**Professor Lupus: Rose’s Experience with SLE**

**Timestamps**

**0:00-0:12: Victoria introduces the podcast and the group introduces themselves**

* Hi! We are Group Number 8, Professor Lupus, here to talk to you about systemic lupus erythematosus or as we will refer to it as SLE. This is Miranda, Shanelle, Victoria, and Kennedy.

**0:12-0:21: Shanelle introduces Rose, a fictitious case study about a 24-year-old Black woman with SLE**

* Today, we’re going to talk about Rose. Rose is a fictitious 24-year-old Black woman attending Cariboo College. She is in her second trimester of her first pregnancy.

**0:21-0:38: Kennedy explains that pregnancy can trigger SLE flare-ups and mentions Rose’s specific risk factors**

* It is common for women to experience flare-ups of SLE during their first or second trimesters (Office on Women’s Health, n.d.). Rose had likely been living with SLE prior to her pregnancy, but the flare caused her to seek medical attention. Some other risk factors affecting Rose include being Black and being in her reproductive years, around 20 to 40 years old.

**0:38-1:20: Victoria discusses SLE prevalence in people of colour, diagnostic challenges, and environmental triggers**

* People of colour are approximately eight times more likely than white people to have SLE (Power-Kean et al., 2023). Delays in diagnosis or misdiagnosis of skin-related conditions are also more prevalent in people of colour. Medical textbooks typically display photos of rashes and conditions on light skin and lack photos displaying the same rash on darker skin tones. In the case of SLE, the characteristic “red rash” over the bridge of the nose can appear more purple, or just have characteristics like “itchy” and “scaly” instead (Ongoro et. al, 2023, Mukwende et. al, 2020). This disease also has genetic connections, and there are examples of affected twins often both exhibiting SLE (Power-Kean et al., 2023). Genetic factors, combined with an environmental trigger, can cause the onset of disease. Some environmental triggers include UV light, smoking, viral infections, and estrogen (Vaillant, et al., 2023).

**1:20-1:40: Shanelle returns to Rose, detailing her symptoms and the reason for her clinic visit**

* Okay, back to Rose. Recently, she has noticed an itchy, scaly rash across the bridge of her nose and her cheeks, she has been feeling more tired than usual and has noticed an increase in hair loss. This causes her to visit the college health clinic where you are working. Having listened to a podcast back in nursing school, you remember that a butterfly rash is a cardinal sign of SLE (Cleveland Clinic, n.d.).

**1:40-2:54: Kennedy and Miranda explain the signs and symptoms of SLE**

* Symptoms for SLE can vary greatly between patients, so it is important to remember that any mix of the following symptoms can be present:
* Skin irritation and rashes are common. It is important to note that ethnicity can alter the presentation of rash. This usually looks like the classical butterfly rash to the face, discoid rashes all over the body, ulcers of the mucosa in the nose or mouth, or rashes in areas exposed to sun and hair loss (Power-Kean et al., 2023, Lupus Foundation of America, n.d-a.; Ongoro et al., 2023). Second is related to damage to body capillaries. This can lead to arthritis in peripheral joints, kidney dysfunction, edema to the lower legs (Health Union, n.d.), and neurological disorders such as seizures or psychosis with no cause (Power-Kean et al., 2023). The third is related to the continual stimulation of the immune system. These are more insidious and present as consistent fever (Timlin et al., 2018), and generalized fatigue (Timlin et al., 2018; Kawka et al., 2021). Symptoms can also be observed via bloodwork. These include the presence of antinuclear antibodies (ANA), and other hematological disorders such as hemolytic anemia (low RBC), leukopenia (low leukocytes), lymphopenia (low WBC), or thrombocytopenia (Low platelets) (Power-Kean et al., 2023, Power-Kean et al., 2023).
* Her doctor decides to do some diagnostic testing.

