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ACUTE HIV-1 INFECTION

Antonio E. Urbina, MD

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[video transcript]

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Urbina in tuning Urbina is the medical director of the Mount Sinai Institute for Advanced medicines downtown clinic in New York City and medical director of C eyes New York State Department of Health AIDS Institute HIV Primary Care and Prevention Center of Excellence, as well as a professor of medicine at the Icahn School of Medicine at Mount Sinai. Since completing his residency in internal medicine at St. Vincent Catholic Medical Center, Manhattan in 1995, Dr Urbina has pioneered innovative educational programming for community based clinics, hospitals and public health departments. He has directed more than 10 HIV clinical trials research protocols, and from 2007 to 2009. Dr. Urbina served on the president Shoal Advisory Council on HIV AIDS, as well as Governor Cuomo his task force to end the AIDS epidemic in New York State. Thank you so much for being with us today, Dr. Urbina. And now I will turn the presentation over to you.

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Great. Thank you so much, Mark, for that introduction. And today I'm going to talk about acute HIV infection, I think a super important topic, in particular, how it relates to ending the epidemic. So I think it's very important that we're able to recognize the signs and symptoms of acute HIV infection. And this is really one of the times where I am very adamant about really starting treatment as soon as possible. And hopefully Today's presentation will explain why.

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All right, so these are my individual speaker disclosures. And our learning objectives are to define acute HIV infection, explain the impact of acute HIV on HIV transmission dynamics, and I have a couple of examples of those, describe the pathophysiology and clinical manifestations of acute HIV infection. And then review the algorithms that I use to diagnose and treat persons in acute HIV infection. So let's start off by asking, Why is diagnosing acute HIV infection really so vital. So the reasons are that persons in acute HIV infection are highly infectious, they have high titer viremia, in their plasma and in their genital fluids. So, for example, if we were to encounter a patient, and he reports that they'd say a recent sexual exposure that the source patient was in acute HIV infection, well, then that sexual exposure has a greater probability of leading to transmission. So persons in acute HIV infection because the infection is so early, there's an absence of immune factors that are that have time to kick in and neutralize the infection. And it's estimated that persons and acute HIV infections are anywhere from 100 to 3000 times more infectious than persons in chronic HIV infection. In fact, persons in acute HIV infection are at their most infectious during any time of their HIV infection. So here is a graph from a now older study by Pilcher, then at the University of North Carolina where he was able to identify persons in acute HIV infections who were absolutely able to date the time that they became exposed to HIV. And what they did is that they logged the viral loads logarithmically. And then also plotted against days from infection. And what they found is that you get this really increased peak in viremia, that peaks at about day 23. And then we have these immune factors that starts to kick in, and viral loads start to decrease gradually over time, but that peak viremia at day 23, is when

persons are eight to 10 fold increased risk from day 50. So again, high high rates of viremia that occurred just a little over two weeks from infection, and that's in the blood and plasma, but we also see that We have this peak kind of also in genital fluids, and same study. So in addition to doing phlebotomy, they also collected seminal samples and perform viral loads in the samples. And what they found is that a similar curve of peak viremia, but that the peak viremia occurred about seven to 10 days after viremia in the plasma, so about day 30. So, again, high plasma and genital secretions in persons in acute HIV infection. And that's why it's really felt that persons in acute HIV infection really disproportionately contribute to onward, or secondary HIV transmissions. And it's estimated that 29 to 50% of new HIV transmissions are attributable to persons in acute HIV infection, again, new infection, high amounts of virus in general and plasma. So that one chance encounter with either someone in their sexual or their needle sharing network is much more likely to lead to an HIV transmission event. And any infection that we can prevent is going to be highly cost effective, not just for the individual. Because we know that currently there is really no scalable cure for persons living with HIV. But treating HIV is also just hugely expensive. It's estimated that the lifetime cost of HIV care in the US in the current modern era of antiretroviral therapy, including the long acting injectables is about $1.5 million dollars. So that's estimated somebody diagnosed in their 20s, living into their 70s and 80s. And it factors in the cost of the antivirals, the cost of medical visits, laboratory testing, and hospitalizations, about $1.5 million. So you'll see that any infection that we can avert is going to be cost savings. So let's talk a little bit about the pathogenesis of acute HIV infection. And we're going to start off by our first polling question. So which Oregon or organ system contains the most amount of T cells?

