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BOOP following mitral valve replacement surgery (A case report)

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Abstract: *We report a patient because of rarity of presentation who developed hypoxaemia, interstitial pneumonitis both bases & atelectasis, after mitral valve replacement surgery developed as a result of bronchiolitis obliterans organizing pneumonia (BOOP).To the best of our knowledge such complication post cardiac surgery on mitral valve disease is unheard in the literature.*

Key words **:** *Cardiac valve replacement , BOOP*

I. Introduction :

Bronchiolitis obliterans organizing pneumonia (BOOP) is a distinct entity with various clinical , radiographic & histologic features.Bronchiolitis is a generic term describing a variety of inflammatory diseases affecting bronchioles with a common pathologic change.The term obliterans refers to the ability of such pathologic changes to obliterate or destroy the airways.The term Bronchiolitis obliterans organizing pneumonia was first described in early 1980s as a clinico pathologic syndrome characterized symptomatically by subacute & chronic respiratory illness & histopathologically by granulation tissue in the bronchiolar lumen, alveolar ducts & some alveoli associated with a variable degree of interstitial & air space infiltration by mononuclear cells & foamy macrophages[1,2] **.** In this condition airway lumens are occluded from within, with generally preserved airway architecture.The inflammation & the occlusive infiltrates in the bronchioles leads to restrictive rather obstructive physiologic changes.We report a patient with classic clinical , radiological & histopathological description of BOOP seen in post mitral valve replacement surgery.

II. Case Report :

A 35 yrs old woman presented with history of fever, dry cough, pleuritic chest pain & shortness of breath for 2 weeks. She remained symptomatic despite antibiotics & symptomatic management. 1 month prior to the present hospitalisation she underwent mitral valve replacement for her rheumatic heart disease, mitral regurgitation with 25mm St Jude Bileaflets mechanical valve sutured with 2/0 prolene. She was discharged on 12th day of surgery without any major problems.There was no any contributing factors in history to elucidate the etiology of her symptom complex. She denied history of allergies. As per medical advice she was on warfarin 5 mg OD & spironolactone.

Systemic examination revealed a young, alert & oriented female in mild respiratory distress with respiratory rate 28/min, with resting oxygen saturation of 85%.Her temperature was 100^0 F, heart rate 110/min regular,BP 110/70 mm of Hg.There was no cyanosis, jaundice , oedema or pallor. Lung auscultation revealed bilateral inspiratory crackles all over the chest predominantly at bases with decreased breath sounds at bases.The chest Xray film revealed reduced lung volumes, bilateral lower lobe infiltrates & few nodularities.CT chest showed ground glass opacities with triangular areas of consolidation with base along pleural surface & apex towards the hilum (FIG 1).There were small multiple nodularities in the postero basal segment of left lower lobe.The lung function test demonstrated moderate restriction.The arterial PaO2 while breathing at room air was 55 mm of Hg with an alveolar:arterial gradient of 20 mm Hg. Laboratory findings included Hb : 11.3 gm%,Tlc 30,000/cumm, Platelets 3.14 lac/cumm, INR : 2.19 on warfarin. There was no any evidence of renal or metabolic abnormalities., Saline induced sputum, blood & urine cultures were negative for micro organisms.She received furosemide , nasal oxygen, piperacillin tazobactum empirically for possible chest infections .On day 3rd although she was afebrile but her white cell count had increased to 37,520/cumm with neutrophils : 72%, Lymphocytes : 26% , monocytes : 1% & eosinophils : 1%.Patients chest condition remained same despite changing antibiotics. Bronchoalveolar lavage & Transbronchial biopsy with fibreoptic bronchoscopy was performed on day 10 of hospitalistion. No endobronchial lesions were seen. BAL smear revealed squammous cells, columnar cells & macrophages with foamy vacuolated cytoplasms , very few polymorphs were seen. Microbiological cultures of BAL were negative. The transbronchial biopsy also showed foamy histiocytes & inflammatory cells & ill defined granulomas , diagnosis consistent with BOOP. (FIG 2). The patient was instituted on a regimen of prednisolone 0.5mg/kg b.w/day with clarithromycin 500 mg BD for 2 months &

steriods for 3 months initially. 4 months later patient has shown significant improvement both clinically & on imaging.She is advised a prolong course of steroids & regular frequent INR check ups.

