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Case Report

Phenobarbital Treatment For Malignant Infarcts: A Case Report

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Summary

Malignant infarct is a well established cause of morbid raised ICP. The mainstay of management remains osmotic diuresis and hyperosmolar therapy. Hyperventalation, hemicraniectomy and hypothermia also have good outcomes. Phenobarbitals are used in cases of refractory raised ICP. We present a case of malignant Rt. MCA infarction where phenobarbital, instead of well established agents like pentobarbital and thiopental was used. The aim was to prevent cytotoxic edeme within 48 hrs of ictus. The desired effects of phenobarbital was noted on repeat CT scan with restricted perilesional edema, without any significant drop in GCS. Neurological examination was possible, which is not possible with pentobarbital and thiopental.

Keywords: malignant infarct, raised ICP , cerebral edema, barbiturate coma,phenobarnbital

Introduction

Barbiturates, mainly pentobarbital is the drug of choice for treatment of refractory raised ICP, there are also reports of thiopental sodium being used to manage raised ICP. Raised ICP in ischemic infarcts occur due to malignant infarcts.[1] Any infarct > 50 % of MCA territory or > 145ml (Kothari's formula) can be predicted to behave malignantly.[2] Malingnancy implies to the cytotoxic edema that surround the infarct area. This is disruption of Monroe Kelly doctrine, causes raised ICP and presents as raised ICP leading to herniation. Numerous agents like mannitol, hypertonic saline, hyperventilation,CSF diversion and hemicraniectomy have been used to treat raised ICP of malignant infarcts. Barbiturates are recommended at the end of protocols when raised ICP is deemed refractory. We present a case of malignant right MCA infarct which was managed with barbiturate; phenobabital to prevent the edema which is maximum about 48 hrs from the onset of infarct.

Case Presentation

A 65 yr old diabetic male patient presented with sudden decrease in sensorium and preferential movement of right extremities. The GCS of the patient was E3V34M5 and the pupils were isocoric. A MRI DWI (Fig:1) was performed which revealed > 50 percent of right MCA infarction. The patient was admitted to the NCCU(NeuroCritical Care Unit). He maintained the blood gases and he was put on nasal O2, not requiring intubation. A single dose of maniitol 150 cc, atorvastatin and other medicines were given. Asprin was held for 2 days as it may aggravate HT (Hemorrhagic Transformation) which has a higher occurance in the first 48 hrs . Along with the above medicines he was also given single dose of 400 mg of phenobarbital followed by 100 mg q8 hrs intravenously.

The sensorium of the patient decreased, GCS (E3V3M4) with marked hemiparesis of right side for the next day. However the vitals were stable. 48 & 90 hrs post ictus a CT scan (Fig:2) was done which showed a

