**Lecture 17:**

* Prophylactic antibiotics

Principles of using prophylactic antibiotics:

1. Risk of infection should be significant.
2. Correct narrow spectrum antibiotics should be chosen; broad spectrum antibiotics may lead to increased resistance.
3. Antibiotic level should be high in the pt’s blood; that’s why we ask the patient to take the antibiotic half or at least one hour before the procedure. IV.: ½ an hour before the procedure. Oral: an hour before the procedure.

 following treatment suggestions from the American Heart Association's guidelines on antibiotic prophylaxis.

1. Time antibiotic administration correctly; administered at least 2 hours before the procedure.
2. Use correct doses.
* Factors related to postoperative infections:
1. The size of the bacterial inoculum.
2. Duration of surgery: any procedure more than 2 hours we must give prophylactic antibiotics.
3. The presence of a foreign body like an implant or dead space.
4. State of host resitance.
* Factors necessary for distant infection:
1. Distant susceptible site.
2. Haematogenous bacterial seeding.
3. Impaired local defense.
* Cardiac conditions with the highest association with endocarditis:
1. Prosthetic cardiac valve or prosthetic materials used in valve repair.
2. Previous endocarditis.
3. Congenital heart disease only in the following categories:
4. Unrepaired cyanotic congenital heart disease.
5. Completely repaired congenital heart disease with prosthetic material or device, whether placed by surgery or catheter intervention, during the first six months after the procedure.
6. Repaired congenital heart disease with residual defects at the site or adjacent to the site of a prosthetic patch or prosthetic device (which inhibit endothelialisation).
7. Cardiac transplantation recipients with cardiac valvular disease.
* Prophylaxis is reasonable because endothelialisations of prosthetic material occurs within six months after the procedure.
* Condition placing patient at risk for prosthetic joint infection:
	1. Prosthetic joint place within 1st 2 year
	2. pt with rheumatoid arthritis
	3. History of prosthetic joint infection
	4. Congenital or acquired immunosuppression disease
	5. Malnutrition
	6. Systemiclobusebethemimatous\* (notsure)
	7. Haemophilia
	8. Diabetes
* Dental treatment for diabetic patients has a local risk of infection based on finger stick blood glucose testing:

|  |  |
| --- | --- |
| Finger-stick results of blood glucose/mg (%) | Treatment |
| Less than 85 | Administrate glucose and postpone any elective treatment |
| 85-200 | Stress reduction , with a prophylactic antibiotic for extraction. |
| 200-300 | Stress reduction, a prophylactic antibiotic, and referral to a primary care physician. |
| 300-400 | Avoid elective treatments, referral to a primary care physician, send to a nearby ER room (in a hospital). |
| >400 | Avoid elective treatment, send to a nearby ER room (in a hospital). |

**Lecture 18:**

* Pain management:
* We have **3 characteristics** of pain **after extraction**:
1. Mild pain; can be managed by mild anelgesics.
2. Peak pain; occurs 12 hours after extraction and diminishes rapidly after that.
3. Significant pain rarely persists after 2 days.

If it persists it means that there is something else going on either a dry socket or infection.

* Pain control is more effective if you start analgesia before the pain starts.

It becomes more predictable to control pain with mild analgesics.

* Analgesics should be: 1. Cheap. 2. Effective. 3. Safe.
* Acetaminophen, ibuprofen and naproxen are the most common octs for the relief of dental pain.

Mainly acetaminophen and ibuprofen.

1. **Acetaminophen:**

It’s the safest and most widely used analgesia

It’s called Tylenol in USA & paracetamol or Panadol in UK.

It is still unknown how it works but it has a central component.

No anti-inflammatory effect.

Adult dose: 325-1000 mg 3 to 4 times daily, not to exceed 4 grams over 24 hours.

* Side effects:
1. Free of side effects at the standard doses.
2. Hepatotoxicity; since it’s metabolized in the liver.

Cut down the dose with liver disease.

1. If u give Panadol to someone who takes warfarin, you should cut down the dose because it has been discovered that Panadol interacts with warfarin, so if the patient is on warfarin, probably you may need to give a maximum dose of 2g of Panadol only, or if you decide to give it at a higher dose, you should monitor the INR for the patient because it may increase and the patient becomes at a higher risk for bleeding.

