OM \4th year , lec 17-20

**Orofacial pain**

 Pain is considered as a fifth vital sign

**History of pain**: **SOCRATIS**

|  |  |  |  |
| --- | --- | --- | --- |
| Relieving/radiating facto | Character | Onset | Site |
| Signs | Intensity | Treatment | Aggravating factors |

**Examination:**

-Local structures and medical history

-cranial nerves

 –psychological assessment

-pain mapping/questionnaires

**Investigation :**

-tooth vitality test. -temporal artery biopsy.

- X-ray. -chest X-ray.

-endoscopy. –ANA.

- MRI/CT. - Psychological assessment.

- ESR.

**Burning mouth syndrome:**

* can't be attributed to local or systemic factors (pt can't find any local or systemic factors to explain pain )
* Mostly **post menopausal females**
* strict **oral** location ( no pain other than orally )
* **Bilateral** **symmetrical** and not related to the nerve pathway
* Variable pain intensity (mild moderate sever)
* Sometimes associated by xerostomia and altered taste

2 types:

1. Primary: no cause

2. Secondary: we have a cause like Geographic tongue or iron deficiency anemia or B12 and folic acid deficiency

\*To differentiate between 1ry and 2ry BMS we have to exclude any doubt of systemic cause:

- Blood tests: Hb level, folic acid, Blood glucose level, urea, electrolytes

- Drug history (ex ACE inhibitors causes burning sensation)

 -Candida (Candidal swab),

- Sjogren's syndrome (xerostomia)

-allergic to of food or materials (uncommon)

**Mainly blood and urea and glucose and electrolytes**

Tx :

* **primary** : medications and psychological support

 Topical glunazepam , alpha- lipoic acid

Systemic medications like tricyclic anti-depressant (Amitriptyline

mainly) , opiods , diazepam may , selective serotonin re-uptake inhibitors,

gabapentin, capsaicin

* **secondary** : treat cause

**Trigeminal Neuralgia**

* Severe
* Sudden **unilateral**, recurrent stabbing pain
* It’s located exactly at the **distribution of one of the Trigeminal nerve branches**
* Mainly affects old age, 60 year olds.
* Electric shock in nature or like being burned.
* There’s a **trigger zone**
* the patient will not complain of paresthesia or anesthesia

2 types L

1. **Primary** : no cause , most common
2. **Secondary** : affect patient who have CBA (cerebellopontine angle), or it could be the first manifestation of multiple sclerosis.

**Investigations:**

* + blood tests: rarely helps in the diagnosis we make them to check the liver and the kidney function, because the medications might affect them
	+ MRI >> Most important to exclude any brain lesion or injury or local cause that might affect the nerve.
	+ sensory testing
	+ psychological assessment ( adverse psychological reactions may occur as a result of the pain (stress, depression, anxiety)

**Management:**

* Mostly by drugs : golden standard drug is **carbamazepine**
* Psychological therapy
* Surgery ( rare )

**post herpatic neuralgia**

caused by herpes zoster , after 3-6 months of the infection

at sights of the previous herpes zoster infection.

mostly in pts above 70 years old specially if they were immunocompromised

any pt complains of herpes zoster infection should take antiviral therapy to prevent PHN occurrence
\* if HN occurs pt is treated with ( carbamazepine )

 **Glossopharyngeal neuralgia**

most severe types of pain

pain in the throat or the neck or when the pt swallow

**Giant cell arteritis**
**\* temporal arteritis , Horton disease**

Unilateral or bilateral headache

histology: giant cells in the blood vessels
immune-mediated

It's a life threatening disease , it may cause blindness

Old females above 60 years old

 **investigations** :

1- **ESR & C-reactive protein are very important** here because they will be significantly elevated ( >50)
2- CBC ( detect anemia or thrombocytopenia)
3- elevated serum enzymes such as ( aspartate transaminase and alkaline phosphatase)
4- temporal artery ultrasound may appear thickened
5- temporal artery biopsy : uncommon because usually the lesion is skipping lesion , the deposition is not uniform on the vessel wall

**management** :

1- high doses of steroids (sometimes hospital admission is required for the IV steroids)
2- monitoring of the ESR >> every week we check its level
when the S&S disappear and the ESR level returned to normal the pt undergoes methotrexate orally (immunosuppressor)
3- folic acid to prevent the occurrence of anemia due to folic acid deficiency

**Migraine**

Always **Unilateral** , pulstile in nuture , moderate to severe

Aggrevated by routine physical activity + associated with autonomic featuers : nausea,vomiting,photophobia & phonophobia that are not attributed to any other disorder

-started in children , more in females

-**Management** :-

1-avoid triggering factors

2-Acute ( during pain attacks )

 Paracetamol, NSAID,caffeine

 Triptan or ergotamine ( seratonin Analog drugs)

 Antiemetic drugs (due to nausea & vomiting)

3-prophylactic: in patients with episode frequent pain (3 or 4 times/week)

4-Severe frequent pain not responsive to acute therapy :

Beta blocker, calcium channel blocker ,Antiepileptic agents , tricyclic antidepressant , Potocs therapy , Acupuncture