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Is it the lymph nodes? The gastrointestinal tract, the blood, or the spleen?

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Okay, all right. So about 24% said the lymph nodes, which is a good guess, another 17% said the gastrointestinal tract 24% said the blood and I guess the highest proportion or 34% said the spleen? And the correct answer is the gastrointestinal tract, or what's called the gut, associated lymphoid tissue. And that kind of makes sense that your gastrointestinal tract all the way from your small intestines to your large intestines, including anal rectal tissue, would be studded with lymph tissue. Imagine all the things that we ingest into our body, you really need that very robust proximate immune system very close to where pathogens exist. And so that's the gastrointestinal tract. So this factors into our current model of pathogenesis for acute HIV infection. So I think I described previously that, you know, peak viremia, about day 23. So those viral load starts to really ramp up, and then your immune system kicks in. But before it does that, we get this acute drop in our T cells. And that's because of this overwhelming amount of viremia. And we see this acute drop and CD for cell counts doesn't last very long. That's why very rarely, sometimes the initial presentation of somebody in acute HIV infection may be an opportunistic infections such as CMV

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candidiasis in the mouth

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because this acute drop can sometimes occur and be of a magnitude where the CD for counts even dropped to below 200. And then the second arm of the immune system, the cytotoxic lymphocytes or CD eight arm starts to kick in, starts to neutralize viral replication. And then about three weeks after infection is when our antibody so that's your body's response against the virus starts to kick in. But again, the, the mucosal CD for cells and mucosal, that's another name for this gut associated lymphoid tissue. early HIV infection destroys the gut, associated lymphoid tissue in about two weeks. And that tissue isn't really effectively restored or reconstituted, even with effective antiretroviral therapy. And it's one of the Achilles heel to why persons

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with HIV

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have an impaired immunological system, and also why they may have defects in their mucosal barriers that may lead to microbial translocation, leaky gut syndrome, and hyper hyper immune function and inflammation. It's because of that big hit that the gut associated lymphoid tissue takes early on. And that persists even despite effective antiretroviral treatment. So another cartoon to really exemplify acute HIV infection and why it's important that we notice the signs and symptoms. So on the top here, we have these little, very yawns. And here in the purple is some mucosal barrier, it could be the lining of the mouth, vagina, or anus. It's hypothesize that the first immune cell that HIV encounters is this dendritic cell. See, these got these little spikes or dendrites? Once it attaches to HIV, then it ferries HIV to a regional lymph node. And that takes about two days. From there, HIV starts to replicate within these regional lymph nodes.

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So that about

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after an additional three days, or five days after translocation by the dendritic cell, that HIV starts to spill out into the bloodstream, and this is an animal model. So really, within five to seven days, after it crosses the mucosal barrier, we have HIV now that becomes systemic. What that means is HIV is in the bloodstream, and it now has invaded every organ system, every tissue, including brain, spleen, lymph, node, skin, heart and lung. Once this infection spills into the bloodstream, and patients have viremia, then that infection cannot be reversed. That's why post exposure prophylaxis it's so important that we get these antivirals to the patient within 72 hours, 72 hours after a potential exposure, so that we can prevent these first few cycles of viral replication, contain the infection locally, and then allow for your own immune system to abort the infection. If we wait too late or beyond the 72 hours really beyond five days, we get to the point where the virus may have already slipped into the bloodstream.

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So polling question number two,

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all patients that go through acute HIV infection will be symptomatic. Is this statement true? Yes or no? So all patients that go through acute HIV infection will be symptomatic. Is this statement true or false? Okay, and 92 percent of you gave the correct answer no. Well, we know in medicine when we say all every time, it tends to be false. Yeah. So it's difficult to exactly point towards the true prevalence of symptomatic HIV. But I would say anywhere from 50 to 80% of patients have symptoms, sometimes they're often mild and we're going to go through what those symptoms are, but there are some persons that go through acute HIV infection asymptomatically.

