

# Evaluation of a Case of Fibrosing Mediastinitis Due to Histoplasmosis Appearing as Bright Mediastinum on MR STIR

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## Abstract

We describe a case of fibrosing mediastinitis causing superior vena cava syndrome due to histoplasmosis in a 51 year old woman evaluated by MR venography and inversion recovery MR. The mediastinum on inversion recovery MR was bright which is usually described in malignant, rather than benign causes of fibrosing mediastinitis

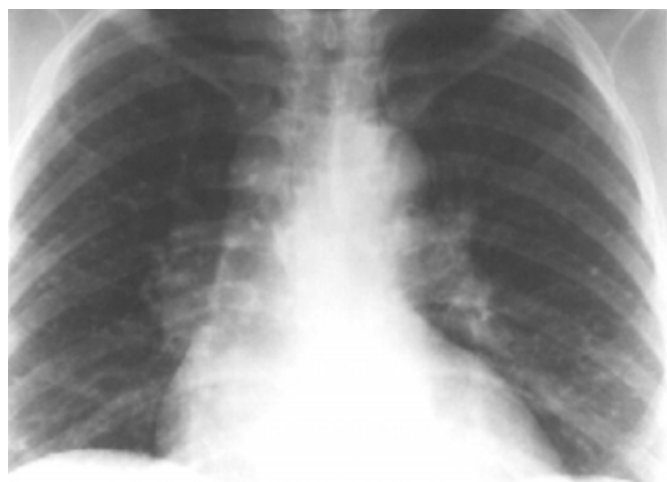
## BRIEF REPORT

A fifty-one year old caucasian woman with a history of; diabetes, hypertension, atypical angina, thrombocytopenia, and past histoplasmosis, presented with three weeks of swelling in her hands, face and upper extremities, as well as worsening cough. A chest roentgenogram taken one week later revealed multiple small pulmonary nodules with left hilar adenopathy. [Fig. 1] Infectious processes and superior vena cava obstruction were suspected in causing the above etiology.

axial time of flight venographic studies revealed fullness of the SVC, adenopathy, and high signal in the central mediastinum encompassing the paratracheal, subcarinal, and azygous regions [Fig 2]. Venous congestion and obstruction due to adenopathy were noted. Findings were consistent with superior vena cava syndrome. Fibrosing mediastinitis secondary to histoplasmosis was suggested as the cause in this patient with negative node biopsy and negative tuberculosis skin test but reactive to histoplasmosis antigen.

## Figure 1

Figure 1: 51 year-old woman with history of histoplasmosis, presenting with face and upper extremity swelling. Chest roentgenogram demonstrates miliary infiltrate.



MR evaluation (Philips Gyroscan ACS, 1.5T, Best, Netherlands) with coronal inversion recovery MR and with

**Figure 2**

Figure 2: Coronal MR STIR of mediastinum showing increase of signal in paratracheal, subcarinal, and aortopulmonary nodules without nodal enlargement in same 51 year-old woman.



**DISCUSSION**

Superior Vena Cava Syndrome (SVC syndrome) is usually caused by malignant lesions (78-93%); however, a minority (up to 22%) of cases are due to nonmalignant causes. These benign entities include various central venous lines and wires which have become an enlarging aspect of modern medical care, aortic aneurysms, radiation-induced changes, and fibrosing mediastinitis.<sup>(1)</sup> Symptoms of SVC syndrome, which include headaches and facial and upper extremity swelling, can frequently (39% in one study) be the initial presentation in patients with fibrosing mediastinitis. <sup>(2)</sup>

