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A CASE REPORT OF CRYPTOGENIC ORGANIZING PNEUMONIA

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ABSTRACT

Introduction: Cryptogenic organizing pneumonia (COP) is a rare respiratory condition characterized by the filling of alveolar airspaces with fibrin and granulation tissue, with no indetifiable cause. **Case Report:** This article presents a case of a 3-year-old child with COP and discusses the condition's clinical, histopathological and radiological features. **Discussion:** COP is diagnosed through biopsy and histology; and it is essential to rule out other potential causes. Our article outlines the pathogenesis of COP and its three stages of development. The primary treatment for COP is systemic corticosteroids, with a significant success rate. However, further research is required to explore more effective therapeutic methods, reducing the need for surgery and improving patient outcomes. **Conclusion:** COP is now well-characterized, but ongoing research is needed to understand its underlying mechanisms and optimize treatment approaches.

I- INTRODUCTION

Cryptogenic organizing pneumonia (COP) is a rare respiratory condition characterized by the filling of alveolar airspaces and alveolar ducts with fibrin and granulation tissue, and for which there is no identifiable cause.[1]

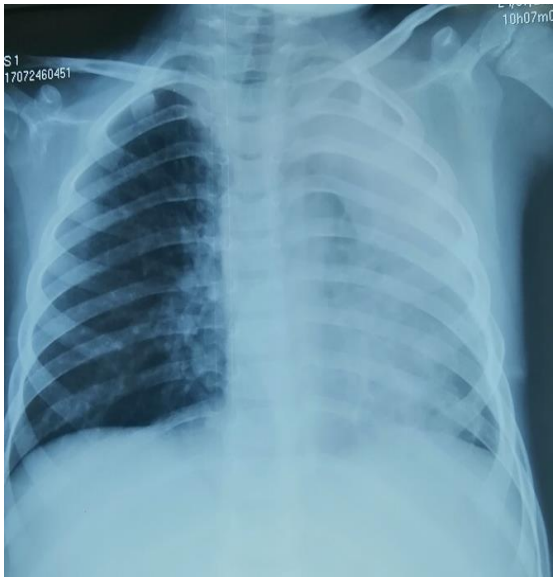
Because buds of granulation tissue are present within the lumen of distal airspaces including the bronchioles, COP had been formerly referred to as bronchiolitis obliterans with organizing pneumonia (BOOP), a nomenclature now abandoned, because OP (and not bronchiolitis) is clearly the major lesion of COP.[2]

Typical histopathologic features are patchy cellular airspace fibrosis involving alveoli and alveolar ducts, foamy alveolar macrophages, and fibroblasts embedded in a myxoid matrix.[3]

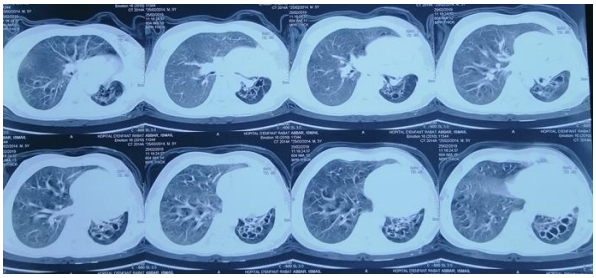
II- CASE PRESENTATION

A 3-year-old child was presented with repeated respiratory infections, more than two episodes per year, he was hospitalized in the pediatric department for a chronic cough with dyspnea and fever, he presented respiratory discomfort with left condensation syndrome.

The chest X-ray showed a homogeneous opacity occupying the entire left hemithorax with attraction of the trachea (atelectasis).

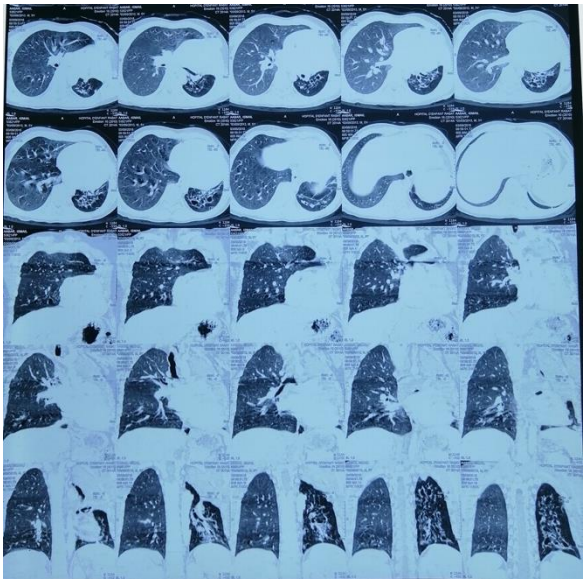


Chest computed tomography was performed and showed a cyst in the middle and posterior mediastinum under the arch of the aorta, completely compressing the left main bronchus resulting in non-aerated collapse of the left lung, with attraction of the heart and large mediastinal vessels to the left, concluding with a bronchogenic cyst responsible for a non-aerated collapse of the left lung by compression of the left mainstem bronchus.



The biological tests were normal

The patient benefited from a surgical operation, we started with a left anterolateral thoracotomy, musculo-aponeurotic dissection, opening of the pleura, and after exploration we found a shriveled left upper lung lobe, with bumpy contours, inflamed, adherent to the thoracic wall as well as to the pericardium and to the lower lobe, while the lower pulmonary lobe is healthy in appearance, then the patient benefits from a left lung lobectomy.



