



## Early onset idiopathic costochondral calcification can simulate costal fracture

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### A case report

Costochondral calcification (CC) is uncommon in people under 30 years of age. Age is not the sole factor for development of calcification. Premature costochondral calcification is associated with infections, mineral metabolism, thyroid disease, chronic renal failure, some malignancies and genetical factors. Sexual difference in the human costal cartilage patterns is predictive for sex determination. Radiolucent linear zones in CC may mimic fracture. In this report we present a 16-year-old female patient with CC. Costal cartilage calcification should be taken into consideration in patient with history of subcostal trauma.

Key words: cartilage calcification, costal fracture, trauma

### Introduction

Costochondral calcification (CC) is uncommon under 30 years of age and calcification of the costal cartilages follows gender-related patterns (1). In the lower ribs males tend to show calcification at the periphery of the cartilage, however females have central, tongue like calcification pattern. CC correlated with many pathological situations as arteriosclerosis, nutritional problems, metabolic or endocrine changes (2, 3). Often irregular outline with radiolucent transverse linear zones could simulate fracture. Idiopathic costal



cartilage calcification may occur in children as well as in adults, appears very dense, symmetric, and homogeneous lesions, but usually they have no pathologic significance (4).

In this article, we discussed a 16-year-old female patient diagnosed with Idiopathic costochondral cartilage calcification who referred to outpatient clinic with post traumatic subcostal region pain.

### **Case report**

16-year-old female patient, was admitted to our outpatient clinic with complaints of post traumatic pain in the lower thoracic region. These complaints were the last 2 weeks and gradually increased over time. She explained that a hard object hits 2 weeks ago. She expressed that the subcostal region pain increased with movement, reduced by rest. There was no pain at night. The past medical history was unremarkable. Her mother had type II diabetes mellitus. Physical examination of the lumbar region there was minimal scoliosis and paravertebral muscle spasm revealed. Subcostal regions was sensitive by pressure, there was no palpable swelling. Neurological examination revealed no abnormality. Laboratory tests including erythrocyte sedimentation rate, C reactive protein, hemogram, liver and kidney function tests, calcium, phosphorus, alkaline phosphatase, vitamin D and parathormone were normal. Conventional radiograph of the lumbar and thoracic region tenth, eleventh, twelfth ribs demonstrated calcified structure and consultation with specialists in radiology, was diagnosed with idiopathic CC (figure 1). Other possible pathologies were excluded with abdominal ultrasonography, and Chest x-ray.



Myorelaxant and nonsteroidal anti inflammatory drugs were administrated to patient. Cold application proposed fifteen minutes twice a day. After the treatment, the patient's pain reduced. It needs to give advice to the patient to be attention to his activities of daily living.

### **Discussion**

The costal cartilages extend medially from the anterior rib ends and articulate with the sternum or costal arch. These cartilages are bound to the sternal ends of the ribs by means of continuity of the periosteum of the bone and the perichondrium of the cartilage. The cartilages of the 11th and 12th ribs terminate in the abdominal wall (1).

Generally calcification in the remaining cartilages is uncommon in people under 30 years of age. Age is not the sole etiology for developing calcification (5). Investigators defined that heavy premature CC is associated with infections, mineral metabolism, nutrition, thyroid disease, chronic renal failure, some malignancies and genetical factors (6, 7). An increased incidence of CC in children with hyperthyroidism has been reported (3). However premature CC can also be seen in individuals without any medical problems as considered idiopathic. In this case, radiographies of the lower costal region, costal cartilages were detected calcified. Other laboratory investigations were evaluated as normal. The patient was diagnosed with idiopathic CC considering the presence of bilateral homogeneous calcification.

Some loss of thoracic cage compliance may occur and fractures can be identified in trauma, also radiolucent transverse linear zones that can simulate fracture (5). Radiolucent



transverse linear zones were seen in this case. We excluded the diagnosis of fracture due to bilateral and homogeneous appearance of calcification.

Diffuse enlargement with the calcification of costal cartilages occurs in acromegaly. Somatotropin hypersecretion may reactivate endochondral bone formation at existing cartilage-bone junctions and induce periosteal bone formation (1).

Many tumors elaborate proteins with parathyroid hormone like activity that alter calcium metabolism by increasing renal reabsorption of calcium and increasing bone resorption. The much higher incidence of early CC in thyrotoxic adolescents than in the general population (3). A crucial role of the thyroid hormones in normal skeletal development is linear growth and the maintenance of adult bone mass. In hyperthyroidism skeletal metabolism is altered. Childhood thyrotoxicosis results in accelerated growth, advanced bone age. The effect of increased bone resorption has been shown to be a direct action of thyroid hormone, also can increase bone formation. Due to the fact that cartilage calcification may be occur. Therefore changed calcium balance might account for the occurrence of premature calcification of the costal cartilages in chronic renal failure. Tietze syndrome is an inflammatory condition that affects the costal cartilages, also infectious costochondritis can occur due to the pulmonary infection or postoperative complication. In these situations radiological findings include chondral destruction, chondral enlargement and cartilage calcification (1, 8).