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So, correct answer,

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no. All right, polling question number three, which of the following symptoms is typically not present in persons experiencing acute HIV infection? So, which of the symptoms is typically not present?

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Is it generalized lymphadenopathy rash, fever, cough

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or men in dismiss and men and dismiss means meningeal symptoms such as headache photophobia. So, which of these following symptoms is typically not present

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in persons

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experiencing acute HIV infection? So about 7% said lymphadenopathy is not present. 12% said rash 5% Fever, okay. 32% said cough, and then the single most common response was meningeal symptoms or meningitis months, which is not a bad guess. But the correct answer is cough.

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So

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persons in acute HIV infection generally do not have sinal pulmonary symptoms,

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which means

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they don't have runny nose. They don't have sneezing. They don't have cough or bronchitis or pneumonia. This is an atypical viral infection, really, without sinal pulmonary symptoms, and we're gonna go through the algorithm. Many geo symptoms actually do occur, because an acute HIV infection, HIV does cross the blood brain barrier and the lumbar fluid symptoms of acute HIV infection. So really the most common signs and symptoms and these are taken from HIV guidelines.org is fever. Generally, the majority of patients that go through acute HIV infection are going to have fever. Another common symptom is a more Bill form rash, and I'm going to kind of show you some pictures of what that looks like, but it's like a sunburn. And it's a type of rash that blanches meaning when you apply pressure to the rash, it goes from red or a dark color to kind of lighter color. And then upon release it it re colorizes to either read or a purplish color pharyngitis can be present lymphadenopathy and that's generalized lymphadenopathy, cervical, posterior occipital, inguinal, axillary. And then headache and meningitis occur at a frequency of between 24 and 70% mucocutaneous ulcers, so ulcers in the mouth and in the anus, and then if we start to look at laboratory measures, they can have thrombocytopenia leuco P leukopenia. And a mild trans ammonite is not like an acute Hepatitis A or B, or even C. It's kind of milder. And these are kind of the laboratory changes that we see with acute viral infections. And in persons in particular younger persons. It may mimic Epstein Barr Virus or mononuclear. doses. So these are some of the comparisons of acute HIV versus EBV. So in acute HIV exudates in the throat are rare, whereas with mononucleosis, exit AIDS are more common. In acute HIV, there's a greater chance of presence of painful Muco. cutaneous ulcers whereas with mono no ulcers, again, more below form rash is more common in acute HIV than in EBV. Mono. And GI symptoms can occur in both but it's rare. And again, for mono, you're going to see an abundance of these atypical lymphocytes. And of course, this the mono spot test for for EBV is going to be positive. So here's an example of a person darker skinned with this more Bill form rash. This was during his presentation for acute HIV infection. Here's a lighter skinned individual with this kind of fainter more subtle rash on the upper chest and torso. And here's kind of like a zoom in so. And then these are shallow mucocutaneous ulcers in the upper part of the mouth. And here are oral ulcers kind of served pigeon, this painful on the tongue in a person in acute HIV infection. And one of the more common oral manifestations is linear gingival erythema. So we see these bands of hyperemia, that kind of hug the gingival line, and it's the most common oral presentation of persons and acute HIV infection. Also, genital ulcers can be present in persons in acute HIV infection. So this is a general genital ulcer, syphilis screening was negative. But keep in mind that patients can present can comment intently with acute HIV infection and an acute sexually transmitted infection, either syphilis

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or other STIs. So how do we detect

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persons and acute HIV infection? So remember, symptoms are going to occur about two weeks after an exposure. But the single most sensitive and test for detecting HIV infection is going to be the viral load. And that is going to be the PCR or more typically the HIV RNA test. And that typically can pick up persons and acute HIV infection by day 10. The P 24 antigen, which is a antigen, that's part of a fourth generation antigen antibody tests can detect HIV by about day 17.