Figure

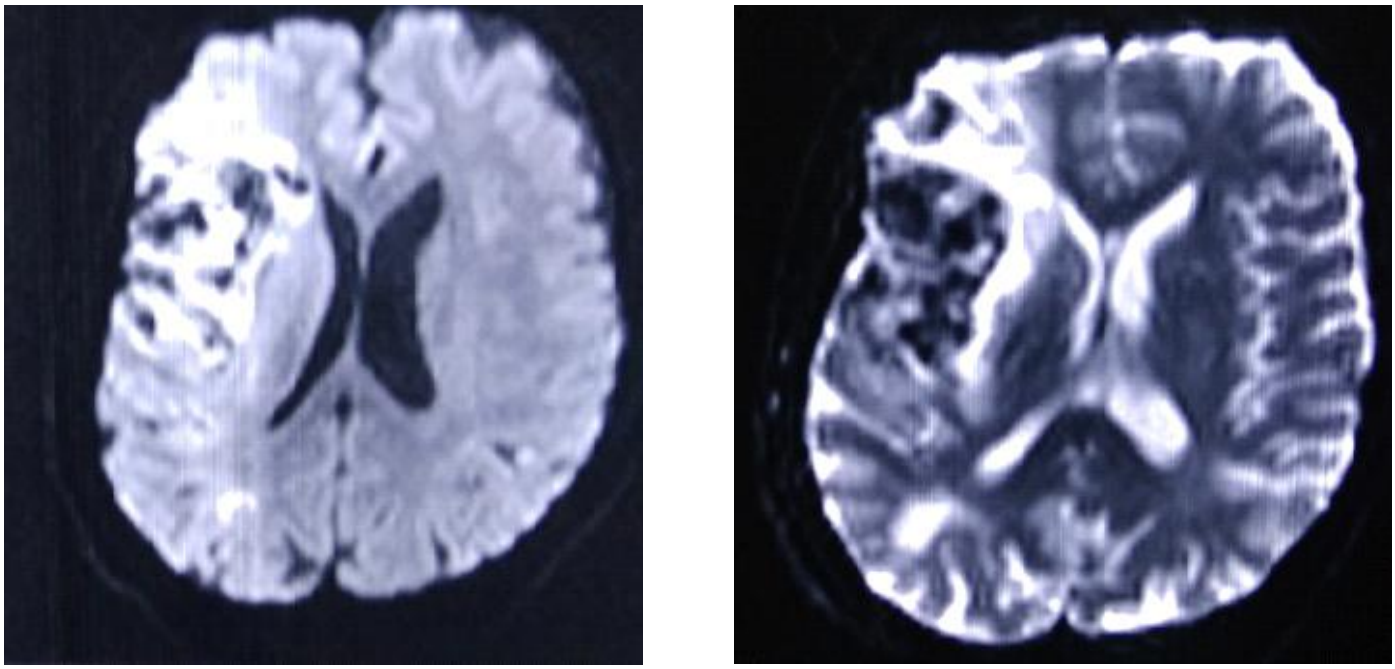

Fig.1 : MRI –DWI ADC showing Rt MCA malignant infarct; 6 hrs post ictus

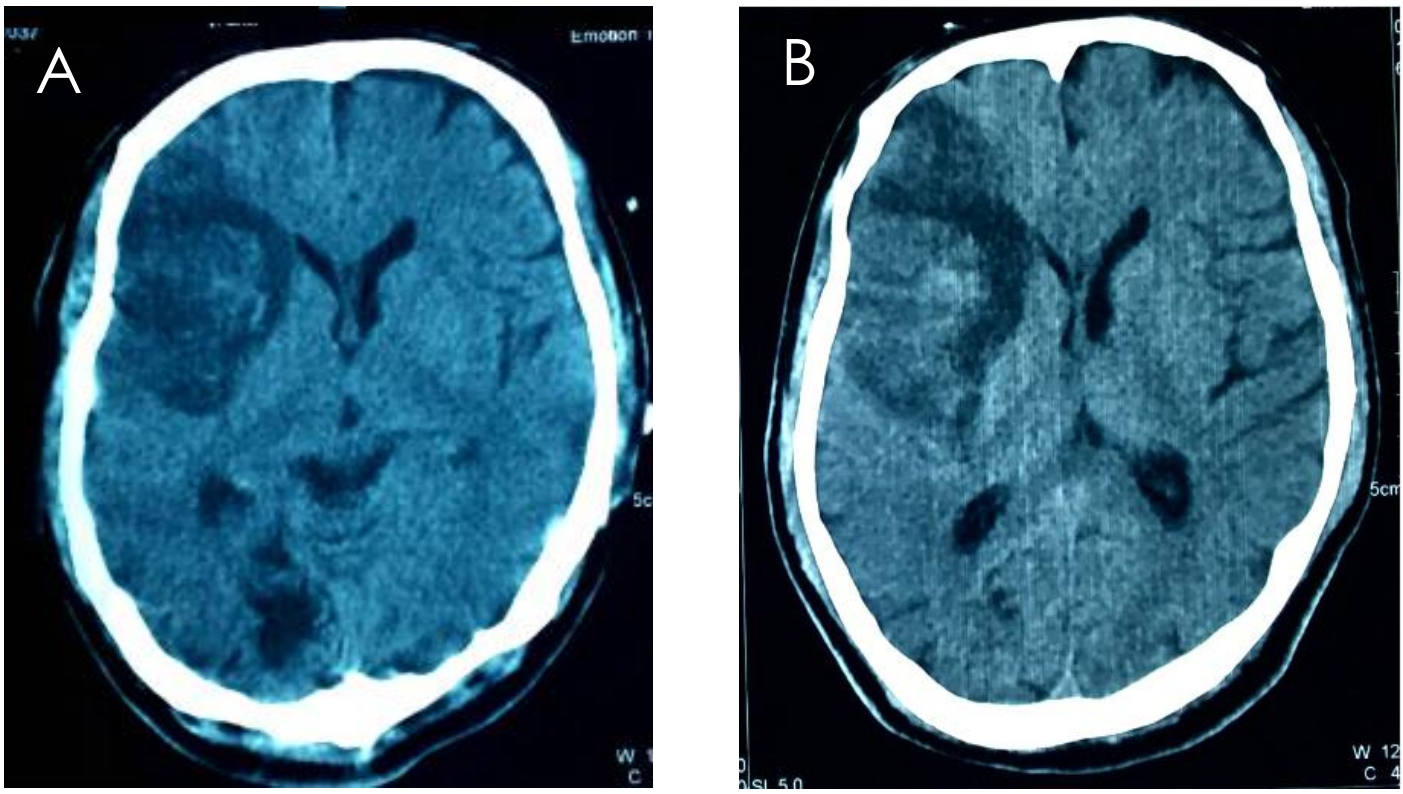

Fig.2 : CT Scan (A) 48 hrs after ictus, (B) 90 hrs after ictus

stable infract with minimal edema and stable midline shift, similar findings were noted on subsequent scans. Four days later the patient was shifted to ward with E4V4M6,right hemiparesis, and the NCCU stay was uneventful. After fifteen day the power of right hemiparesis was 4/5 and resolved completely after a month, with mRS0.

Discussion

There are numerous mechanisms of cerebral edema, cytotoxic edema is caused by cellular injury secondary to ischemia, as a result there is cellular edema which accounts to malignant effect on the cerebral compliance. As the volume of edema increases it causes raised ICP leading to undesired effects, most notoriusly herniation.[3] Pentobarbital and thiopental are well established agents used to treat raised ICP.[4,5,6]High doses of barbiturates have been used in past with remarkable reductions in ICP, with favourable outcome.[7] Notebly both require intubation and ventilatory support. Phenobarbital is a well established antiepileptic prescribed in OPD(outpatient department) as an oral agent. The same can be used intravenouly with out the warranted ventilatory support.

In this case phenobarbital was chosen as the patient was awake and ventilatory support was not indicated. A >50 % of MCA infarct was a certain predictor of the malignant imminent, hence it was decided to start barbiturate to prevent edema and not to wait for it may become refractory. Low dose (400mg) of phenobarbital was loaded, suboptimal by loading standards of seizures and a manitainance doses (100 mg qid)were administered(weight of patient 58 kg) . Thus over sedation was avoided at the same time not requiring intuabation as with the other two barbiturates. Pentobarbital limits neuroexamination allowing only celiocolic reflex and thiopental also limits neuroexamination. However phenobarbital, provides a broader range of neuroexamination and all patients might not require ventilatory support. After 48 hrs we started tappering phenobarbital and it was stopped after 7 days.

Conclusion

This is an anecdotal case of prevention of cytotoxic cerebral edema following admistration of a lesser barbiturate used for ICP reduction, however the effect and outcome is remarkable and well noted. We suggest more studies to validate phenobarbital for prevention of raised ICP in malignant edema rather than for managemnt of refractory edema, with out ventilatory support and with a broader window for neuroexamination.

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