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A modified peritoneal shunt for the management of hydrocephalus in a pup

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Abstract

A three-month-old male Shih Tzu pup was presented to the University Veterinary Hospital, Kokkalai with the complaint of dome–shaped head, ataxia, abnormal behaviour and vocalisation. On physical examination and diagnostic imaging, it was confirmed as a case of hydrocephalus. A 5FG infant feeding tube was used to shunt the cerebrospinal fluid from the subarachnoid space into the peritoneal cavity. It was managed on post-operative antibiotics, analgesics, antiseizure medication and supplements. Upon worsening of clinical symptoms, the pup was euthanised three months later and subjected to detailed post-mortem and histopathological studies.

Keywords: *Hydrocephalus, modified peritoneal shunt, pup*

Hydrocephalus is a condition characterised by the distension of ventricular system of the brain caused by obstruction of flow of cerebrospinal fluid (CSF) from its point of production to its point of absorption. It can be congenital or acquired due to neoplasia or inflammatory lesions (Thomas and Narak, 2017). Medical therapy aims to reduce the production of CSF and utilises drugs like acetazolamide, furosemide, omeprazole and prednisolone (Thomas and Narak, 2017; Dewey and Fossum, 2019). The only definitive treatment is a surgical procedure to divert CSF, most done by a ventriculoperitoneal shunt. This case report describes the use of a 5FG infant feeding tube to shunt CSF from the subarachnoid space to the peritoneal cavity and its outcome.

A three-month-old male Shih Tzu pup was presented to the University Veterinary Hospital, Kokkalai with the complaint of enlarged skull, ataxia, abnormal behaviour and vocalisation. On physical examination, the skull was dome shaped with an open fontanelle and had bilateral squint (Fig.1). It was exhibiting neurological signs like abnormal coordination of limbs with occasional star gazing posture. It was diagnosed as a condition of hydrocephalus which was confirmed on radiography (Fig.2). Ultrasonography using a high-frequency probe (10 MHz) revealed the distended ventricles (Fig.3).

The dorsum of the pup was prepared for aseptic surgery under general anaesthesia. It was premedicated with inj. butorphanol at the dose of 0.1 mg/kg and inj. midazolam at the dose of 0.1 mg/kg. Induction was achieved using inj. propofol calculated at 2 mg/kg and administered “to effect”. The pup was intubated and anaesthesia was maintained

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Fig.1. Dome shaped head with bilateral squint

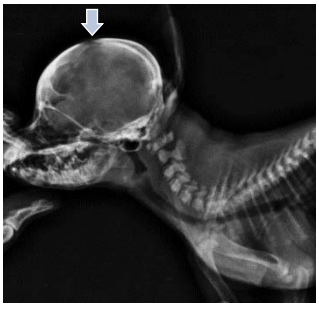

Fig. 2. Radiograph showing dome shaped skull and open fontanelle (arrow)

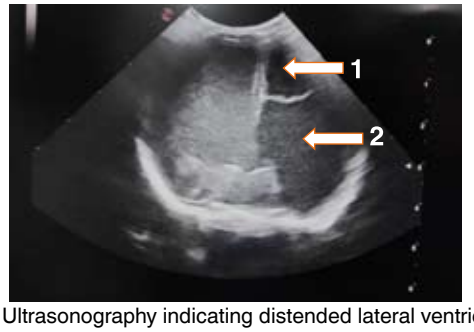

Fig. 3. Ultrasonography indicating distended lateral ventricle (1) and hypoechoeic area indicating fluid accumulation in the cerebrum (2)

on isoflurane (1-1.5% in 100% oxygen) using an open circuit. A midline linear incision was made on the head to expose the cranium (Fig. 4a). A hole was drilled on the calvarium lateral to the sagittal suture on the parietal bone at an angle of 30° using a dental scaler (Fig. 4b) just enough to accommodate an 5FG infant feeding tube, until the cerebrospinal fluid was flowing out freely (Fig. 4c). The fenestrated tip of the tube was inserted into the subarachnoid space and tilted laterally to a depth of two centimetres to confirm the flow of cerebrospinal fluid (Fig. 4d). The infant feeding tube was fixed on the cranium with interrupted sutures using fine nylon (Fig. 4e).