Yet still, Panadol is the safest analgesic that we can prescribe but the only precaution is for a patient having a liver disease.

1. **NSAIDs:**

MOA:

Most NSAIDs act as nonselective inhibitor of the enzyme clooxygenase **(COX),** inhibiting both cox1 and cox2.

Cox catalyzes the formation of prostaglandins and thromboxane from arachidonic acid.

Prostaglandins act as messenger molecules in the process of inflammation.

Prostaglandins also have a protective role in preventing **GI ulcers**, **regulating** **platelet action**, and are **necessary for kidney function**.

Cox2 enzyme produces prostaglandins but only as a result of inflammation and pain. Inhibition of both cox1 and cox2 enzymes not only reduces inflammation and pain but has the potential to inhibit the protective action of cox1 resulting in **GI toxicity**.

Long term use of NSAIDs also has been associated with **renal toxicity.**

1. **Ibuprofen.**

Dose:

For mild-moderate pain: 400-600 mg every 4 to 6 hours not to exceed 2400 mg/day.

* Precautions:
* Don’t prescribe it longer than 5 days never ever.
* You have to be cautious when prescribing a drug for patients above 60 years old because they are more prone to GI bleeding.
* Those who are alcoholic, because their liver is impaired.
* Who takes blood plugs.
* During the last month of pregnancy.
* Before and after surgery.
* People who are allergic to aspirin.

Ibuprofen is the most commonly used NSAID cuz it’s safe, effective & cheap. In dentistry it’s the drug of choice cuz it’s Anti-inflammatory and superior to Panadol. If u are expecting a moderate pain, the best combination is to take Panadol and ibuprofen together.

1. **Naproxen:**
* More expensive yet not more effective than ibuprofen.

Dose: **initial dose is 550 mg followed by 275 mg every 6 to 8 hours** is equivalent to ibuprofen 400 mg every 4 to 6 hours for mild to moderate dental pain.

* Voltarin: IM injection, 50 mg/day.
* Cardiovascular effects of nonselective NSAIDs:

Congestive heart failure, patients over 65 years, and previous history.

According for FDA in 2005: the administration of ibuprofen for pain relief to patients taking aspirin for cardio protection may interfere with aspirin’s cardiovascular benefits.

Acetaminophen or an acetaminophen-narcotic combination analgesic would be suitable for patients taking cardioprotective aspirin who require analgesics for postoperative pain relief.

* The appropriate timing of ibuprofen in relationship to aspirin therapy:

Patients who take immediate release aspirin should take ibuprofen at least **30 minutes or longer after aspirin** ingestion **or more than 8 hours before** to avoid attenuation of aspirin’s effect.

* Ibuprofen/ narcotic combination: more effective than each agent alone, ibuprofen allows for significant dose reduction of the narcotic.

400 mg ibuprofen with 15 mg hydrocodone was superior to 600 mg acetaminophen with 60 codiene.

* Drug interactions with NSAIDs:
	+ 1. Anticoagulants.
		2. Antiplatelets.
		3. Bisphosphonate derivatives.
		4. Lithium.
		5. Methotrexate.
* **Selective cox2 inhibitors:**

Rofecoxib (Vioxx), celebrex,and valdecoxib (bextra), indicated increased risk of heart attack and stroke associated with long term use of these drugs. Esp.Vioxx.

Bextra was taken off the market in 2005, showed a risk of skin reactions, heart attack and stroke.

The major interest in these drugs was related to lower incidence of GI bleeding.

* **Celebrex (celecoxib):**

Single doses of celecoxib relieved moderate-severe acute pain within 60 minutes.

Dose: 400 mg initially followed by additional 200 mg dose on the first day, then 200 mg twice daily as needed.

Celecoxib does not inhibit platelet aggregation at recommended doses.

No evidence of advantage over ibuprofen.

Increased risk of cardiovascular incidence.

* Cox2 inhibitor will be selected for patients at risk for GI bleeding. Use the lowest effective dose should be used for a limited time.

Monitoring of kidney function and blood pressure also is advisable.

1. **Narcotic analgesics for dental pain:**

NSAIDs are treatment of choice for mild-moderate pain. Narcotics are used for more severe intensities of pain.