III. Discussion :

BOOP comprises a distinct part of the spectrum of infiltrative lung diseases. The pathogenesis of the disease is that of an inflammatory lung disease rather than a fibrosing process such UIP/IPF[3,4] **.** A proposed clinical classification of bronchiolitis obliterans includes multifactorial components :[5,2]
1) Idiopathic ;2) Rapidly progressive ;3) Focal nodular; 4) Multiple nodular; 5) Post infectious; 6) Drug related ; 7) Auto immune diseases; 8) Bone marrow transplantation ; 9) Lung transplantation; 10) Renal transplantation; 11) Radiotherapy; 12) Environment related 13) Miscellaneous – Conditions like lymphomas, biliary cirrhosis , inflammatory bowel disease , coronary artery bypass graft surgery. In many instances the cause of BOOP cannot be determined & further no link exists between types of BOOP except that all types lead to a final a common clinical pathway.Persons of all ages can be affected. 50 % of patients invariably present with influenza like illness.Persistent & non productive cough is also a common presenting symptom.Patients often have mild dyspnea & chest pain.Crackles are often almost present & wheeze usually absent despite obstructive nature of disease[6]. The main imaging patterns that suggest BOOP are 1) Alveolar & diffuse interstitial infiltrates observed bilaterally.The ground glass opacities are often peripheral in location with base at periphery[7,8,9] . 2) Multiple foci of consolidation or nodular lesions single or multiple often 5 to 6[10,11] **.** As many as 65 % of patients have peripheral lower lobe consolidation[12,13] **.** BAL as a diagnostic tool has been used effectively by many workers[2,9,10,14] **. In** the la**vage** percentage of lymphocytes are often 25 – 40 % of white blood cells.Foamy macrophages which are lipid laden are characteristically seen.The debris inside the macrophages gives the cells the appearance of bubbling foam[15] **.** The open lung biopsy is preferred over transbronchial biopsy for accurate diagnosis of BOOP. Well taken transbronchial biopsy showing all components may well be effective tool for the diagnosis[16] **. Beca**use BOOP is generally non infective infiltrative disease , antibiotics are not effective & hence not the part of the treatment regimen. However some authorities have used macrolides such as clarithromycin 250 mg BD for 2 months[17] **. The** main stay treatment for BOOP is long stay treatment of oral prednisolone with recommendations to provide 0.75 – 1 mg/kg/day for 3 months then 40 mg/day for 3 months , reducing the doses finally to 20 mg/day or alternate day for 6 months[2,9,10,18] . The prognosis is good with 65 % of patients having complete clinical & physiologic recovery & 30 % with varying degree of improvement[1] .In small subgroups of patients the disease follows a fatal course & in some, the disease recurs when the dose of steroids are reduced necessitating the continuation of the steroid regime further[18] . The present case has aptly demonstrated clinical, physiological, imaging & histopathological evidences as cited above. The pathogenetic mechanisms of development of BOOP entity in post cardiac surgery remains obscure. Guzman & associates in 2000 reported a case of BOOP occurring post coronary bypass surgery. Inter relationship of the entity with the associated disease needs evaluations. To the best of our knowledge, we have not found BOOP following cardiac surgery for mitral valve replacement.

IV. Conclusion :

The diagnosis of BOOP should be considered if no explanation remains evident for the cause of hypoxaemia, fever , breathlessness & pulmonary infiltrates observed post cardiac surgery. The clinicians should use all available tools in pursuing the diagnosis since effective treatment depends on accurate diagnosis.

