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Crowned Dens Syndrome: Recent Progress on Diagnosis and Treatment

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Abstract

Defined as an association of acute cervical pain and calcifications in the peri-odontoid space, crowned dens syndrome (CDS) is a major imaging manifestation of "coronary". CDS is a rare but under-recognized cause of severe neck pain in older adults. As such, it is often misdiagnosed. So, we review the literature with particular attention to the clinical and radiological aspects of this syndrome.

Keywords

Crowned Dens Syndrome, Radiology, Calcification, Computed Tomography, Nosogenesis

1. Introduction

In 1985, crowned dens syndrome (CDS) was first described by Bouvet *et al.* [1]. The pathogenesis is widely believed to be caused by crystal deposition disease [2] [3]. On radiographic images, calcium salt deposition around the odontoid is seen, mainly on the posterior side, like a crown on the odontoid process, from which it got the name. Clinical manifestations are mostly sudden neck pain and muscle joint stiffness, with limited neck movement. There are also chronic onset or acute-on-chronic onset [4] [5]. CDS often has a good prognosis [6]. In the foreign research [7], 2556 cases cervical CT plain scans were screened, among which 69 were diagnosed with calcification around odontoid process, demonstrating that this phenomenon was not rare. According to incomplete statistics,

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the incidence of CDS can be as high as 45% in the elder population over 85 years old [8]. However, due to little understanding of CDS, misdiagnosis or missed diagnosis often occurs in daily clinical work, causing unnecessary examination or treatment, and delaying the diagnosis and treatment of the disease. In this paper, the research status of CDS was discussed based on individual cases, theoretical works and reviews published at home and abroad in recent years, combined with actual clinical work.

2. Etiology and Pathogenesis

2.1. Crystal Deposition

At present, most scholars believe that crystal deposition may be the main cause of CDS, which is mainly related to the deposition of CPPD crystals (calcium pyrophosphate dihydrate deposition disease, CPPD) and HA crystals (hydroxyapatite, HA). Some scholars believe that CDS is caused by the abnormal deposition of bone salt caused by the abnormal activity of osteoblasts. However, in the CDS cases reported at home and abroad at present, the incidence of CDS caused by CPPD deposition is far more than that of HA deposition. Scutellari [3] *et al*. found through CT scan results calcification deposits in the check ligament, transverse atlas ligament, odontoid ligament and ligamentum flavum in 12 patients (3 males and 9 females), with horseshoe-shaped or coronal calcification around the odontoid process. Scutellari *et al*. suggested that the deposition of HA crystals could cause CDS, but it was rare and only middle-aged women seemed to be affected. H Cox *et al*. [9] performed CT scan on a 74-year-old male patient with sudden neck pain and limitation of motion, and found soft tissue calcification in the ligament around the atlas. There was also radiographic evidence of diffuse idiopathic skeletal hyperostosis (DISH). Kandel found an association between CPPD and DISH in his study of ape populations in the Barbary region of North Africa, but this phenomenon has not been confirmed in humans [10].

2.2. Gender and Age Factors

According to numerous case reports and relevant literature on CDS at home and abroad, its occurrence seems to be highly related to age and gender, and the higher the age, the higher the incidence, mostly occurring in elderly women over 60 years old [11]. Lu [7] *et al*. analyzed the CT of 2556 patients and found that 69 cases were diagnosed with calcification around odontoid process and 19 with CDS, most of which were elderly female patients over 65 years old. Okazaki *et al*. [12] reviewed 72 cases, including hospitalized patients and case data published on the Internet, and calculated that the average age of CDS was 71 years old in accordance with their data. Most of the patients admitted for acute neck pain and neck stiffness with activity limitation and finally diagnosed with CDS were elderly women [13]. Sano *et al*. [14] performed cranial CT scan on 577 patients with intracerebral hemorrhage or subarachnoid hemorrhage hospitalized

in neurosurgery department, and found calcification around odontoid process in 88 patients (15.9%), including 32 males and 56 females. The prevalence of odontoid calcification was higher in women than in men. Although patients undergoing cranial CT scans varied in age, CDS was mostly found in patients older than 60 years. At the same time, Goto *et al*. [15] found that CDS was more common in Asians, especially in elderly women, with a male-female ratio of about 0.6 and 2/3 CDS patients were older than 70 years old. Oak [12] believed that hormone levels were related to calcifications surrounding the odontoid process. Decreased estrogen production in postmenopausal women promoted bone resorption and an increase in intra-articular calcium, leading to an abnormal deposition of calcium salts and crystals. Mula and Malca also found that in CDS patients, the proportion of CPPD deposition was much higher than that of HA deposition. Meanwhile, the number of reported cases in males was much smaller than in females [16] [17].

