

## What's Causing These Yellow Nails?

Goslin BJ, Dasgupta RR

Michigan State University College of Human Medicine

Grand Rapids Campus

Yellow Nail Syndrome (YNS) is a rare disorder that presents as a triad of lymphedema, pleural effusions and slow growing, yellow nails.<sup>1,2</sup> Approximately 100 cases were cited from the time YNS was first described in 1964 until 1986.<sup>3</sup> While the classic triad of symptoms represents the most consistent findings, YNS has also been associated with chronic sinusitis, bronchiectasis, malignancy and autoimmune disorders such as lupus and most notably, rheumatoid arthritis (RA) which accounts for 24 of these cases, all of whom had the typical signs and symptoms of RA.<sup>4</sup> It has been suggested that YNS appears in patients as a result of RA drug therapy or other mitigating factors induced by RA.<sup>5</sup> Nails of patients with YNS are thick with ridging on the perimeter of the nail and onycholysis is often present.<sup>6</sup> The traits of YNS are well known while the etiology remains to elusive. Presently, altered lymphatic structure and function are highly suspected to be the basis of this disorder.<sup>4</sup> Regarding the transmission of YNS, most cases are sporadic, however, familial transmission has been documented.<sup>7</sup>

We present a case of Yellow Nail Syndrome in a 60-year-old Caucasian female with a one week history of shortness of breath and chest pain who presented to the emergency department. The onset of symptoms was insidious throughout the one-week period. Provoking factors included a chronic mild cough and sinusitis with sputum production. The patient denied previous symptoms of fever, chills, night sweats, PND, orthopnea, weight fluctuation, tuberculosis and a history of malignancy. The patient also had a waxing and waning history of extremity edema and a three-year history of yellow nails. Physical exam revealed vitals within normal limits, decreased O<sub>2</sub> saturation, decreased breath sounds in the left lower lobe and egophony. Drug therapy was started for probable pneumonia. Progressive imaging studies revealed a left inferior lobe pleural effusion with compressive atelectasis and mild bronchiectasis bilaterally at the bases, mediastinal and right hilar lymphadenopathy and a thickened lining of the sphenoid sinuses and ethmoid air cells. Concurrent lab data throughout the hospital admission included a total white cell count of 13,300/dL, marked elevation in CRP at 259mg/L, serum albumin at 2.9g/L, rheumatoid factor positive (517 U/mL), and citrulline antibody positive (57.1 U/mL). Metabolic panel and culture performed on pleural exudate was unremarkable with exception of a lymphocyte predominant exudate.

Our patient had the typical triad of YNS with lymphedema and pleural effusions, but this may be the first reported case of YNS in association with an elevated rheumatoid factor titer but without the signs and symptoms of RA. Our patient was never treated for RA, so the previous suggestion that YNS is associated with RA therapy seems less likely.

## References

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