**2:57-3:12: Kennedy and Victoria explain the ANA blood test used to diagnose SLE.**

* Diagnosis often starts with a positive antinuclear antibody blood test, called an ANA. ANA detection can come years before the onset of symptoms. Because Rose is symptomatic, it is likely she will have antinuclear antibodies present (Power-Kean et al., 2023).
* 98% of those with SLE test positive for ANA (Power-Kean et al., 2023).

**3:12-3:21: Kennedy notes Rose’s advantage in early diagnosis due to her doctor’s familiarity with SLE.**

* Rose is also lucky, in that her MD has a relative with SLE, and thought to test her ANA right away, due to recognizing the disease pattern.

**3:21-3:29: Victoria shares that SLE diagnoses can take years and lists additional tests.**

* It takes 47 months, on average, to reach an SLE diagnosis (Kernder et al, 2021).
* Some additional diagnostics include testing for double-stranded DNA, or Smith antibodies

**3:29-3:50: Shanelle explains Rose’s interactions with the healthcare team at the clinic, and provides context on Rose**

* The doctor, being short on time, goes over the diagnosis of SLE with Rose very quickly and is unable to answer most of Rose’s questions. This leads her back to you. Rose is studying psychology and has above-average health literacy levels. She is currently in the middle of exam season and has limited access to supportive resources. Rose is wondering what happened to cause this

**3:50-4:57: Miranda explains the genetic and cellular mechanisms involved in SLE.**

* Although there is not a definitive causative agent for SLE, it is currently thought that there is some sort of cellular damage involved. This leads to changes in DNA expression where an excess amount of anti-nuclear antibodies are produced. In general, a recognized mutation within genetic material will typically cause cells to undergo cellular death or apoptosis that releases antibodies. In a person with SLE, there is a genetic predisposition that decreases the ability of our macrophages to recognize and get rid of these particular mutagens. This leads to a high number of antibodies and not enough antigens- leading to the problem of insoluble antigen-antibody complexes. This is also known as a type 3 hypersensitivity reaction. Complexes are deposited in global capillaries, such as in the kidneys, skin, joints and heart. The cellular damage done to the blood vessels stimulates the classical complement system pathway: this leads to mast cell degeneration, chemotaxis, opsonization of foreign material and the membrane attack complex! (Power-Kean et al., 2023, Osmosis from Elsevier, 2016, Vaillant, Goyal & Varacallo, 2023)

**4:57-6:00: Victoria discusses the role of neutrophils and their impact on SLE symptoms.**

* Neutrophils also play a role in the symptoms of SLE. In SLE, neutrophils are more active than normal, and when they die through apoptosis, the macrophages that clear them don’t work effectively. As a result, the dying neutrophils release more DNA into the body, particularly a type called double-stranded DNA (dsDNA), which can trigger further immune responses. (Ma et al., 2023) Neutrophils release structures called NETs, or neutrophil extracellular traps, to trap and fight off large pathogens (Ma et al., 2023). In SLE, NET production (known as NETosis) is overactive and not well-regulated, leading to tissue damage instead of protection (Papayannopoulos, 2017). NETs contain: self-antigens including dsDNA, histones, chromatin, granule proteins, and mitochondrial DNA (Ma et al., 2023). Neutrophils in SLE also act like antigen-presenting cells (APCs), showing more “self-antigens” to the immune system, which increases the risk of immune attacks against the body’s own tissues (Ma et al., 2023). Neutrophils also encourage B cells to make auto-antibodies; antibodies that mistakenly target the body’s own tissues (Ma et al., 2023). And they release a lot of pro-inflammatory cytokines, which spread inflammation, and damage tissues throughout the body (Ma et al., 2023).

**6:00-6:12: Shanelle conveys Rose’s concerns about SLE's impact on her and her baby.**

* Rose is concerned about the implications of all of this. She asks you about the prognosis of SLE for herself and her baby. Keeping in mind the limitations of your scope as a nurse, you formulate a response.