2.3. Other Factors or Conjectures

At present, CDS is still in the stage of exploration and empiric therapy, and clinical researchers at home and abroad have put forward a series of conjectures or hypotheses. The current mainstream view, namely inflammation theory, believes that crystal deposition can cause aseptic inflammation while compressing and stimulating the C1 and C2 nerve roots. Acute and severe pain can be caused by neck muscle tension, muscle contraction or neck movement [18]. However, in the actual clinical work, it is undeniable that some patients have no typical symptoms of CDS such as pain or limited mobility, or even no symptoms at all. For them, CDS is detected by accident during the examination of other diseases or routine physical examination. Therefore, the simple inflammation theory cannot fully explain the etiology and symptoms of CDS. Studies have shown that high calcium, high phosphorus and low magnesium, joint wound, hypothyroidism, hyperlactinemia, hypertriglyceridemia, hemochromatosis, severe anemia, diabetes, cerebral infarction, intraparenchymal hemorrhage, subarachnoid hemorrhage, Wilson disease, postmenopausal syndrome, severe osteoporosis, myocardial infarction, systemic inflammatory response, or other major internal and surgical diseases may become high risk factors of CDS. Zhang *et al*. [19] pointed out that the occurrence and development of CDS might be related to diabetes with poor blood glucose control for many years. Nakano *et al*. [20] reported a unique case of CDS after endoscopic retrograde cholangiopancreatography (ERCP) in 2016, in which the patient mainly had a fever and neck pain. Considering that excessive mechanical load of neck movement may be a factor triggering CDS, it might be caused by joint injury resulting from limited cervical rotation when the patient was in prone position in the ERCP. Sano *et al*. [14] performed head CT scan on 577 patients with cerebral hemorrhage or subarachnoid hemorrhage hospitalized in the neurosurgery department, and calcification around odontoid process was found in 88 patients (15.9%). They were

more likely to get CDS than normal patients, indicating that severe systemic inflammation caused by critical illness could increase the incidence of CDS. Zunkeler *et al*. [21] assumed that systemic CPPD deposition was clinically associated with a variety of metabolites, including hyperphosphatemia and Wilson's disease. Zunkeler *et al*. also believed that factors such as age, genetics and various muscle stress imbalance might also promote CPPD crystal formation. Sugihara *et al*. [22] claimed that the occurrence and development of CDS might be associated with hyperuricemia. In 2000, Ho *et al*. [23] found ANK gene, meaning progressive ankylosis, in the study of mouse mutant strains with severe ankylosis of spine and other joints. The ANK gene was cloned to obtain a human homolog called ANKH gene. Preliminary studies show that ANK is a transporter of inorganic pyrophosphate (PPi), a potent inhibitor of hydroxyapatite mineralization. Therefore, it is speculated that the loss of ANK/ankmice translocator function leads to the loss of PPi transport function in the affected animals, thus resulting in excessive mineralization. Further biochemical studies of ANK function in frog oocytes performed by Gurleyet [24] showed that the ANK/ANK mutation (E440X) led to a significant decrease or even loss of PPi transport activity, which might be one of the causes of CDS. However, the specific mechanism of ANKH gene needs to be further studied.

3. Clinical Manifestations, Diagnosis, and Differential Diagnosis

Crowned dens syndrome is a special case of pseudogout that occurs in the atlantoaxial joint [25]. Its typical clinical manifestations include acute neck pain (100%), neck stiffness with confined movement (98%), and some cases may be accompanied by fever and chills (80.4%) and other symptoms [12]. There have also been reports of cases with chronic neck pain as the chief complaint and eventually diagnosed with CDS [4] [5], and such chronic cases are easily misdiagnosed as common diseases such as cervical spondylosis. Zhang *et al*. [19] reported a case of advanced CDS, for which the X-ray showed diffuse odontoid calcification and destructive intervertebral disc lesions and joint. In a few cases, aseptic inflammation will gradually erode the atlantoaxial ligament, and even form a cavity, leading to atlantoaxial instability, spinal cord compression and progressive tetraplegia, forming symptoms similar to cervical spondylosis, such as root symptoms of the upper limb, walking difficulties, stumbling gait, acragnosis and other symptoms [26].

Currently, CDS is mostly clinically diagnosed by CT, which is taken as the "golden standard". Studies have shown that the detection rate of CT in the diagnosis of CDS is as high as 97.1% [12], which can clearly show the high-density calcification shadow around the odontoid process, and assess the degree of its calcification. So its diagnosis is mainly based on the typical clinical manifestations and CT. Flurodeoxyglucose (18F-FDG) labeled with fluoro-18 is a new contrast agent based on radionuclide imaging technology, and its diagnostic

value for fever of unknown origin has received gradual attention in recent years [27]. Its diagnosis covers a wide range and can show aseptic inflammatory calcium deposition surrounding the odontoid process. However, 18F-FDG PET/CT is not the first choice due to its high cost, cumbersome operation and low popularity rate. Goto [16] summarized the possibility of CDS calcification occurring at different locations of the odontoid process in accordance with numerous radiographic data: posterior (50%), posterolateral (27.5%), annular (12.5%), anterior (5%) and lateral (5%). Since the calcification of CDS is more common in the rear of the odontoid process, it is difficult to clearly show the local calcification with X-ray in the diagnosis of CDS due to the overlapping radiography, so plain X-ray film examination and diagnosis is of little significance. MRI is better than CT in showing local inflammatory changes, and can determine whether there is nerve compression, but it is less sensitive to calcifications than CT [28]. A large number of actual clinical cases also suggest that X-ray and MRI are far less valuable in CDS diagnosis than CT. CDS cases showed elevated levels of serum inflammatory markers in laboratory tests, such as C-reactive protein (CRP), erythrocyte sedimentation rate (ESR), and white blood cell ratio [29]. The detection rate of serum inflammatory markers was about 88.3% [12], while procalcitonin did not increase. Some patients with CDS often have pseudogout gout [25] in the knee joint and hip joint, so the discovery of crystal deposition by puncture examination can also be considered as one of the diagnostic basis.