Codeine, hydrocodone and oxycodone are the usual narcotics used.

* Repeated administration can cause:
1. Psychic dependence.
2. Physical dependence.
3. Tolerance.

The block pain reception in the cerebral cortex by binding to opiate receptors.

Recommended only for limited acute dosing.

They are available in combination with acetaminophen and ibuprofen.

1. **Non traditional analgesics (Tramadol):**

Atypical opoid analgesics.

Acts on opiate-mediated systems and monoaminergic pain-inhibitory pathways.

Not controlled and does not seem addictive.

Dose: 50-100 mg every 4 to 6 hours (up to 400mg daily).

Useful in cases where NSAIDs are contraindicated.

* Usually mild analgesics are required for 3 days only.

**Lecture 19:**

**Facial space infections:**

* Microbiology: part of normal flora.
* Aerobic gram +ve cocci:

Streptococcus milleri group.

Streptococcus viridians group.

Streptococcus anginosus.

Streptococcus constellatus.

Streptococcus intermedius.

* Anaerobic gram +ve cocci:

 Peptostreptococcus

* Anaerobic gram –ve rods:

Prevotella and Fusobacterium.

1. Mixed 50%
2. Aerobics alone 6%
3. Anaerobics alone 44%
* Stages:
1. Initial inoculation into deeper tissues, the S. milleri group can synthesize hyaluronidase. Which allows the infecting organism to spread through the connective tissues.

 Cellulitis.

1. Metabolic byproducts from streptococci create a favorable environment for the growth of anaerobes.

The release of essential nutrients, lowered PH in the tissues and consumption of local oxygen supplies.

1. The anaerobic bacteria are then able to grow predominate and cause liquefaction necrosis of tissues by collagenases.
2. As collagen is broken down and invading white blood cells necrose and lyse, microabscesses form and may coalesce into a clinically recognizable

 Abscess.

* Clinical summary:
1. First 3 days of symptoms a soft mildly tender doughy swelling represents the **inoculation stage.**
2. After 3 to 5 days the swelling becomes hard, red, and acutely tender as the infected mixed flora stimulates inflammatory response of **cellulitis stage.**
3. At 5 to 7 days after the onset of swelling, the anaerobes begin to predominate causing liquefied abscess in the center of the swollen area.
4. Resolution stage begins as the immune system destroys the infecting bacteria, and the processes of healing and repair start.
* Infection spreads equally in all directions, but preferentially along the line of least resistance.
* Antibiotics alone my arrest, but do not cure the infection.
* We should treat the cause.
* Most odontogenic infections penetrate the bone in such a way that they become vestibular.

Weather it becomes vestibular is determined primarily by the relationship of the muscle attachment to the point at which the infection perforates.

* What are fascial spaces?

Potential spaces that do not exist in healthy people.

They are only created by infection.

* Fascial spaces:
* Principal maxillary: canine, buccal, infra temporal.
* Principal mandibular: submental, buccal, submandibular and sublingual.
* Secondary: masseteric, pterygoid, superficial and deep temporal, lateral pharyngeal, retropharyngeal and prevertebral.
* Determine the severity of infection:

3 factors:

1. Anatomic location.
2. Rate of progression.
3. Airway compromise.
* Perimandibular space:
1. Submandibular space:

3rd molar and 2nd molar.

Opens to secondary spaces.

Bellow mylohyoid attachment and between platysma and mylohyoid.

1. Sublingual:

 between oral mucosa of the floor of the mouth and mylohyoid muscle.

Opens at the back with submandibular space and the secondary spaces.

Mostly with lower premolars and 1st molar.

1. Submental:

Between anterior bellies of digastrics, between the mylohyoid muscle and skin.

Lower incisors, infection erode apical to mentalis muscle.

Isolated submental space infection is rare.

* Maxillary spaces:
1. Canine:

Upper canine.

Infection erodes superior to the origin of the elevator angularis oris below the origin of the elevator labii superioris.

Obliterates nasolabial fold.

1. Buccal space:

Posterior maxillary teeth mostly molars, infection perforates above the attachment of the buccinators muscle.