The length of the infant feeding tube was measured over the dorsal line of the body to the abdominal region to leave at least 10 cm of the fenestrated free end of tube inside the peritoneal cavity. The excess length was cut off. A subcutaneous tunnel was created from the cranial incision using a 3 mm Steinmann's pin in two stages (Fig. 5a) and the infant feeding tube was drawn through it using an artery forceps to a level, caudal to the costal arch on the left flank, where it was inserted into the peritoneal cavity through a nick incision on the abdominal wall (Fig. 5b) and secured with interrupted sutures (Fig. 5c). The skin

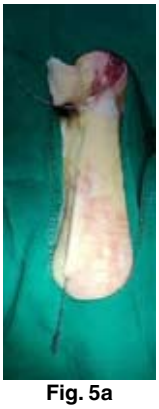

Fig. 5a

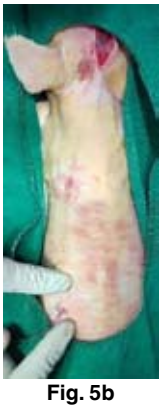

Fig. 5b

Fig. 5c

Fig. 5d

Fig. 5a: Subcutaneous tunnel created using 3 mm Steinmann's pin; **5b**: The infant feeding tube drawn through the tunnel and introduced into the peritoneal cavity; **5c**: Securing the infant feeding tube to the abdominal wall; **5d**: Skin sutures completed.

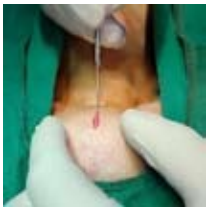

Fig. 4a

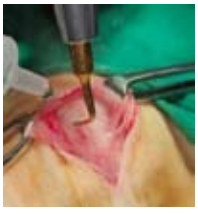

Fig. 4b

Fig. 4c

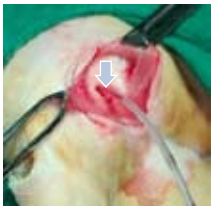

Fig. 4d

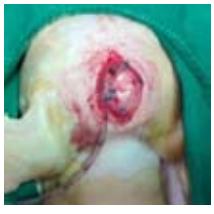

Fig. 4e

Fig. 4a: Skin incision; **4b**: Hole being drilled on the calvarium using dental scaler; **4c**: CSF flowing through the hole; **4d**: The infant feeding tube placed into the subarachnoid space and the flow of CSF confirmed; **4e**: Infant feeding tube fixed on the cranium with interrupted sutures.

incisions were closed in a routine manner (Fig. 5d).

Post-operative radiographs were taken to confirm the placement of the infant feeding tube (Fig. 6a and 6b)

Fig. 6a

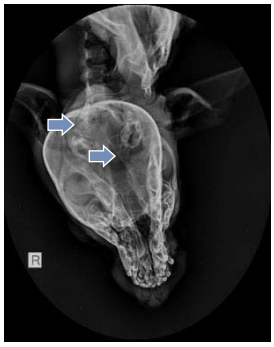

Fig. 6b

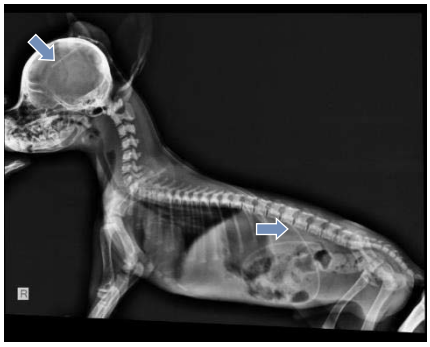

Fig. 6a: Dorso-ventral view of skull and **6b**: Lateral radiograph showing the placement of the infant feeding tube.

Post-operatively, the pup was maintained on inj. ceftriaxone @ 25 mg/kg iv, inj. ranitidine @ 1 mg/kg iv and inj. Nurokind @ 0.1 mL im for five days. The neurological symptoms were managed with syp. phenobarbitone @ 1 mg/kg per os and syp. levetiracetam @ 20 mg/kg per os. Advised syp. furosemide @ 3 drops bid per os, susp. meloxicam @ 2 drops od per os and multivitamin drops @ 5 drops bid per os. The skin sutures were removed on the 10th post-operative day. The puppy showed improvement in the neurological symptoms from the second week with reduction in squinting and overall size of the head. Then on, the clinical symptoms worsened, with vocalisation and uncontrolled movements of the head. Two months later, the pup was euthanised with the consent of the owner and subjected to detailed postmortem.