Although CDS is not a rare disease, it is prone to misdiagnosis and missed diagnosis because of the lack of understanding in practical work. It is necessary to distinguish it from some common diseases. The most important differential diagnose is meningitis, whose clinical manifestations are very similar to those of CDS, such as neck pain, stiffness, fever, etc. One of the important bases of meningitis is that MRI shows a nodular or thickened line-like enhancement [30]. Meningeal lesions should be taken into account when the length of meningeal enhancement is greater than 3 cm [31]. CDS did not show meningeal changes on imaging, and the deposition of CPPD crystals also helped to exclude meningitis. Cervical myofascial pain syndrome also has similar clinical manifestations as crowned dens syndrome, which can also be seen as elevated levels of serum inflammatory markers. However, this disease often results from cervical myofascial overwork or acute trauma, and cords can be felt in the affected area during a physical examination, which can be alleviated after manual massage [32]. The typical clinical manifestations of polymyalgia rheumatica (PMR) are stiff and painful neck, shoulder and hip muscles, and limited upper limb lift. Almost all patients (95%) have shoulder fatigue, and neck fatigue accounts for approximately 70% [33]. PMR can also be accompanied by low fever, tiredness, drowsiness, systemic muscle soreness, anorexia, weight loss and other non-specific systemic manifestations [34]. These are not found in the cases of CDS. CDS also requires differential diagnose with rheumatoid arthritis, gout, discitis, cervical spondylosis and neck muscle strain.

4. Current Treatment of CDS

At present, non-steroidal anti-inflammatory drugs (NSAIDs) or hormones are mainly used in the treatment of CDS, but which type is the first choice is still controversial. NSAIDs are recommended as first-line therapy in most cases reported in the literature, and symptoms usually improve within a few days after oral administration of NSAIDs. Oka *et al*. [13] believed that the use of NSAIDs could effectively and rapidly relieve pain and activity limitation and other symptoms. In 1995, Malca [18] *et al*. used diclofenac intramuscular injection treatment and achieved good analgesic effect.

Low-dose hormone (15 - 30 mg) was also recommended by some literature [35] as the first-line drug, which has achieved ideal curative effect. Therefore, it can be considered for patients who are intolerant to NSAIDs or who do not respond to NSAIDs treatment. CDS is more common in elderly patients, and their use of hormone drugs may lead to osteoporosis, femoral head necrosis, infection and other serious complications. Therefore, it is necessary to consider whether there are contraindications to hormone therapy in combination with the actual situation of the patients before careful medication, and long-term use is not recommended [36]. However, some studies have shown [37] that hormone drugs are better than NSAIDs in terms of efficacy, with which the symptomatic relief is more pronounced and lasting. Valnet suggested the combination of NSAIDs drugs and low-dose hormones as the best treatment for CDS [37].

Colchicine appears to be effective in some cases of refractory CDS, but its efficacy is less certain than that of NSAIDs or hormones. In addition, colchicine has slow onset and long treatment cycle, and may be accompanied by severe complications such as diarrhea, so it is not considered as the drug of choice. Risk factors for CPPD deposition, such as low magnesium, hyperparathyroidism, hyperglycemia and other complications should also be addressed.

The dialectical thinking of traditional Chinese medicine regards CDS as xiang bi disease (cervical spondylosis), which is caused by qi stagnation and blood stasis, and obstruction of meridians. Clearing wind-damp to relieve pain is required. Qianghuo Shengshi Soup (literal meaning of soup made of notopterygium root to dispel dampness) can be used as the prescription for clearing wind-damp, dredging the channels and collaterals and relieving pain [38].

5. Summary

To sum up, CDS is prone to misdiagnosis and missed diagnosis due to its unclear pathogenesis, relatively simple diagnosis method and less exposure at work. Ho *et al*. [23] reported the effect of ANK gene on crystal deposition in mice, so ANKH gene may be the key to the development of targeted drugs in the future. Patients with CDS often go to the emergency department because of acute neck pain, and its incidence can be as high as 45% in the elderly population over 85 years old. In most cases, CDS has a good prognosis, and the condition can be relieved within a few weeks after correct treatment, but attention should also be

paid to the possibility of protracted course or further aggravation of the disease. Therefore, early diagnosis and timely treatment are particularly significant to avoid unnecessary tests and treatment or delay of the illness.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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