Fibrosing mediastinitis may result from tuberculosis or most commonly, histoplasmosis. <sup>(2,3)</sup> Histoplasmosis, while worldwide in distribution, is endemic to certain areas of North and Latin America, including Arkansas, where our patient contracted the disease. It can have a variable course in the acute phase; however, fibrosing mediastinitis is considered a late sequelae of the disease. Over time, mediastinal lymphadenopathy produced by the disease may form a coalescent mass which develops into granuloma. The granuloma may rupture, inciting a local inflammatory reaction in the surrounding tissue, which eventually leads to fibrosis. The fibrotic reaction then may encase the esophagus

or structures such as the SVC or pulmonary vessels, leading to obstruction and thrombosis.<sup>(2,4)</sup> A similar process has been described in cases of tuberculous mediastinitis.<sup>(5)</sup> Fungal tissue stains are often positive, while cultures are generally negative, suggesting that this form of disease is the result of prior, rather than active *Histoplasma* infection.<sup>(2,4)</sup>

The radiographic evaluation of fibrosing mediastinitis may involve multiple modalities during various phases of the workup. Initial chest roentgenogram may demonstrate mild subcarinal or superior mediastinal widening,<sup>(4)</sup> or more commonly, clearly show hilar and/or mediastinal lymphadenopathy, though plain film is generally inaccurate in assessing the extent of disease.<sup>(6)</sup> CT, which is superior for evaluation of this entity, may delineate the invasion and fibrotic restriction of mediastinal structures, with involvement of hilar, paratracheal, and subcarinal nodes.<sup>(4)</sup> CT may demonstrate one of two types of mediastinal masses which are typical of fibrosing mediastinitis. The majority have a well defined soft tissue mass,<sup>(2,7)</sup> frequently containing calcifications.<sup>(2)</sup> Less common is a diffuse infiltrative mediastinal mass,<sup>(2,4,7)</sup> possibly with calcifications.<sup>(7)</sup> The former type is most typical for histoplasmosis.<sup>(2)</sup> Variable enhancement of the masses has been described.<sup>(2,7)</sup> With contrast enhancement, SVC involvement can also be documented by CT.<sup>(7)</sup>

While CT has been the primary modality for evaluation of fibrosing mediastinitis, and CT findings have been well described, MRI has become a useful adjunct, providing complementary information about the disease. MRI is able to clearly delineate the relationship of the fibrosing process to the major vascular structures.<sup>(6)</sup> An area in which MRI has been thought to be useful is in the differentiation of benign from malignant adenopathy in the mediastinum.<sup>(6)</sup> Benign mediastinal fibrosing masses caused by histoplasmosis have been described as low signal intensity on T1W1 and low or heterogeneous signal on T2W1,<sup>(6,7)</sup> with heterogeneous enhancement after administration of Gd-DTPA.<sup>(7)</sup> Mediastinal nodal masses caused by tuberculous mediastinitis are described as iso-tense<sup>(8)</sup> with areas of low intensity after contrast administration.<sup>(5,8)</sup> These low-signal intensity areas in both types of benign disease are characteristic on MR of fibrous tissue,<sup>(5,6,7,8)</sup> and possibly calcification.<sup>(6)</sup> By contrast, malignant lymphadenopathy has been described as higher signal intensity in T1 and T2 weighted images.<sup>(6,8)</sup> This difference in signal intensity characteristics between benign and malignant lymph node

masses on T1 and T2 weighted images has been proposed as a criteria for differentiating these diseases on MR imaging.

In our patient, plain film and MR imaging demonstrated a number of the above findings, including the demonstration of infiltrate and adenopathy on chest roentgenogram, vena caval obstruction and adenopathy on the inversion recovery sequence. However, this case demonstrates an atypical appearance of a mediastinal lymph node mass in fibrosing mediastinitis due to histoplasmosis as bright signal intensity on T2 weighted MR. While the signal characteristics of mediastinal disease cannot be used conclusively as a determinant of benign-versus-malignant disease, benign disease is expected to produce hypo- or iso-intense T1 and T2 signal. The unexpected high MR signal intensity of the benign lymph node mass in this case demonstrates further that, though traditionally considered a sign of malignancy, bright mediastinum is not a reliable predictor of malignant disease. Further investigation into this appearance is warranted to determine the utility of MR signal intensity as predictor of benign-versus-malignant etiology is the diagnosis of fibrosing mediastinitis.

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