Acute paralysis

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Acute asymmetric paralysis

1. poliomyelitis
2. non-polio viruses
3. pseudo-paralysis;
   Trauma, toxic synovitis
   Osteomyelitis
4. post- injectional paralysis

Acute symmetric paralysis

1. Guillain-Barre syndrome
2. post-diphtheric paralysis
3. botulism
4. transverse myelitis
5. spinal cord compression
6. with ataxia and chorea
7. rare causes e.g. rabies ,tetanus

Poliomyelitis

It is a viral disease of anterior horn cells. In under developed countries, it is still a cause of acute paralysis especially in unvaccinated or partially vaccinated infants and young children (3mo-3years). Paralysis is sudden (occur in few hrs), massive (involve lower limbs , trunk and other areas), flaccid (sever hypotonia), asymmetric more in one side and purely motor (no sensory loss).

Extension of paralysis ; spinal polio is the commonest form .

Asymmetric paralysis of lower limb is most common paralysis extends to affect trunk, upper limbs and neck.

Respiratory paralysis ; due to inter-costal and diaphragmatic paralysis may occur and can be life threatening.

In spino-bulbar poliomyelitis manifestation of bullar paralysis as weak cry , weak cough and nasal regurgitation are also present.

Stages of paralysis;

1. acute stage (1^{st} 2wks); during this stage the patient is infectious(isolation is important) and paralysis may extend to involve other areas(observation is important) As IM injection may precipitate more paralysis, it is important to avoid injection during this stage, this called(stage of pediatrician).
2. restoration stage ; (2wks-6months) during this stage gradual but incomplete recovery usually occur, physiotherapy 2-3 times per week is very important to prevent muscle wasting and deformities this is (stage of physiotherapist).
3. stage of residuals > 6months , no further improvement is expected and residuals as wasting, shortening and deformities usually occur in sever and neglected cases, this stage is stage of (orthopedic surgeon).

Management of poliomyelitis;

A) Acute stage;

1. isolation; especially from susceptible infants.
2. observation ; especially muscles of respiration, evaluation of degree o f involvement is important and blood gas analysis to be done to exclude central respiratory failure. In case of hypoxemia and co_2 retention, mechanical ventilation is life saving.
3. rest of the paralysed limb; should be kept in neutral position, absolute contraindication of intramuscular injection during the 1^{st} week of illness, passive movement of the limb is gradually allowed during the second week.

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B. stage of restoration ; physiotherapy 2-3 times per week is essential to prevent wasting and deformaties.
C. stage of residuals ; correction of residual shortening and or of deformities is the main line of treatment.

Guillain –barre syndrome
Post-infectious polyneuritis

It is the most common cause of acute paralysis in children, although infants can be affected too, most cases are seen in children more than 3years old. History of preceding viral infection one or two weeks before the onset of paralysis is usually obtained.
Important characteristic features are:
Acute symmetric paralysis ; paralysis started in the lower limbs and usually ascends over few days to involve the trunk and upper limbs (tetraplegia), affected limbs are flaccid and tendon reflexes are lost, respiratory paralysis may occur in some cases and necessitates hospitalization and mechanical ventilation for two weeks.
Bulbar paralysis occur in 50% of cases and lead to aspiration pneumonia.
Atypical forum of paralysis with descending nature (bulbar, respiratory then peripheral may occur). Other neurological findings , peripheral sensory loss is minimal or absent and autonomic disturbances as urinary retention and disturbances of vital signs may occur occasionally, initial disturbed consciousness for hrs or few days may occur.
The coarse is benign in most cases and gradual complete recovery usually occur over few weeks or few months. Relapse may occur in 10% of cases.
Confirmatory investigations ; include nerve conducting velocity decrease and CSF examination 2wks after the onset there is increase CSF protein only.
Management of acute G.B.syndrome ;
1. no isolation is required; as it represents an altered immune response, the lymphocyte become sensitized to myelin protein and then migrate to peripheral nerves and cause myelin break down.
2. observation ; at hospital , for involvement of respiratory and or bulbar muscle, intubation and mechanical ventilation for several weeks may be necessary and in bulbar paralysis N/G tube feeding is required.
3. in rapidly progressive and sever paralysis ;
Immunoglobulin IV 300mg /kg infusion over 6-8hrs dialy for 2-5days, others when treatment with immunoglobulin is not effective, other measures used as; corticosteroid prednisone 1-2mg/kg/day and with prolonged use alternate day treatment is used, plasmaphresis (5 cessions are usually needed with two days interval).
Immunosuppressive agents is other mode of treatment.
4. supportive treatment ;
As respiratory support , treatment of secondary infections and prevention of contractures by physiotherapy.