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Legends to figures :

Fig 1 : HRCT chest shows multiple small nodular opacities in posterobasal segment of left lower lobe and interstitial pneumonitis with ground glass appearance at bases with base at periphery.

Fig 2 : H & E smear (1000 x) from bronchial lavage shows squamous cells , columnar cells ,. There are inflammatory cells with foamy histiocytes .

Fig 3 : Transbronchial biopsy H & E stains (1000 x) shows inflammatory cells with lipid laden foamy histiocytes with fibrous tissue having whorled arrangement with myxoid element . Ill defined granulomas seen.

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Fig 1 (A & B) :

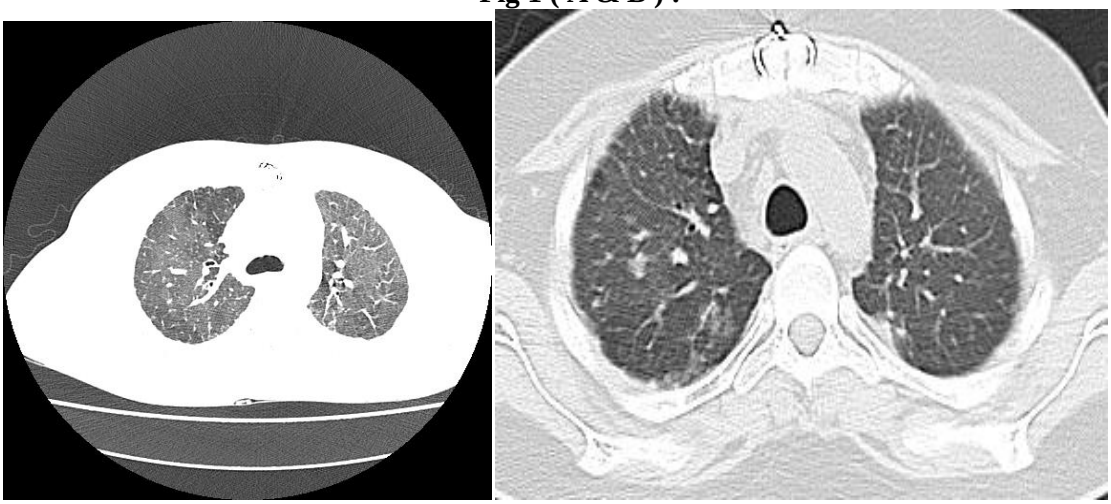

Fig 2 :

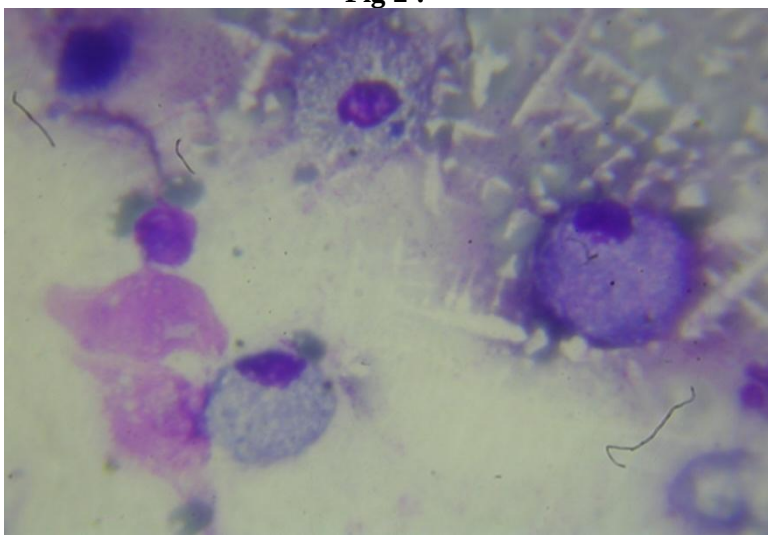

Fig 3 :