**6:12-6:44: Kennedy outlines the general prognosis of SLE, including flare-ups and remission periods.**

* As a nurse, you know: That the prognosis greatly depends on the severity of Rose’s disease at this time and can range from mild to severe. And there are flare-ups when her body becomes stressed where symptoms may become overwhelming and may interfere with her ability to complete normal tasks. Then there will be remission periods in which the symptoms associated may be mild or even absent. It is very important for Rose to be engaged and follow her care plan as it will ensure the best outcome for her. Worst-case scenarios could include multiple organ failure, infection, confusion, headache or even psychosis. (Lupus Foundation of America, n.d.-b). (de Souza et al., 2021). (Dong et al., 2024).

**6:44-7:50: Miranda discusses pregnancy risks associated with SLE, potential complications, and genetic factors.**

* Pregnancies for patients with SLE pose a greater risk of fetal loss and pre-eclampsia. Babies are at risk of long NICU stays, intrauterine growth restriction (IUGR), prematurity, low birth weight and neonatal lupus (Aly et al., 2015, Limaye et al., 2020). The most common maternal manifestations of SLE are skin lesions (93%), lesions of the cartilage of joints (92%), kidney dysfunction related to lupus (53%), hypertension (39%) and secondary antiphospholipid syndrome (APS) (38%) (Aly et al., 2015, Limaye et al., 2020). 44% of pregnant people with SLE have antenatal flares, mostly occurring in the second trimester (70%). Some patients can have postpartum flares as well (7%) (Aly et al., 2015). In the future, Rose’s baby may have the genetic information to develop SLE. SLE triggers are not well understood as some individuals with SLE do not have genetic markers of SLE, and some have genetic markers but never develop the disease. It is a combination of both the presence of genes plus an environmental trigger that causes the onset of SLE (Harley & Sawalha, 2022).

**7:50-8:08: Shanelle describes Rose’s feelings of uncertainty about her future with SLE.**

* Rose is pretty overwhelmed at this point. So much seems to be up in the air right now. She doesn’t know how often she will have flare-ups, she doesn’t know when remission might start, and she doesn’t know what the next steps are. As the nurse caring for Rose, it is your responsibility to support her through this transition of being newly diagnosed.

**8:08-8:40: Victoria talks about SLE treatment options aimed at symptom control.**

* Unfortunately, there is no cure for SLE. Typically, the goal of treatment is symptom control by reducing the autoimmune response. This can be achieved by a variety of medications: NSAIDs, like ibuprofen, acetylsalicylic acid (aspirin), and naproxen can be used to manage pain and inflammation. Corticosteroids are used during flares to suppress the immune response. Immunosuppressive medications are used to treat symptoms of organ damage such as methotrexate, cyclophosphamide, and azathioprine. Antimalarial medications such as hydroxychloroquine are used during periods of remission (Power-Kean et al., 2023, p. 210).

**8:40-9:22: Kennedy suggests lifestyle changes to support Rose’s mental and physical health.**

* Rose might benefit from some non-medicalized, lifestyle changes to manage her disease. Having an SLE diagnosis can lead to depression and/or anxiety. This could in turn lead to reduced health-related quality of life. Psychosocial interventions can reduce these symptoms. Additionally, regular physical activity can also contribute to lowering the mental health issues related to SLE, while also reducing fatigue, and increasing physical and aerobic functioning. Diet changes can help reduce inflammation, and there is evidence to suggest that moving to a Mediterranean diet, or low-fat diet, can be supportive, while also decreasing cardiovascular risks. As UV exposure can cause exacerbation, using sunscreen and decreasing exposure can reduce the risk of disease flares (Tsoi et al., 2024)

**9:22-9:51: Miranda introduces ongoing research and new treatment avenues for SLE.**

* Outside of the realm of current treatment, there are some really interesting clinical studies going on towards SLE treatment and possible remission. In one instance, there is a specific type of chemotherapy that targets naive B cells, which is showing very promising results in helping move people from symptomatic to periods of remission. (Taubmann et al., 2024). While this is still a new path and further study is required, it is hopeful to see these new treatment types coming into the scope.

**9:51-9:55: Victoria thanks listeners, wrapping up the podcast.**

* Thanks for listening and Team Professor Lupus hopes you learned something new!

**9:55-9:56: CCL displayed**