CNS INFECTIONS * Dr.Dawood Al-Azzawi

Causes of CNS infections;

A- meningitis

1- bacterial (septic) meningitis; e.g. pneumococci , meningiococci, H.influenza &others.
2- viral (aseptic) meningitis ; e. g. Enteroviruses, mumps & Herpes Simplex virus.
3- tuberculous meningitis
4- other infections, brain abscess, epidural abscess, cerebral thrombophlebitis.

B- Encephalitis

1- viral encephalitis ; e.g. Enteroviruses, Mumps & Herpes Simplex.
2- non-viral encephalitis; mycoplasma, fungals & protozoal.
3- para-infectious encephalitis (allergic) following infections &vaccine.
4- unknown causes(70%)

Clinical manifestations of CNS infections ;
Can be classified into two groups:
1- evidence of acute infection ; fever, anorexia, vomiting and irritability are usually present , high fever, toxemia and purpuric skin rash always suggest the diagnosis of meningococcemia.
2- evidence of acute CNS disease ; Infections of CNS presented clinically with one or more of the following (**5**) neurological findings;
a- altered consciousness ; lethargy→ deep coma , phobia , hallucination.
b- convulsion.
c- increased intracranial pressure ; sever persistent vomiting and or sever headache, in infants bulging of anterior fontanel is most important sign while in children bradycardia and increased blood pressure are suggestive.
d- features of meningeal irritation; in meningitis as neck rigidity , kernig's sign (is tested with the patient supine on the bed by passively extending the patient's knee when his hip is fully flexed, this movement cause pain and spasm of the hamstring muscle in meningeal irritation affecting the lower part of the spinal subarachnoid space).
e- lateralizing signs as focal paralysis, unequal pupil, facial or ocular paralysis.

Bacterial (septic) meningitis:

usually with abrupt onset with increasing fever and vomiting , the patient appears critically ill and marked toxemia . Convulsion and meningeal irritation are main neurological signs. In infants convulsions are more common , while features of meningeal irritation are less evident while in children it is the reverse.
In meningococcal meningitis ; high fever and toxemia with purpuric skin rash be the main presenting sign(meningococcemia), the coarse is fulminate and unless urgent proper management is started death or serious neurological sequels occur.

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Viral (aseptic) meningitis:
It is with acute onset , mild to moderate fever, vomiting, but the patient does not look so critically ill as those with bacterial meningitis, mild meningeal irritation (neck rigidity) is the only neurological finding, convulsions are rare in absence of encephalitic elements. Preceding or concomitant exanthema as (herpes simplex or chicken pox)is present, mumps is also an important cause. Generally the coarse is usually benign and complete recovery is the rule.

Encephalitis or meningoencephalitis;
Usually of acute onset of fever and vomiting , abnormal bizarre behavior and or altered consciousness are the most evident neurological findings. In some cases the presenting signs, fever and coma are the only neurological findings, thus the clinical diagnosis can be made. In cases of meningoencephalitis ; convulsion and increase intra cranial pressure are present and clinical differentiation from bacterial meningitis is difficult. Preceding or concomitant exanthema also present, in mycoplasma encephalitis a preceding lobar pneumonia is usually present. In allergic encephalitis history of exanthema or vaccine few weeks before the onset of the illness can be obtained.
The coarse is extremely variable, ranging from rapid complete recovery within few days to a prolonged coma, and sever neurological sequels or death.

Tuberculous meningitis;
The onset is insidious and characterized by non-specific features as mild fever, irritability , vomiting and conistipation and remain for about one week before appearance of neurological manifestations. So tuberculous meningitis is usually not confused clinically with bacterial meningitis or viral meningoencephalitis.
This illness can be divided into three stages, each for 1-2 wks.
Stage 1 (prodromal stage) ; non-specific manifestation of infection.
Stage 2 (transitional stage); convulsion and meningeal irritation and cranial nerves palsies as ocular and facial paralysis.
Stage 3 (terminal stage); hyperpyrexia and deep coma, death is usually occur if patient reaches this stage.
Other manifestations suggesting tuberculosis as weight loss, lymphadenopathy or chronic chest disease be evident. With early diagnosis the patient during first stage and early 2nd stage and proper treatment recovery is possible with minimal sequels.