**Tension headache**

**Bilateral** , Pressing in nature , Mild to moderate

Not aggravating by routine physical activity (may be reduced by it) + No nausea, vomiting or photophobia

Managements:-

-pharmacological : paracetamol , NSAIDS

-Chronic type : response to tricyclic antidepressant , physical therapy , Acupuncture

**Atypical facial pain (persistent Idiopathic facial pain ) :-**

1- Pain in the face, daily, **persistant** , Confined at onset to **limited area** on one side , deep & poorly localized , not associated with sensory loss or physical signs

More in female, patients with certain type of personality

Management:-

1. doctor relationship is the most important aspect
2. reassurance and avoid excessive dentistry
3. Behavioral or psychological therapies
4. drugs : antidepressant is more effective in early stage

Seratonin analog , anexiolytic , anticonvulsant

**Fibromanalgis disease**

similar to chronic facial pain

Pain in all muscles in the body + facial pain

**hematological disorders**

Normal range of Hb :

Males: 13\_16 , Females : 12 \_15

The size of RBCs measured by mcv ((mean cell volume)

**There’s two causes of anemia :**

1. Decrease RBCs and Hb production :

\*Haematinics deficiency (*e.g*. *Iron, B12, Folate*)

\*Bone marrow failure (e.g. *a plastic anaemia*)

\* Bone marrow replacement (e.g. *leukaemia, lymphoma*)

\* Hypothyroidism

\*Chronic renal failure (e.g. *erythropoietin deficiency*)

1. Increase destruction or excessive loss of RBCs :

\*Acute haemorrhage

\*Chronic blood loss (e.g. *haemorrhoids, menorrhagia, peptic ulcer, cancer)*

\* Haemolytic anaemia

**Investigations :**

* Hb
* MCV
* RBC count
* Haematinics
* Disease specific investigations (Hb electrophoresis for thalassemia ) .

**1- Iron deficiency anemia**

clinical features :

* koilonychia (spoon-shaped nails)
* Dysphagia due to post cricoid webs ( Plummer-Vinson syndrome)with malignant transformation susceptibility
* Atrophic glossitis

 Diagnosis :

* Clinical features
* Decreased Hb, microcytic anaemia, decreased serum ferritin, high TIBC (total iron binding capacity )
* GI endoscopy

**2\_ VB12 deficiency**

Diagnosis :

* Clinical features
* Decreased Hb, macrocytosis,decreased B12
* In pernicious anaemia the cause is ((Auto-antibodies against gastric parietal cells and intrinsic factors which responsible for VB12 absorption .
* Schilling test (*radiolabeledB12 absorption test*)

**3\_ Folate deficiency**

Diagnosis :

* Clinical features
* Decreased Hb, macrocytosis, decreased serum level of red cell folate

**Haemolytic anemia**

* Increased destruction of RBCs is accompanied by compensatory bone marrow hyperplasia and increased production of reticulocytes (immature RBCs).

In addition, hyperbilirubinaemia causes jaundice

**Sickle cell anemia**

* In the deoxygenated state, HbS undergoes polymerisation within the erythrocytes which become rigid, sickle shaped and unable to pass through capillaries .

\*\*Sickle crisis( clustering of destructed RBCs inside the blood vessels and it becomes painful ), infarction (fever, malaise, acute pain)

Diagnosis :

* Family history
* Clinical features
* Decreased Hb, increased reticulocytes, sickled erythrocytes
* Hb electrophoresis (HbS)

**Thalassemia**

characterised by reduced rate of production of the alpha or beta globin chain in the haemoglobin molecule

* two types of thalassemia; alpha and beta

Beta thalassemia is the most common type

Diagnosis :

* Clinical features
* Decreased Hb, microcytosis
* Haemoglobin electrophoresis shows high HbF ( fetalHb)

**Dental aspects:**

* Patients with low Hb level are poor candidate for GA
* Elective oral surgical procedures should be avoided in severely anaemic patients (so if Hb<10 g/dl) because of increased risk of infection and impaired wound healing.
* **Oral manifestations** **of deficiency anaemia include:**
* Glossitis
* Angular chelitis
* Aphthous ulcers
* Burning/sore tongue
* Candidiosis
* Pallor of oral mucosa
* Postoperative pain is best managed by paracetamol not aspirin or NSAIDs for sickle cell anemia patients (because NSAIDs when metabolized will cause acidosis and this acids can trigger sickle cell crisis ).
* Iron overload in thalassemia patients may cause diabetes, pancreatic damage ,kidney failure and xerostomia due to the accumulation of iron in the salivary glands .
* Routine dental procedure can be carried out safely in thalassemia minor patients
* Antibiotics prophylaxis before surgical procedures is indicated in splenectomised patients and in patients with hypersplenism .
* Oral surgical treatment in thalassemia patients is influenced by three major problems ; anaemia, susceptibility to infection, and associated organ damage due to iron overload may cause diabetes , pancreatic damage ,kidney failure and xerostomia due to the accumulation of iron in the salivary glands .
* Patients who had repeated blood transfusion are at risk of blood born infections (HIV, HBV) and therefore represent a cross infection hazard .