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## Anatomy 210 assignment

1. The pandemic outbreak of coronavirus disease 2019 (COVID-19) is rapidly spreading all over the world. Reports from China showed that about 20% of patients developed severe disease, resulting in a fatality of 4%. Since the sudden outbreak of coronavirus disease 2019 (COVID-19) in Wu Han City, China caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), in just two more months, the epidemic has rapidly spread all over the world. On March 11, 2020, the World Health Organization (WHO) declared the COVID-19 outbreak a pandemic. China have indicated that about 20% of patients developed severe disease, older adults, particularly those with serious underlying health conditions, are at higher risk of death than younger ones. In severe COVID-19, although patients have lymphocytopenia, the lymphocytes were activated. One study analyzed the lymphocyte subsets and cytokines in 123 patients, all patients had lymphocytopenia, The percentage of CD8 + T cell reduction were 28.43% and 61.9% in mild and severe group respectively, and the NK cell reduction were 34.31% and 47.62% respectively, in mild and severe groups.

Lymphocytopenia is one of the most prominent markers of COVID-19, it's also one of the diagnostic criteria for COVID-19 in China [12]. Both T cells and NK cells in patients with COVID-19 were reduced. In addition, memory helper T cells and regulatory T cells were obviously decreased in severe cases. It was revealed that the secondary lymphoid tissues had been destroyed in COVID-19 patients, which is very unusual from other CS related diseases. Spleen atrophy was observed in all reported cases with decreased numbers of lymphocyte, and significant cell degeneration, focal hemorrhagic necrosis, macrophage proliferation and macrophage phagocytosis were found in spleen. There are two possible reasons for the destruction of the immune system in patients with COVID-19, lymphocytes directly invaded by virus or indirectly damaged by CS.

Another prominent clinical manifestation in severe COVID-19 patients is endothelium damage. Mimicry of vasculitis could be seen in severe COVID-19 patients. Clinically, many critical ill patients have vasculitis-like manifestations, or even gangrene at their extremities; Pathology examination revealed the blood vessels of alveolar septum were congested and edematous, with modest infiltration of monocytes and lymphocytes within and around blood vessels. Small vessels showed hyperplasia, vessel wall thickening, lumen stenosis, occlusion and focal hemorrhage. Hyaline thrombi of micro-vessels were found in a proportion of severe cases.

Intriguingly, some patients were tested positive with high titer antiphospholipid antibodies, including anticardiolipin antibodies and anti- $\beta$ 2 glycoprotein antibodies, and were associated with severe thrombosis (unpublished data). The underlying mechanism of vascular damage may be due to the direct injury of endothelial cells by virus, leading to DIC, anti-phospholipid syndrome (APS) and mimicry of vasculitis. The pathological autoimmune responses involved in the anti-virus immunity are worth to be emphasized.

2. The subsartorial canal is also called the adductor canal and the Hunter canal . It is an intermuscular passageway deep to the sartorius by which the major neurovascular bundle of the thigh traverses the middle third of the thigh. It is a long (approximately 15 cm), narrow passageway in the middle third of the thigh. It extends from the apex of the femoral triangle, where the sartorius crosses over the adductor longus, to the adductor hiatus in the tendon of the adductor magnus .

The adductor canal is an important part of the lower limb because it serves as a passageway for structures moving between the anterior thigh and posterior leg. It transmits the femoral artery, femoral vein (posterior to the artery), nerve to the vastus medialis and the saphenous nerve – the largest cutaneous branch of the femoral nerve. As the femoral artery and vein exit the canal, they are called the popliteal artery and vein respectively.

The adductor canal is bordered by muscular structures:

Anteromedial: Sartorius.Lateral: Vastus medialis.Posterior: Adductor longus and adductor magnus.

The adductor canal runs from the apex of the femoral triangle to the adductor hiatus – a gap between the adductor and hamstring attachments of the adductor magnus muscle.

3. The extraocular muscles are the six muscles that control movement of the eye and one muscle that controls eyelid elevation (levator palpebrae). The actions of the six muscles responsible for eye movement depend on the position of the eye at the time of muscle contraction. Four of the extraocular muscles have their origin in the back of the orbit in a fibrous ring called the annulus of Zinn: the four rectus muscles. The four rectus muscles attach directly to the front half of the eye (anterior to the eye's equator), and are named after their straight paths. The medial rectus is the muscle closest to the nose. The superior and inferior recti do not pull straight back on the eye, because both muscles also pull slightly medially. This posterior medial angle causes the eye to roll with contraction of either the superior rectus or inferior rectus muscles. The extent of rolling in the recti is less than the oblique, and opposite from it.

The superior oblique muscle originates from the back of the orbit (a little closer to the medial rectus, though medial to it), getting rounder as it courses forward to a rigid, cartilaginous pulley, called the trochlea, on the upper, nasal wall of the orbit.

The last muscle is the inferior oblique, which originates at the lower front of the nasal orbital wall, and passes under the LR to insert on the lateral, posterior part of the globe. Thus, the inferior oblique pulls the eye upward and laterally.

The movements of the extraocular muscles take place under the influence of a system of extraocular muscle pulleys, soft tissue pulleys in the orbit. The extraocular muscles develop along with Tenon's capsule (part of the ligaments) and the fatty tissue of the eye orbit.

#### Nerve supply

The subsequent nerve supply (innervation) of the eye muscles is from three cranial nerves which are the Oculomotor nerve, Trochlear nerve, Abducens nerve.

The Oculomotor nerve supplies the Superior rectus muscle, Inferior rectus muscle, Medial rectus muscle, Inferior oblique muscle and the Levator palpebrae superioris muscle.

The Trochlear nerve supplies the Superior oblique muscle

The Abducens nerve supplies the Lateral rectus muscle.

The intraocular muscles include the ciliary muscle, the sphincter pupillae, and the dilator pupillae. The ciliary muscle is a smooth muscle ring that controls accommodation by altering the shape of the lens, as well as controlling the flow of aqueous humor into Schlemm's canal. The ciliary muscle is attached to the zonular fibers which suspend the lens. Upon contraction of the ciliary muscle, the tension on the lens is lessened which causes it to adopt a more spherical shape to focus on

near objects. Relaxation of the ciliary muscle has the opposite effect, optimising distant focus.

The iris sphincter muscle (pupillary sphincter, pupillary constrictor, circular muscle of iris, circular fibers) is a muscle in the part of the eye called the iris. It encircles the pupil of the iris, appropriate to its function as a constrictor of the pupil.

The iris dilator muscle (pupil dilator muscle, pupillary dilator, radial muscle of iris, radiating fibers), is a smooth muscle of the eye, running radially in the iris and therefore fit as a dilator. The pupillary dilator consists of a spokelike arrangement of modified contractile cells called myoepithelial cells. These cells are stimulated by the sympathetic nervous system. When stimulated, the cells contract, widening the pupil and allowing more light to enter the eye.

Each muscle has its nerve innervation . The Ciliary muscle is innervated by the Parasympathetic nerve .

The iris sphincter muscle is innervated by the Parasympathetic nerve.

The iris dilator muscle is innervated by the sympathetic nerve.

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