Rejtarova O. et al. (6, 7) confirmed that the sexual difference in the human costal cartilage patterns is statistically significant and highly predictive for sex determination. They have shown that finding a peripheral type of ossification determines male sex with a high



probability and finding a central lingual type of ossification determines female sex with a high probability. We present a case of 16-year-old female who had lingual pattern. CC also increases with age and continues throughout life, reflecting the aging process (3).

Treatment should be directed to the underlying cause of the disease. In this case we suggested that NSAID treatment and cold application for pain. Pain decreased significantly within two weeks.

In daily practice, costochondral calcification is often to escape review. Costochondral calcification can be associated with metabolic, inflammatory, infectious disease, chronic renal failure and some malignancies. As well as in this case, should be remembered that may confuse with rib fracture. Costochondral calcification should be taken into consideration in patient with a history of subcostal region trauma.

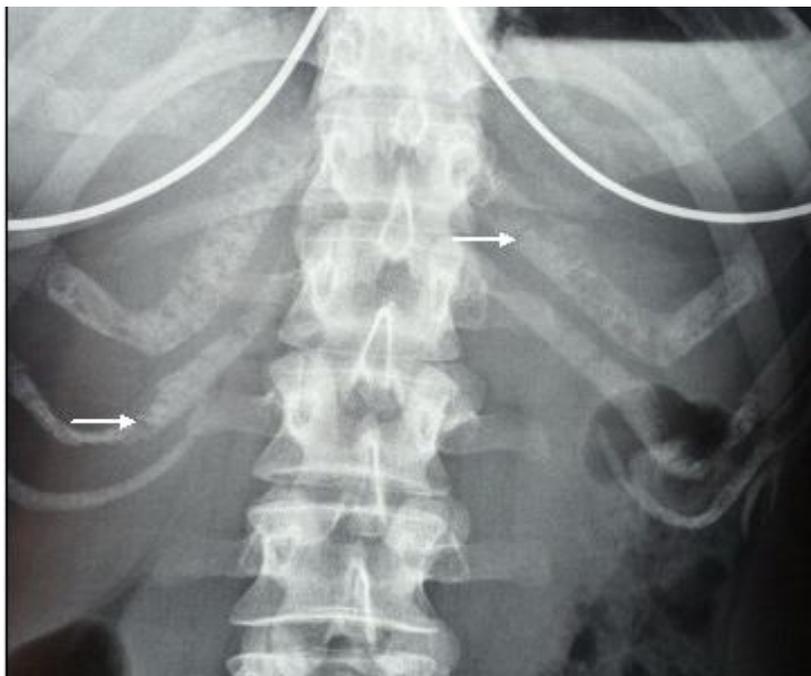


Figure 1: Linear radiolucent areas within the costochondral calcification.



Conflict of interest: All authors declared no conflicts of interest

### References

1. Ontell FK, Moore EH, Shepard JA, Shelton DK. The costal cartilages in health and disease. *Radiographics*. 1997;17:571-577.
2. Rejtarová O, Slízová D, Smoranc P, Rejtar P, Bukac J. Costal cartilages a clue for determination of sex. *Biomed Pap Med Fac Univ Palacky Olomouc Czech Repub*. 2004;148(2):241-243.
3. Senac MO Jr, Lee FA, Gilsanz V. Early costochondral calcification in adolescent hyperthyroidism. *Radiology*. 1985;156:375-377.
4. Rowe LJ, Yochum TR. Masqueraders of musculoskeletal disease. In: Yochum TR, Rowe LJ, editors. *Essentials of skeletal radiology*. 3rd ed Lippincott Williams & Wilkins. 2005:1766-1767.
5. Guebert GM, Rowe LJ, Yochum TR, Thompson JR, Maola CJ. Congenital anomalies and normal skeletal variants. In: Yochum TR, Rowe LJ, editors. *Essentials of skeletal radiology*. 3rd. ed. Lippincott Williams & Wilkins. 2005:321-325.
6. Rejtarová O, Hejna P, Rejtar P, Bukac J, Slízová D, Krs O. Sexual dimorphism of ossified costal cartilage. Radiograph scan study on Caucasian men and women, Czech population. *Forensic Sci Int*. 2009;191:110-115.
7. Rejtarová O, Hejna P, Soukup T, Kuchar M. Age and sexually dimorphic changes in costal cartilages. A preliminary microscopic study. *Forensic Sci Int*. 2009;193:72-78.
8. Bassett JH, Williams GR. The molecular actions of thyroid hormone in bone. *Trends Endocrinol Metab*. 2003;14:356-364.