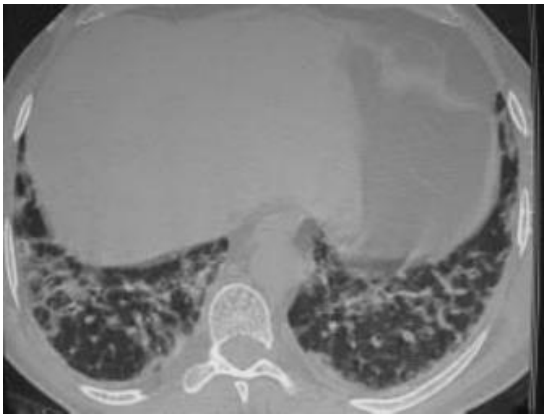
The histological study showed a pulmonary parenchyma whose alveoli are filled with foamy histiocytes and red blood cells. The surface epithelial lining is often abraded with widening of the interalveolar space. the terminal bronchioles show dilation lesions with abrasion and exulceration exposing the fibroedematous lamina propria which protrudes into the bronchiolar lumen producing a polypoid appearance obstructing its lumen, a reaction with multinucleated giant cells is associated with it. We concluded a cryptogenetic obstructive bronchopulmonary disease (COP) with macrophage alveolitis lesions.

III- DISCUSSION

Cryptogenic organizing pneumonia (COP) was first described in 1983 by Davison et al. in eight patients who presented with respiratory symptoms, bilateral radiologic manifestations, elevated inflammatory markers, and intra-alveolar organization but no identifiable etiology.[4]

The term cryptogenic organizing pneumonia is reserved for the primary entity in which no cause or association is recognized.[5]

Clinical manifestations of OP are non-specific. The typical presentation is of a subacute or chronic clinical course with fever, cough, weight loss and dyspnea, and less commonly with chest pain and hemoptysis[6], and sparse crackles on auscultation.



The three main characteristic imaging patterns of COP consist of multiple alveolar opacities (typical COP), solitary opacity (focal COP), and infiltrative opacities (infiltrative COP).[3]

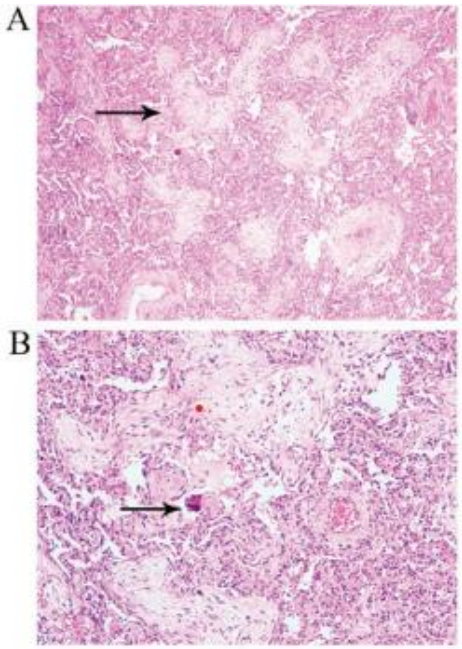
Symptoms with similar clinical and histologic features include infections, autoimmune diseases, connective tissue diseases, and drug injury among others. It is important to exclude all these causes to be able to arrive at the diagnosis of COP.

The diagnosis of OP is made by biopsy and histology. However, clinical and physical examination findings (including an investigation of possible causes), together with imaging changes, can suggest the diagnosis.[7]

The pathogenesis of the pathological lesions of COP has been reported schematically to go through three stages:[2,7]

- The Alveolar epithelial injury is the first event (injury phase), it is characterised by the formation of fibrinoid inflammatory cell clusters, with necrosis and sloughing of pneumocytes resulting in the denudation of the epithelial basal laminae
- The second stage (proliferating phase) is characterised by the formation of fibroinflammatory buds. Fibrin is fragmented by macrophages and inflammatory cells are present but less numerous. Fibroblasts migrate from the interstitium through gaps in the basal laminae, colonise the fibrin remnants, proliferate, differentiate into myofibroblasts, and form cell clusters within the distal airspaces
- The third stage (mature phase) is characterized by "mature" fibrotic buds. Inflammatory cells have

disappeared in most buds, and there is no longer any fibrin within the alveolar lumen, alveolar buds are now mostly constituted by typical myofibroblasts organized in concentric rings alternating with layers of collagen bundles.



So far, the most effective methods of treatment are systemic corticosteroid therapy and surgery.[8] In 2013, the American Thoracic Society/European Respiratory Society[9] recommended the following strategy of monitoring COP: Short-term observation to confirm wall response to the treatment, and long-term observation to ensure the preservation of the response. Therefore, long-term follow-up observation is often necessary. antibiotic therapy is not effective on COP.[10] Treatment with systemic corticosteroids typically gives rapid improvement in symptoms and has a cure rate superior than 65%, without significant sequelae.[6,11] Long-term administration of corticosteroids at high doses is the most effective therapy, and is gradually increased during a treatment period of 6-12 months.[12]

Conversely, improvement without treatment has been observed in some patients who have spontanously regressed.[13,14]

CONCLUSION

Although rare, COP is now a well characterized entity with characteristic clinical and radiological features and pathological diagnostic criteria. The main treatment until now is systemic corticosteroids which is often effective; however, it typically requires a lengthy treatment period and is accompanied by adverse side effects. Improvement of diagnostic techniques is required, and further research should be conducted to identify more effective therapeutic methods, in order to reduce or avoid trauma to patients from unnecessary surgeries. There may not be a single cause of COP but biopathological studies are needed to identify the mechanisms whereby a limited wound healing reaction switches to an idiopathic persistent inflammatory process which is nevertheless very responsive to corticosteroids.

ABBREVIATIONS

- COP: Cryptogenic organizing pneumonia
- BOOP: bronchiolitis obliterans with organizing pneumonia.

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