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Basal cisternostomy for severe traumatic brain injury: illustrative case

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BACKGROUND Basal cisternostomy (BC) is increasingly utilized for intracranial pressure (ICP) management in severe traumatic brain injury (TBI), particularly by neurosurgeons in some high-incidence regions like India and China. It is performed as an adjunct to decompressive craniectomy (DC) or as a stand-alone procedure with bone flap replacement if brain laxity permits after basal cisternal drain placement. Emerging research from regions where this method is used supports its efficacy for ICP control.

OBSERVATIONS A 17-year-old male was involved in a severe road traffic accident. He presented with a Glasgow Coma Scale score of 5 and bilaterally dilated, nonreactive pupils. CT revealed a left acute subdural hematoma (ASDH) with significant midline shift, severe global brain edema, effaced basal cisterns, and brainstem compression signs. Emergency BC was performed, followed by ASDH evacuation. The bone flap was replaced. Postoperatively, the patient showed remarkable recovery, with extubation on day 2 and early mobilization. By day 7, he was walking with assistance, and by discharge on day 13, he exhibited only mild word-finding difficulty, which improved significantly at follow-up.

LESSONS This case highlights the potential of BC as an alternative or adjunct to DC in severe TBI, emphasizing the need for larger, multicenter studies to validate its efficacy.

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KEYWORDS traumatic brain injury; decompressive craniectomy; basal cisternostomy; acute subdural hematoma; brain swelling; case report

Since the first significant paper on basal cisternostomy (BC) in severe traumatic brain injury (TBI) from Nepal was published in 2013,[1] neurosurgeons in some developing countries, notably India and China,[2–6] as well as in the West,[7] have adopted this innovative approach. These countries, which together represent a large fraction of the global population, also experience a high incidence of severe TBI, predominantly stemming from road traffic accidents.[8,9]

Edema in TBI is known to mainly arise from cytotoxic and vasogenic mechanisms.[10] The glymphatic pathway, demonstrated in mouse models, facilitates rapid and bulk CSF influx from the subarachnoid spaces into the brain interstitium, primarily via aquaporin channels in the Virchow-Robin spaces.[11] The "CSF shift" edema hypothesis suggests that a trauma-induced rise in ICP causes CSF to move into the brain through this pathway. Opening the basal cisterns equalizes their pressure with atmospheric pressure, rapidly reversing the CSF shift, leading to ICP reduction.[12,13] Current evidence from mouse models indicates that increased ICP reduces both CSF influx and efflux. Notably, these measurements were taken more than 40 minutes after ICP elevation, including a 10-minute waiting period after ICP elevation, followed by slow fluorescent tracer infusion into the CSF, and an additional 30-minute waiting period postinfusion. Furthermore, these studies do not address edema formation.[14] As a result, the early-phase glymphatic pathway response, within the first 40 minutes of severe TBI, still remains unknown. But the accumulating clinical evidence from regions that have adopted BC consistently indicates that the procedure reverses brain edema and effectively reduces intracranial pressure (ICP).[3–6]

Relevant Surgical Anatomy for BC

Excellent microsurgical knowledge of the relevant anatomical structures encountered during BC is required to perform it safely.

ABBREVIATIONS ACP = anterior clinoid process; ASDH = acute subdural hematoma; BC = basal cisternostomy; BCD = basal cisternal drain; CIS = Cisternostomy Indication Score; DC = decompressive craniectomy; EVD = external ventricular drain; GCS = Glasgow Coma Scale; GCSP = GCS-Pupil; ICA = internal carotid artery; ICP = intracranial pressure; ICU = intensive care unit; OMB = orbitomeningeal band; SAH = subarachnoid hemorrhage; TBI = traumatic brain injury.

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Those structures include the following: 1) sphenoid ridge, which is the prominent bony ridge formed by the lesser wing of the sphenoid bone separating the anterior and middle fossae exposed after the craniotomy; 2) orbitomeningeal band (OMB), a dural fold connecting the frontotemporal basal dura mater to the periorbita, located at the lateral border of the superior orbital fissure; 3) roof of the superior orbital fissure, made of the lesser wing of the sphenoid bone, forming part of the posterior orbital roof (it also forms the lateral aspect of the anterior clinoid process [ACP]); 4) two surgical corridors that provide access to the basal cisterns, the opticocarotid window, which is a triangular space positioned between the optic nerve medially and the internal carotid artery (ICA) laterally, and the carotico-oculomotor window, which is located between the ICA and the oculomotor nerve; and 5) Liliequist membrane, a thin arachnoid structure beneath the third ventricle's floor, separating the chiasmatic, interpeduncular, and prepontine cisterns (it consists of a diencephalic leaf, which divides the suprasellar and interpeduncular cisterns, and a mesencephalic leaf, which separates the interpeduncular and prepontine cisterns).