Below the zygomatic arch and above the inferior border of the mandible (both are palpable.)

* It is important to differentiate between submandibular and buccal space infections.

Condition of the molars: buccal space infection can happen from lower molars too.

Palpate anatomical structures: inferior border of the mandible is not palpable when it is a submandibular infection.

1. Infra temporal space:

Lateral pterygoid plate medially, base of the skull superiorly, laterally open to the deep temporal space.

Mostly from macillary 3rd molar.

**Lecture 20:**

* Secondary fascial spaces:
1. Submasseteric space:

From buccal space or pericoronitis from lower 3rd molar.

Area over the angle of the mandible and ramus becomes swollen.

Moderate-severe trismus.

1. Pterygomandibular space:

From sublingual and submandibular spaces.

Significant trismus.

Little or no facial swelling.

Needle tract infection from ID block.

1. Temporal space:

Rarely secondarily involved usually in severe infections.

Swelling superior to zygomatic arch and posterior to the lateral orbital rim.

The pt come with swelling limited by outline of temporalis fascia,trismus,severe pain(superficial temporal).

Less swelling,trismus,difficult to diagnose (deep temporal).

* Cervical fascial spaces:

Uncommon.

Spread from the primary and secondary spaces.

Life threatening.

* they are divided into:
1. lateral pharyngeal.

Inverted cone or pyramid in shape ,the base at sphenoid bone and the apex on hyoid bone, has two compartment: anterior muscular and posterior vascular compartments.

can come from pterygomandibular space (2ndry space).

1. Retropharyngeal.

Posteromedial to lateral pharyngeal space and anterior to the prevertebral space . between serratus sup. Post.muscle and alar fascia .

1. Prevertbral space.

It can also cause mediastinitis as retropharyngeal space.

* What are the things I worry about and need to be handled as an emergency situations or need a hospital admission?
1. When a pt come with trismus : limitation of mouth opening.
2. Dyspnea: difficulty in breathing,breathlessness.
3. Presence of draining sinus,fistula.
* Airway compromise:
* Can be complete or partial.
* The respiratory rate can be increased or decreased
* We can use the pulse oximeter which is a device that measures the oxygen saturation in the blood by putting it in the patient’s finger, values below 60mm Hg (<94%) indicates that the hemoglobin is not saturated and the pt has hypoxia.
* **Management of patients with facial spaces infection:**
1. Medical support with correcting host defenses.
* Support:

The state of infection can cause physiologic load on the body.

Fever, dehydration, glycogen depletion and catabolic state.

Fever belowe 39.4 is probably beneficial; promotes phagocytosis, increase blood flow to the area.

1. Antibiotics.

If the pt is allergic to penicillin then give clindamycin

1. Surgical removal of the source ASAP.

Primary goal of surgery is to remove the cause of infection.

Secondary goal is to provide drainage of the accumulated pus and necrotic debris.

1. Incision/ drainage.

The aim of the incision and drainage is to allow the oxygen to inter the site of infection, and increase the blood supply to that area and that’s mean the antibiotics now can reach to that area, it decreases the load of bacteria by getting rid of the puss or any exudate, reducing the hydrostatic pressure that is compressing the tissues against the air ways.

If the infection involves **temporal space**s then do an incision in the temporalis muscle and use your fingers to allow the puss to come out and put the mosquito in the space. Then you need a drainage to prevent the incision from healing for the next 24 hours, and that’s by cutting a finger from the surgical gloves and put it inside then you stitch it.

For the **submasseteric space** involvement the incision is done at the angle of the mandible I go through skin, platysma, cervical fascia, and I go in, from there I can reach the sublingual space and we go to the medial aspect of the border. Be cautious of the facial artery.

1. Reevaluation.

If the pt didn’t get better in the next 48 hours then you know that you missed an infected space.

* **ludwing’s angina:**

Used to be a very fatal. As you know the temporomandibular spaces are connected to each other, sublingual and sub mandibular spaces as well, in ludwings angina the two sub lingual and two submandibular spaces will be affected, so the floor of the mouth will be raised and the tongue become raised against the palate, and this will suffocate the pt and cause trismus, the treatment is by incision and drainage but here we will need multiple incisions.

Joud Omari

Best of luck!!