Hey Clinical Problem Solvers! My name's T.J. La, a MS4 at Baylor College of Medicine and graduate of the University of Houston Law Center, and I am so excited to share with you more about anaplasmosis. Let's get started!

Human granulocytic anaplasmosis is caused by *Anaplasma phagocytophilum*, a gram negative, obligate intracellular organism. It tends to multiply in granulocytes within vacuoles that may form morulae that can be seen on a peripheral blood smear (hence the name, human *granulocytic* anaplasmosis)

In the U.S., its arthropod vector, the *Ixodes scapularis* (black-legged deer tick), is endemic in the Northeast, mid-Atlantic, and upper Midwest. On the West Coast, its vector is the western black-legged tick (*Ixodes pacificus*). Of note, the Northeast and the upper Midwest are the most endemic regions.

- It's important to note that because anaplasmosis has the same tick vector and endemic area as Lyme disease and babesiosis, ticks and the people they bite may be infected with more than one type of organisms at the same time.
- Also, there have been some reported cases of anaplasmosis after blood transfusions and transplants.

Anaplasmosis is more common than HME, otherwise known as human monocytic ehrlichiosis, which is an infectious disease also transmitted by ticks and causes similar symptoms. You should suspect either infection if a patient presents with a possible exposure in an endemic area from spring to fall seasons.

There is a high mortality rate in anaplasmosis patients who are taking immunosuppressants (eg corticosteroids, chemotherapy, long-term immunosuppressant treatment after solid organ transplantation), HIV infection, or splenectomy.

Although some anaplasmosis infections are asymptomatic, **most cause an acute onset of an influenza-like illness** with nonspecific symptoms including fever, myalgias, weakness, headache, malaise, and GI symptoms (eg, nausea, vomiting, diarrhea, anorexia) with symptom onset usually around 7 days after the tick bite (range 5-14d).

Lab tests commonly show hematologic and hepatic abnormalities including leukopenia that is characterized by relative and absolute lymphopenia and a left shift, thrombocytopenia, and mild to moderate elevations in aminotransferase levels.

Potential complications of anaplasmosis include DIC, ARDS, Bell's Palsy, brachial plexopathy, meningoencephalitis, seizures, and HLH.

If anaplasmosis is suspected, **treatment with doxycycline** should be started before lab results come back. For alternative treatment consider rifampin.

For diagnosis, detection of DNA by PCR of whole blood is most sensitive during the first week of illness.

- You can also do a blood smear to see if there are morulae in the cytoplasm of granulocytes, but blood smear examination is insensitive and should never be solely relied upon to rule anaplasmosis in or out.
- Antibody titers can also be checked, but are often negative in the first 7-10 days of illness. Although testing at the time of acute infection is often negative, you can check for a 4-fold rise in antibody titer from acute to convalescent samples or a single titer >1:256.

Differential diagnosis for HGA is extensive and can include HME, leptospirosis, RMSF, babesiosis, lyme, acute HIV, along with endocarditis, HLH, and TTP.

Overall, anaplasmosis is a tick-borne infection that usually results in an abrupt onset of an influenza-like illness. PCR testing of blood should be done when suspecting anaplasmosis and can result in an early diagnosis. For treatment, choose doxycycline which is best started before lab results are in.

We hoped you enjoyed this schema!