Then patients have symptoms. And then based on the antibody test, and typically we're using the fourth generation antibody test, those can typically pick up by, you know, three weeks. But maybe sometimes as early as 710 days, Western Blot testing isn't really done anymore, but really by definition, then, acute HIV infection is a positive viral load with a negative antibody test really dates the infection to within those first couple of weeks. Now, a person can also be in kind of acute late acute HIV infection if their P 24. antigen test, in addition to their viral load test is also reactive. But again, the single most sensitive tests for picking up early HIV infection is PCR RNA based

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testing. So

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how effective are we at diagnosing acute HIV infection? We have to imagine that every person living with HIV around the world has gone through acute HIV infection and the majority of those have been symptomatic see conversions. However, of the nearly 60 million individuals diagnosed with HIV, fewer than 1000 cases have been diagnosed in acute HIV infection. So that's a one in 60,000 detection rate. So we're, so we're not very good at diagnosing persons and acute HIV infection. And again, I think if we're able to think about this differential in persons when they present to emergency rooms, hospitalizations, or even outpatient dermatological GYN visits, and we're able to diagnose them, then I think we could do a lot, both for the individual and for preventing secondary and onward transmissions.

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So typically,

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I think we've been lacs about diagnosing acute HIV infection, and that they feel this is something that should only be relegated to kind of like a HIV or infectious disease specialist. And that's not really the case, because patients aren't really going to be referred or if they are, then basically, they're going to be on that very kind of unique period of time. So I feel there's a lack of education for how to diagnose acute HIV infection, which we just went over. And also kind of discomfort related to some of the difficult issues surrounding HIV. So you do have to talk about sexual risk, you do have to talk about substance use disorders, in particular, injection drug use. And, again, we are moving away from risk based testing. However, if a person should present with a febrile like illness, and it's not a sinusitis, or COVID, or flu, that I think you should consider specifically asking the patient about any recent sexual or needle sharing exposures, and I would say within the last month, and if so, to consider screening for acute HIV infection, so it really doesn't take a lot of additional time. It's basically understanding signs and symptoms, asking specific pointed questions, and then ordering the appropriate labs, and scheduling follow up. So really, if you look for it, you'll find it. In the literature, you know, 1% of patients with negative tests for EBV had acute HIV infection that was in one cohort. And 1% of patients with any viral syndrome syndrome in a Boston urgent care center also had acute HIV infection. And in a Malawi STD clinic, 2.8% of all male clients with acute with an acute sexually transmitted infection, also had acute HIV infection. So again, it's something that is more common than we

imagined. And in this paper from the Annals of Internal Medicine by T Shachar. At all. He actually looked at the clinical presentation of patients in an acute HIV infection, where he was able, again, to really time the time of exposure, and then symptoms and subsequent testing. And what they found is that the vast majority of patients, and the column on the left is patients who were in acute HIV infection. The one on the right is patients who had seroconversion at study entry already, so 94% of patients who were RNA positive and antibody negative or in the real throes of acute HIV infection. 94% or 16, out of the 17 were symptomatic. 94% of the patients you know, they just didn't stay at home, they actually sought medical help. So they went to the urgent cares and EDS. Five or 29% were hospitalized. But only 71 or 12 had HIV testing during symptoms. The median duration of symptoms was about two weeks. And the median time from onset to, to actually diagnosis of acute antiretroviral syndrome was about 25 days. And here you'll see the mean CD for accounts. So how do we diagnose acute HIV infection? I mean, not just in New York State but also nationally. So there is a specific ICD nine code for acute HIV infection, and its exposure to HIV. And that code is z 20.6. And it's important because remember, the labs that you're going to request for this patient isn't just HIV testing using antibody testing. But you're also going to throw in a viral load test and HIV one viral load assay. Remember, the viral load testing is really just used for persons already living with HIV as a way to monitor the effects or response to treatment. So in order to get this viral load reimbursed, you got to put in this specific ICD 10 code. We no longer need separate written informed consent. So what I always like to say is Hi, I'm Dr. Wood Bina, just as part of routine health care, I'm going to throw in a comprehensive metabolic panel with CBC hemoglobin a one C, but I'm also going to offer an HIV test. So you need to have verbally informed the patient and verbal consent and if the patient a sense, then you can go ahead and perform the test. Now, which of the following lab tests is typically normal in persons in acute HIV infection? So polling question number four, is it CBC?

