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Review Article

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MANAGEMENT OF HEPATIC ENCEPHALOPATHY WITH SUBDURAL HEMATOMA BY AYURVEDA

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ABSTRACT

Hepatic encephalopathy is an altered level of consciousness as a result of liver failure. Onset may be gradual or sudden. Other symptoms may include movement problems, changes in mood, or changes in personality. In the advanced stages it can result in a coma. Hepatic encephalopathy can occur in those with acute or chronic liver disease. Episodes can be triggered by infections, GI bleeding, constipation, electrolyte problems, or certain medications. The underlying mechanism is believed to involve the buildup of ammonia in the blood, a substance that is normally removed by the liver. The diagnosis is typically made after ruling out other potential causes. It may be supported by blood ammonia levels, an electroencephalogram, or a CT

scan of the brain. The symptoms of hepatic encephalopathy may also arise from other conditions, such as cerebral hemorrhage and seizures. In a small proportion of cases, the encephalopathy is caused directly by liver failure; this is more likely in acute liver failure. The mildest form of hepatic encephalopathy is difficult to detect clinically, but may be demonstrated on neuropsychological testing. It is experienced as forgetfulness, mild confusion, and irritability. The first stage of hepatic encephalopathy is characterized by an inverted sleep-wake pattern (sleeping by day, being awake at night). The second stage is marked by lethargy and personality changes. The third stage is marked by worsened confusion. The fourth stage is marked by a progression to coma. A male patient named XYZ of 65yrs old came in emergency with the diagnosis Hepatic Encephalopathy with subdural hematoma, the case is as follows-

KEYWORDS:

Case

PATIENT NAME- XYZ

AGE - 65 yrs. SEX- Male

Occupation- Retired Reg. No. E/4104/3097

D.O.A-12/07/2017 D.O.D-24/09/2017

C/O

o Irrelevant talk all complaints

O Unable to stand without support Since 15 to 16 days

Unable to walk with support also

- Generalized weakness
- Sleeplessness
- o Irritability

No H/O- Malaria /Typhoid /Dengue.

K/C/O- DM -since 2 yrs

On Tab- Glimeperide 1mg ½ OD in morning before meal.

H/O – ICU admission for 10 days before 8 days.

- o Blood transfusion was done 4 bottles as he was diagnosed with megaloblastic Anemia.
- o Also, he was suffering from severe jaundice which was treated in ICU.

No K/C/O- HTN/PTB/BA/Epilepsy.

No H/O- Any Surgical illness.

No H/O- Any Drug Allergy.

O/E- on admission

GC- Moderate & Afebrile

P-96/min BP-150/80 mm of hg

Bed sores on hip joint.

BSL - 36 mg/dl

S/E- RS- Lt. sided crept.

CVS-S1 & S2 Normal

CNS-semiconscious & semi oriented

Rtvc Rtds

Pupils: Rt-Normal size and reacting to light

Lt-Normal size and reacting to light

• Planters-Bilateral flexor

P/A- soft & distended

Urine-catheterized

Stool-Passed

Investigations- 12/7/2017

Hb-11.1%; RBC-3.59; WBC-19.17;

SGOT - 23.40, SGPT- 21.20, Total bil. - 1.04

Direct Bil. -0.59, Indirect Bil. -0.45

Sr. Creat. -1.37, Sr. Ca. -7.84

Sr. Uric acid – 8.70

RA -Negative, VDRL, HbsAg -Negative

CT Brain (29/6/2017) – Thin bilateral hypodense subdural collection of 2-3 mm thickness of rt. Side resp. Diffuse cerebral atrophy. Midline shift.

USG (abdo/pelvis) – (29/6/2017) – Excessive bowel gases.

Treatment Given

When patient was admitted in ipd, his condition was moderate, it took 2-3 days to normalize the patients condition for ayurvedic treatment and after that treatment was started –

| 0 | Oral: - Punarnavashtak kwath | 30ml BD |
|---|---------------------------------------|---------------|
| 0 | -Tab. Smrutisagar rasa. | 2-2-2 |
| 0 | -kutaki kwath | 30ml BD |
| 0 | - Cap -Indukant ghritam | 1-0-1 |
| 0 | -Cap – Ksheerbala (101) avartani | 1-0-1 |
| 0 | - Baladi Kshirpaka | 30ml BD |
| 0 | (Bala, Atibala, AShwagandha, Kapikach | u, Shatavari) |

Dietary supplement.

Panchakarma

- Dressing of bed sores done regularly.
- Shatadhuata ghrita for local application over bed sores.
- Mahamarichyadi tail for sarvang snehana.

- Patrapottali sweda with kottamchukadi tail n kottamchukadi choorna along with aswagandha and bala choorna.(14 days).
- Annalepana was done over left lower limb. (14days).

After Treatment

Catheter was removed. Patient was able to walk with the support of walker. Patient had sufficient sleep at night. There was no irrelevant talk. Patient was discharged with oral medicines at home and regular follow up was taken.

DISCUSSION AND CONCLUSION

In this case, the patient when came in our hospital, he was in first stage of hepatic encephalopathy. From ayurvedic point of view, this stage in that patient was considered to be - one reason is vardhakyajanya awastha, and there was vitiated rasa and raktavaha srotasa. Due to vitiation of rasa dhatu, the ahararasa not formed in proper form and quantity, also the rakta dhatu got formed in excessive amount and there was kshaya of further dhatu. Also, the another cause of dhatukshaya was vardhakya avastha. In this case, the rakta dhatu got formed in excessively and liver is the mulsthana of rakta dhatu and there was functional vikruti of liver no anatomical vikruti confirmed by ultrasound. As, the patient was catheterized, the poonarnavashtaka kwath was given which increases the proper renal function. Only the lipids can cross the blood brain barrier, as majja dhatu is one of the four snehas, the treatment was given accordingly. Kutaki kwath was given, kutaki is the most effective drug on liver. Its function is bhedana at cellular level and pitta rechana. Due to pittarechana there was elimination of excessive blood. It also maintains the proper function of liver. The another name of kutaki is tikta, it is explained shreshtha in tikta rasa and pachnarth due to this rasa dhatu is formed in its own quantity and quality. The panchakarma was given as balya for mansa and asthi dhatu. Thus, it was concluded that ayurvedic treatment is very useful in first stage of hepatic encephalopathy where liver is anatomically normal.

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