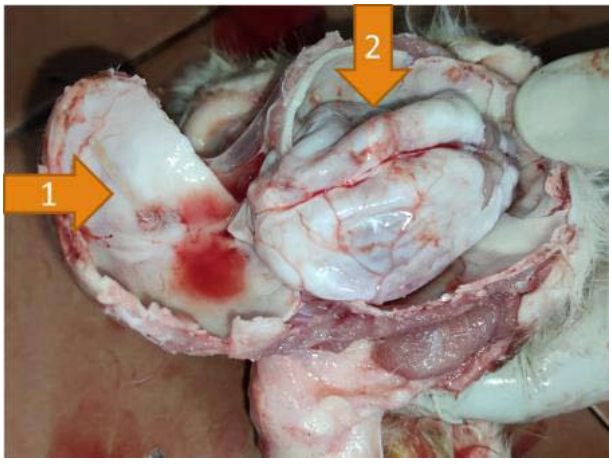

Fig. 7. Skull opened: 1. Intracranial portion of the infant feeding tube. 2. Collapsed brain with thicker meninges, fluid filled ventricular system and atrophied brain tissue

At postmortem, the animal was skinned to expose the subcutaneous infant feeding tube. It was intact and functional with the presence of CSF. On opening the skull, there was a gush of CSF indicating the presence of excess CSF inside the cranium. The portion of the infant feeding tube inside the cranium was also intact and atrophy of the cerebral tissue was noticed (Fig. 7). That correlated with the sonogram wherein, hypoechoic areas were observed in the cerebrum. The dura was thickened with excessive CSF flowing out from the distended ventricular system upon dissection (Fig. 8).

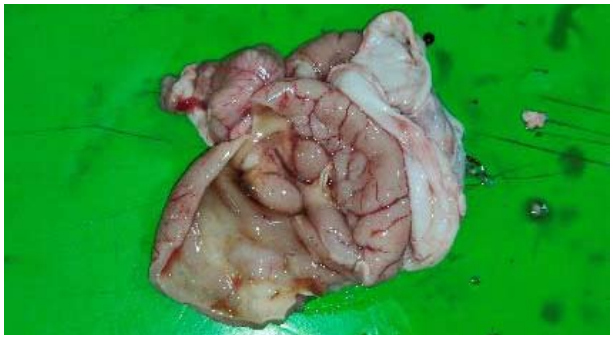

Fig. 8. Gross examination of the brain: Thick meningeal membranes overlying the atrophied brain. The tissues of the brain limited to cortical part with engorged blood vessels. The CSF was drained from the distended ventricular system.

Histopathological examination of the brain tissue was performed to observe loss of neuropil in the cerebral tissue (Fig. 9 and 10).

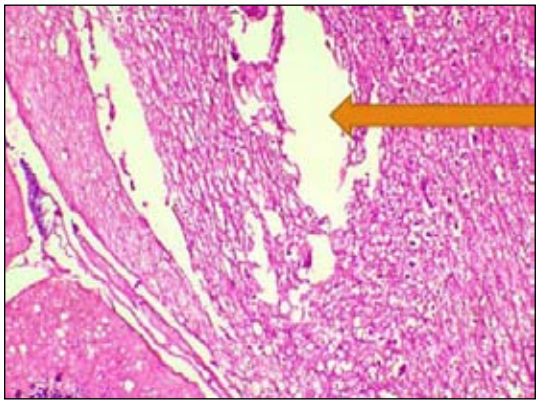

Fig. 9. Subcortical periventricular white matter. Extensive loss of neuropil, dense fibrillary astrogliosis, and cavitation were observed (H&E, 40X)

Congenital or paediatric hydrocephalus is common in toy and brachycephalic breeds of dogs (Thomas and Narak, 2017; Dewey and Fossum, 2019). The present case also falls in this category of dog breeds.

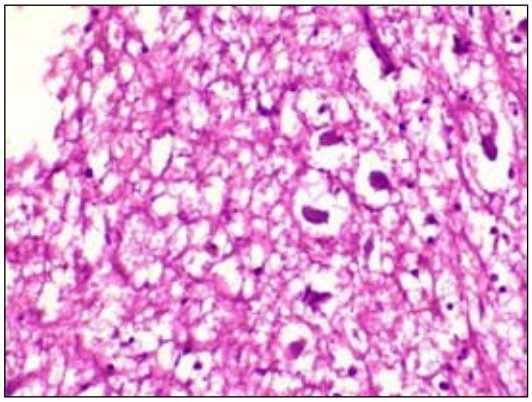

Fig. 10. Subcortical cerebral area. Necrosis of the axons, spongiosis and loss of neurons (H&E, 100X)