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Is it liver function test? Is it metabolic panel?

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Or is it cerebrospinal fluid? All right, so 26% Set CVC. Remember, there is going to be that leukopenia typically that happens, so this slight decrease in WBCs 15% said liver function test remember there's going to be a mild increase in the TransAm and aces in great 41% segment said metabolic panel, and that is correct. And remember with the cerebral spinal fluid remember that HIV does cross the blood brain barrier and can cause a pleocytosis protein spilling into the CSF. And also the presence of WBCs in the CSF. So right, the correct answer is metabolic panel. So sodium glucose, serum creating those should typically be normal unless there's some other coexisting condition. So what are the tests that we order for persons that we believe are in acute HIV infection? Well, we definitely want to get the HIV antigen antibody tests. So the latest up to date CDC recommended fourth generation antibody test, we want to check the HIV viral load test, we want to include a metabolic panel because we want to exclude other conditions like DKA, acute kidney injury, we want to do CBC for the same reason and also liver function test. And if tests do confirm acute HIV infection, then we want to initiate a rapid start protocol by drawing these baseline labs and initiating antiretroviral therapy. So the HIV guidelines recommends, again, the HIV one to antigen antibody test, the HIV quantitative viral load. This is

once it's been diagnosed. HIV genotypic testing resistance profile T cells, check for Hepatitis ABC comprehensive panel, start the sexual health screening and pregnancy testing but again, so if the tests confirm acute HIV infection, we need to bring the patient back in and throw in these additional test. But what I want to argue is that once we have that positive viral load that comes back, is that we immediately encourage the patient to start on antiretroviral therapy, so that we can potentially preserve that gut associated lymphoid tissue. Because if we can preserve that, then we can preserve that mucosal tissue surrounding the gut and prevent these downstream hyper inflammatory complications. Also, if we can halt HIV viral replication early on On then we can also prevent HIV from penetrating into the central nervous system.

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So what do we treat with?

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So typically what's recommended from the guidelines is that we utilize Taff FTC or big tech revere or Taff FTC darunavir boosted with Kobe systat. And again, if patients are on PrEP and develops acute HIV infection these may be also things that we want to consider is that if a person is on PrEP, and they should develop acute HIV infection, what the guideline saying is that they may have serial converter with some resistance. So they recommend using a what's called a mega heart regimen of some tuza right there, which is Taff FTC boosted darunavir, with W Tegra. Vir, wait for the results of the resistance test to come back. And then from that you can simplify therapy. But again, starting patients and acute HIV infection in addition to protecting the gut, you also mitigate the establishment of the central nervous system. So there's data to support that if you treat patients during acute HIV infection, it provides an opportunity to decrease the central nervous system reservoir and to protect the brain from immunologic activation, and inflammation. And we know that in persons living with HIV, even those perfectly controlled that they have more inflammatory markers in the CSF and brain, and higher incidence of neurocognitive decline. So again, treating patients early is one of the proven measures to really mitigate these neurocognitive issues in persons with HIV. After you identify someone in acute HIV infection, I think the most important thing is just to make that person aware, Hey, your viral load came back looks like you're in acute HIV infection, what I want you to do is protect yourself, protect your partners, and avoid breastfeeding or any unprotected sex or sharing of drug paraphernalia. Really until you know, we get your viral load under control. So you want to emphasize safer sex and drug and using practices. So important to state that the patient is highly infectious or to take steps really to prevent any transmission. And you do really want to talk about their sexual and needle sharing contacts, and work with the patient and your local health department to identify other patients within that network. And the good thing is that persons who are informed of being an acute HIV infection typically take steps to prevent onward transmission. So this one multisite acute HIV infection study, there were 27 participants that were diagnosed and acute HIV infection, and they had to complete a survey. And what they found is that after their diagnosis, and after being informed that the majority of those participants had a significant drop in the number of sexual partners. After diagnosis, more than 95% of sex acts were done with people who were of the who were also HIV positive. And even though there was no significant change in the number of sex acts, there was an increase in sex using condoms. So it's important to understand these dynamics in terms of these clusters that occur

