/nutrition supply from vertebral body to the disc so disc would become relatively ischemic, swollen and even

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necrotic. Later, it would calcify. Because of swelling, disc would bulge peripherally or into the

vertebral body as a Schmorl's node. If the vertebral body is "softened" because of inflammation and

6

vasculitis, the disc could bulge in convex direction into the vertebral body [11].

Above mentioned mechanisms could be triggered by trauma, infection, inflammation or

vasculitis. Trauma is found in 7% to 30% of the cases [13] and respiratory infection is found in 15%

of patient history [13]. In our case, both of factors were present.

Juvenile calcification of intervertebral disc is benign and self-limiting. In most cases

conservative treatment is required. It should consist of analgesics, non-steroid anti-inflammatory

drugs, muscle relaxants, cervical collar and limitation of physical activity. Patient symptoms are

relieved between 5 days and three weeks in two thirds of patients and in six months in 95% of patients

[13, 15]. Rapid improvement in symptoms is most probably related to the natural pathophysiology of

JIDC [16]. On radiographs the calcification disappears within few months, although there were cases

where it persist longer [24].

Surgical treatment is rarely required. There are no guidelines for it. Nerve root or spinal cord

compression by calcified are not considered as absolute indication for surgical intervention [7].

Surgical decompression should be reserved only for cases with persistent neurological deficits or

progressive neurological deterioration following an inadequate course of conservative treatment

[16,24]. Anterior cervical discectomy with decompression of spinal cord is safe procedure with good

clinical results [24].

Juvenile intervertebral disk calcification is a rare disease. For emergency medicine practitioners

it's important to have this disease in mind. When found, it should be regarded as benign condition

with excellent outcome by means of conservative treatment. However, patient should be checked

more frequently for the progression of calcification and neurological symptoms. If there are signs of

nerve root and spinal cord compression with calcified disk, CT scan and MRI should be performed. If

neurological status deteriorates further, after attempted conservative treatment, surgery should be

considered.

Conflict of interest: None declared

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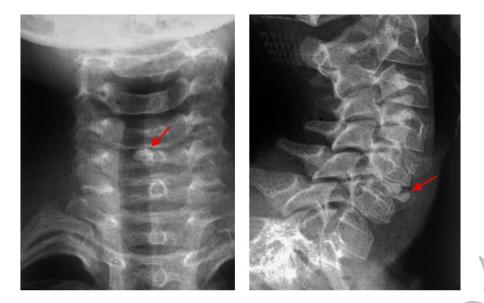
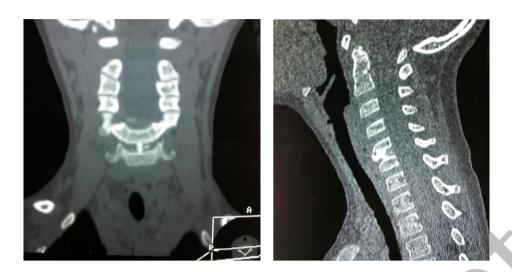


Figure 1. Radiography of cervical spine; calcified lesion between C4 and C5 vertebrae





**Figure 2.** Computerized tomography scan of cervical spine without contrast; calcified lesion between C4 and C5 vertebrae