Brain abscess;
This uncommon infection is characterized by ;
Onset is usually insidious over several days with fever and vomiting.
Increased intracranial pressure (headache and vomiting)and lateralizing signs (focal paralysis are main neurological findings).
Preceding or concomitant bacterial infection as septicemia, empyema or otitis media may be present , polycythemia as in cynotic congenital heart disease is sometimes the main predisposing factor
The coarse is usually prolonged over weeks and if rupture occur fatal meningitis occur, diagnosis by clinical suspicion and brain CT is indicated.

Diagnosis of CNS infections;

A- CSF examination ; includes ; increased pressure, cells and protein with decrease sugar, culture for bacteria.

The contraindications for an immediate lumber puncture include;

1. evidence of increased intracranial pressure (other than bulging anterior fontanel) such as 3rd or 4th cranial nerve palsy, depressed level of consciousness or increased blood pressure with bradycardia and with respiratory abnormalities.
2. sever cardiopulmonary compromise.
3. infection of skin overlying site of lumber puncture.
4. thrombocytopenia.
5. ophthalmological examination for papillodema is necessary

B-other investigations; as (CBP&ESR, CRP) to rule out bacterial from viral causes and blood culture, urine culture, blood culture. Tuberclin test and chest x-ray. CTscan of the head in suspected cases of brain abscess and in the presence of localizing signs.

Complications and sequel of CNS infections;

1. death ; fulminate bacterial meningitis, encephalitis, untreated tuberculous meningitis and ruptured brain abscess.

2. blindness and deafness.

3. neurological complications as ; mental retardation , hydrocephalus, organic epilepsy, motor weakness and bilateral subdural effusion.

In every case of CNS infection and after recovery periodic neurological assessment of mental development, motor development and head circumference measurement are essential.

Treatment of CNS infections.

Once the diagnosis of CNS infection is made or suspected , hospitalization is necessary for observation , urgent investigations and management;

1- antibiotic; until bacterial cause is excluded all patients with CNS infection should receive vigorous antibiotic treatment.

Recommended empirical treatment is (vancomycin 60mg/kg/day + either 3rd generation cephalosporins e.g. cefotaxime 200mg/kg/day 6hrly or cefotriaxone 100mg/kg/day once or twice/day). If patient is allergic to B-lactum antibiotic he can be treated with chloramphenicol 100mg/kg/day 6hrly. Then antibiotic given according to cultureand sensitivity result. The period of treatment depends on the type of bacteria.

Strepto. Pneumonia (10-14 days) , H.influenza (7-10 days) , N. Meningitis(5-7 days)

In case of brain abscess antibiotic treatment should be continued for 6weeks and neurosurgical consultation for possible drainage is important.

In case of tuberculous meningitis triple drug therapy with INH, rifampicin and pyrazinamide is indicated with steroid for 9months. Antiviral treatment may be used e.g. IV acyclovir for herpes simplex.

2- supportive treatment ;

Oxygen is comatose patient with N/G tube for > 2-3 days ,

IVF 2/3 of maintenance due to (inappropriate ADH secretion and cerebral edema)

Care of comatose patient with (fluid, nutrition, prevent bed sore and decrease intracranial pressure.

3- control of convulsion by diazepam, lorazepam, phyenytoin and Phenobarbital.
Treatment of increase ICP by; intubation for hyperventilation and IV fursemide and mannitol. Steroids as dexamethasone 0.6mg/kg/day for 2days in meningitis due to H.influenza and contaversial in others.

4- heath precautions;

Patient with bacterial meningitis are not infectious after 24hrs of treatment.(respiratory isolation for 24hrs).In contrary to what is thought by many people CNS infections are not highly infectios , in close contact the possibility of being infected , infectivity rate is not more than 1%. Chemoprophylaxis is recommended for all close contacts of the patient with mennigococal meningitis regardless of age or immunization state by rifampsin 10mg/kg/dose every 12hrs maximum dose 600mg fo 2days, rifampsin prophylaxis should be given to all household contacts including adult, if any close family member younger than 48months has not fully immunized or if an immunocompromised child regardless household.

5- follow up after recovery;

In case of recovery, several neurological sequel, periodic follow up is essential.
Mental retardation, epilepsy and motor weakness occur, regular periodic measurement of head circumference is important in infants to exclude post-meningitis hydrocephalus and chronic subdural effusion.