around acute HIV infection and this rapid firing of transmission events. So this cartoon here we have one person in acute HIV infection. Let's say he transmits to this person. If you're able to identify this person, through viral load testing, and through partner contact tracing and notification, then you're able to identify person or persons that are what are called efficient disseminators or persons that are contributing to a lot of new onward cases of HIV infection. And at any point during diagnosis, you can break these chains of HIV infection by identifying these networks. Another example, in February of 2005, an African woman gives birth in the Bronx normal, spontaneous vaginal delivery. both the mother and the baby are both HIV negative. The mother visits an ER in the Bronx and she's diagnosed with the viral syndrome is told It's okay for her to breastfeed.

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So that's about three months after.

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And then the infant is admitted for a gastroenteritis. He has thrombocytopenia. Two months later.

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The infant is

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discharged back home, but he's admitted for worsening thrombocytopenia and he's treated for idiopathic thrombocytopenic purpura. But now his condition gets worse. He has fever, periorbital, edema, elevated liver function test and pancytopenia. And they finally console ID and the infant is diagnosed with primary or acute HIV infection. So let me just give you this. So the African woman is tested negative, so Mother and Baby both tests negative. So the mother was negative during a pregnancy, and the baby was also tested negative. What happened is that the mother's

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husband,

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who was recently infected with HIV visited her postpartum they had sexual relations. They HIV one was transmitted to the mother, the mother breastfed, and she transmitted HIV to the infant during breastfeeding. So again, the newborn screen was negative. But when they finally did entertain the diagnosis, the rapid HIV test was positive at that point that utilize western blot testing. And the HIV plasma viral load was very high and T cells were also low for an infant. So again, presentation of acute HIV infection. This was in the infant and they didn't quite do the archaeology and it was not considered in the differential. So last case, 20 year old male we'll call him patient a. On July 29, he developed headache and fever, he went to his local emergency department. And because of the symptoms he underwent lumbar puncture, he was placed on doxycycline. They thought he may had Rocky Mountain spotted fever, and so on. He was told to go home that the symptoms would likely go away on August 4, he presented to

another local emergency department and admitted with headache, fever, nausea and vomiting. His labs wiped out 4.4 platelets 115,000 They finally do a Rocky Mountain Spotted Fever antibody test which did negative and they just do an Elisa antibody test which is negative. Now patients a symptoms resolved but patient a has sex with patient B his partner who's a 21 year old male, and they have unprotected sex three to four times with patients see and he's a 22 year old male who joins them for a three way sexual encounter with patient a and b. Now on August 30, through September 9, patient B and C have sex one to two times per week.

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September 10.

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Patient B develops fever to under four with seven to 10 days also has fatigue and sore throat so he sees his PMD and they're given a Z Pak and Vika den, and on September 30 patients see develops fever, sore throat and rash he sees his PMD and he's only given azithromycin. Patient B and C have a threeway sexual encounter with patient D. Now patient D developed sore throat, oral ulcers, thrush and fever. On October 31, partner D visits his MD and request STI testing and no HIV testing is done. On November 3, patient D is diagnosed with lymphoma and request HIV testing and finally is When HIV testing is done and suggest early HIV infection I can this diagnosis of lymphoma was not due to acute HIV infection I think it was just an incidental finding. But what I wanted to exemplify here are these onward rates of transmission. So you know aNbf sex a transmits to b b a and c have sex and either B or A transmit to see and see B and D have sex and either C or be transmitted to D and at any one of these points. If at one of these encounters, a history was taken or the symptoms a broader kind of differential and screening for acute HIV infection. We could have broken these chains of HIV transmission and in fact it may just be Hey These are your signs and symptoms, it may be some other viral syndrome. However, given your recent sexual exposure, it could be acute HIV infection, what I want you to do is to, we're going to draw some bloods maybe do a rapid test, we're going to wait for this viral load to come back. But in the meantime, what I need for you to do is to prevent is is to protect yourself and protect your partners. So finally, what is the algorithm that we use at Mount Sinai for screening for acute HIV infection? So really, the main thing that we start off with is fever. Does the patient have a febrile illness? So generally asymptomatic patients, we really don't screen for acute HIV infection. The only time we're going to screen them for acute HIV infection, if really, if we're considering starting them on pre exposure prophylaxis. But otherwise routine visit if they have fever. And then we're going to ask, do they have any cough or nasal congestion? So are there any signs and symptoms of a sign of pulmonary infection? If it's yes, and then that's where we're going to start really thinking about an atypical viral infect? I mean, I mean, we're going to start thinking about more common kind of respiratory infections. So yeah, flu, SARS, cov.