The clinical signs of hydrocephalus included an enlarged dome shaped head, persistent fontanelles, ventrolateral strabismus and neurological deficits including abnormal behaviour, cognitive dysfunction, ataxia, circling, blindness, seizures and vestibular dysfunction (Thomas and Narak, 2017). Similar signs were observed in the present case and in addition, there was vocalisation. Przyborowska *et al.* (2013) had reported that hydrocephalic animals exhibited altered mental states ranging from depression to hyperexcitability in addition to the symptoms mentioned above. During the initial presentation, the pup was more depressed but as the condition progressed it was hyperexcitable towards the end, may be due to the damage to the brain tissue caused by the accumulation of CSF.

The clinical signs and diagnostic imaging help to confirm cases of hydrocephalus. The diagnostic modalities usually employed are radiography, ultrasonography, CT and MRI. Radiography was helpful in demonstrating the abnormal size of the cranium and persistent fontanelle. But Przyborowska *et al.* (2013) considered it superfluous since any skull malformations are readily apparent. Ultrasonography was useful in detecting ventricular enlargement and was possible due to the persistent fontanelle. Thomas and Narak (2017) and Dewey and Fossum (2019) had supported this finding. Przyborowska *et al.* (2013) opined that ultrasonography had the advantage of being a non-invasive method to assess ventriculomegaly and to monitor the changes over time, without anaesthesia. Also, they had mentioned the use a high frequency probe to visualise the ventricles.

Medical management of hydrocephalus is conventionally aimed at reducing the production of CSF. The commonly used drugs include, glucocorticoids, diuretics and omeprazole. But as suggested by Przyborowska *et al.* (2013) and Scarpante *et al*., (2013) these drugs provided only temporary relief and surgical management is the treatment of choice. The standard surgical technique for managing hydrocephalus is ventriculoperitoneal (VP) shunting described by various authors (Przyborowska *et al.,* 2013; Scarpante *et al*., 2013 and Orlandi *et al*., 2020). The technique involved the placement of commercially available shunt systems with differential pressure valves, to drain the CSF from the ventricles into the peritoneal cavity. The surgery involved the use of bipolar cautery to coagulate the dura and cortex before introduction of the shunt, an anchoring clip or additional holes drilled on the skull for anchoring the shunt with sutures and a subcutaneous shunt passer to aid in placement of the shunt into the peritoneal cavity. In the present case the procedure was modified at all these levels to utilise available equipment for creating a practical solution with minimum expense. A 5FG infant feeding tube which lacked a valve system, was used in this case instead of the commercially available shunts. Electrosurgery was not necessary since the infant feeding tube was placed only into the subarachnoid space. Anchoring of the infant feeding tube was done by creating a loop which was then secured using nonabsorbable suture on to the cranium. A Steinmann's pin was used to create the subcutaneous tunnel instead of the shunt passer. Post operative supportive therapy was instituted to provide symptomatic relief and prevent complications. But, as the clinical symptoms worsened, in the interest of the welfare of the animal it was euthanised with the owner's consent.

On necropsy, even though, the infant feeding tube was in place and functional, the gross changes in the brain that included the thickened dura mater, excess CSF and atrophied cerebrum indicated that there was a substantial drainage deficit of the CSF despite the lack of a controlling valvular mechanism. The complications associated with VP shunting included obstruction, disconnection and migration, over-drainage and infection (Thomas and Narak, 2017). But in the present case, either, over production or under drainage, which could not be quantified, seemed to be the cause for lack of clinical improvement. Kent *et al.* (2016) had described the necropsy findings in two dogs that were diagnosed with hydrocephalus. The gross and histopathological observations differed from the present case in that the authors reported a normal dura mater and a normal brain parenchyma with inflammatory changes in the choroid and periventricular area. Histologically, in the present case, there was extensive loss of neuropil, dense fibrillary astrogliosis and cavitations, in the periventricular white matter and spongiosis and axonal necrosis in the subcortical cerebral tissue.

Summary

The present case report described a modified procedure for peritoneal shunting of cerebrospinal fluid for the management of congenital hydrocephalus. It was a cheaper alternative to the commercial ventriculoperitoneal shunts. The surgical procedure was successful and the functionality was confirmed at necropsy. Clinical improvement was not achieved in this case due to drainage deficit of CSF and the cortical damage had progressed beyond recovery. Case selection based on advanced diagnostic imaging might improve the success rate of such procedures.

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