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Two

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diagnoses like bronchitis, sinusitis, pneumonia, so acute HIV infection is less likely, we still aren't going to ask about any sexual or needle sharing risks within the last really four weeks, I think it's sensitive enough. And if they do have symptoms, then yeah, we may want to additionally do screening for acute HIV infection. But if they should have fever, and none of these sinal pulmonary symptoms, that's when we're going to start to do a little bit more of a focused exam and history and ask and examined for the presence of rash pharyngitis lymphadenopathy, I'll faroush as my allergist, mucocutaneous, ulcers, signs or symptoms of meningitis. So definitely fever with the absence of the sign of pulmonary and some of these presenting symptoms, then yes, we do want to screen for acute HIV infection. So why don't we screen everybody that walks through our door just with viral load testing? Well, the reason for that is that if you start to screen lower risk populations, or just everyone, your pretest probability for a false positive starts to increase, and you may have these false positive viral loads. So that's why really, we want to specifically hone in on symptoms, the absence of sino pulmonary symptoms, and then a history that suggests potentially recent exposure. So remember that the new HIV testing algorithm is the fourth gen antigen antibody testing member, it has this P 24 antigen that can be detected early on. But if the initial screen is negative, then the algorithm stops. So remember, then that's why we have to include viral load testing, for persons that we suspect are in acute HIV infection, because this fourth gen algorithm is only able to detect this p 24. antigen, which is only released about seven to 10 days, or is detectable seven to 10 days after viral load testing. If the screen is positive, then the algorithm will reflect to a differentiation assay. If it's one or two. If either of those are negative, then it may be the the P 24. That was detected, and the algorithm will automatically reflect to a qualitative HIV one viral load. And if that's positive, that's where this fourth gen can pick up early ish, acute HIV infection but again, if you still suspect acute HIV from the initial presentation, in addition to this, you're also going to throw in viral load testing. So I will end there, this is our CI line you can call for inquiry about any topic HIV HCV, Drug User Health, STD, PEP or PrEP related. So I I will end there. And we have these rapid AR T and HIV testing clinical cards, which will really walk you through our HIV guidelines, New York State Department of Health guidelines for how to initiate rapid anti retroviral therapy and those newly diagnosed how to perform HIV testing. So again, you can request these cards from either an email or from talking with Mark Stratton. And I will end there and see if anybody has any questions.

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Yes, thank you so much for that presentation. Dr. Bina. I know that in the beginning, Dr. Urbina, I'm sure when COVID cases were surging, it must have there were probably I assume people who might have been having an acute HIV infection. And if they didn't test positive for COVID, maybe were turned away. Do you know of any patients like that? Or or did you did you notice any instances of that happening?

51:09
Right, yeah.

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So yeah, I think there was a bit of an overlap between patients early part of the epidemic with SARS code to Where where are they presented, and they thought that they had COVID. And in

fact, they were in acute HIV infection, and they were misdiagnosed, and it was only later when they came in. And because I think of that decreased access to testing. They just missed that opportunity of being diagnosed with acute HIV infection. And there were cases where there was actually transmission within couples. I mean, you can differentiate, you know, typically, the onset of symptoms for COVID are quicker, like five, five days versus the two weeks. And again, they have the sign of pulmonary symptoms. But again, I think there was time where a lot of persons just attributed their acute HIV to COVID and it was not the case. All right. Well, thank

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you everybody for attending.

[End Transcript]