Patient Selection

The Cisternostomy Indication Score (CIS) proposed by Cherian et al. is a structured 10-point system that guides the selection of appropriate candidates for cisternostomy in moderate to severe TBI.[15] It incorporates three objective criteria: the motor response from the Glasgow Coma Scale (GCS), pupillary response, and status of cisterns on CT. Each parameter is scored from 0 to 3, except for the GCS motor score, which extends up to 4 points. A total score between 3 and 7 suggests that cisternostomy is appropriate in the absence of diffuse axonal injury. Scores of 0–2 or 9–10 indicate that cisternostomy is generally not recommended, although in the case of a score of 8, the decision may depend on the overall clinical context (Tables 1 and 2).[15]

We report, according to CARE (Case Report) guidelines, an example case of a young patient with severe TBI who made a remarkable clinical improvement following timely management with BC.

Illustrative Case

A 17-year-old male was brought to the accident and emergency department following a road traffic accident. While crossing the road, he was hit by a two-wheeler, causing him to fall and strike his head on the ground. He had an episode of generalized seizure after the incident. On arrival, approximately 1 hour later, his GCS score was 5 (E1V1M3) and his pupils were bilaterally dilated and nonreacting to light, hence indicating a GCS-Pupil (GCSP) score of 3. He was hemodynamically stable. A cranial CT scan showed a left acute subdural hematoma (ASDH) causing a significant midline shift and severe global brain edema, with effacement of the basal cisterns and evidence of impending midbrain compression, including a subtly beginning white cerebellum sign and a stretched midbrain (Fig. 1A–C). Mannitol was administered and emergency surgery commenced.

The positioning and setup mirror those used in a standard pterional approach. The head is elevated above the level of the heart and secured with 45° of contralateral rotation and 15° of dorsiflexion in a three-point Mayfield holder. The patient's ipsilateral shoulder is padded. A microscope or exoscope is necessary in the latter stages for visualization of deeper brain structures and access to basal cisterns. An exoscope was used in this case. Standard disinfection and draping of the surgical field and exoscope was carried out.

A reverse question mark skin incision, as in a decompressive craniectomy (DC), was made. The temporalis muscle is dissected off the bone and deflected posteriorly and caudally to ensure an unobstructed surgical corridor in further stages. The craniotomy is tailored to facilitate access to the basal cisterns and removal of the acute pathology. The extent of the craniotomy flap may vary depending on the localization of extra-axial or intra-axial posttraumatic space-occupying masses. In this illustrative case, a left-sided pterional craniotomy was performed (Fig. 2A). The sphenoid ridge was removed completely using a Luer and a high-speed drill (Fig. 2B and C). The next step was detaching the OMB (Fig. 2D) to allow mobilization of the brain in the axial plane as well as to facilitate removal of lateral aspect of the ACP. Removal of the lateral ACP (Fig. 2E) is required in more severe cases with significant brain edema. Both the removal of the sphenoid ridge and this step allow for better mobilization of the brain in the sagittal plane and enhance access to the cisterns later intradurally. The dura is opened in a C shape and deflected toward the base. The dural opening may be kept small at the beginning, as is demonstrated (Fig. 2F), especially in younger patients with severely edematous brain to minimize the risk of brain swelling out uncontrollably. In this case, the BC was performed first, followed by complete removal of the ASDH. The next step is accessing the basal cisterns. Under the exoscope and with gentle dynamic retraction of the frontal lobe, we advanced to the arachnoid membrane covering the opticocarotid window. We sharply incised the membrane and identified the opticocarotid window (Fig. 2G). Then, we incised the mesencephalic leaf of the Liliequist membrane, which is exposed through the opticocarotid window carefully to not injure any perforators (Fig. 2H). The carotico-oculomotor window may also be used. A drain is carefully inserted directed toward the interpeduncular cistern, not more than 2.5 cm from the opticocarotid window (Fig. 2I). A usual 3-mm-diameter external ventricular drain (EVD) may be used for this purpose. Constant slow irrigation is commenced through the basal cisternal drain (BCD). Note that sylvian fissure dissection, as typically done in vascular surgeries, is not required in this procedure. The next step addresses hematomas or contusions. The ASDH was evacuated in this patient. An ICP sensor should be implanted for

TABLE 1. CIS calculation

Category	Score 0	Score 1	Score 2	Score 3	Score 4
GCS motor response	M1 or M2	M3	M4	M5	M6
Pupillary status	Bilaterally dilated & nonreactive	Unilaterally dilated & nonreactive	Unilaterally dilated but reactive	Normal pupils	—
Cisterns on CT scan	Complete brainstem herniation w/ or w/o PCA infarction	CPA cistern obliterated	Suprasellar cisterns obliterated, CPA cistern widened	All cisterns open	—

CPA = cerebellopontine angle; M = motor score; PCA = posterior cerebral artery.
Clinical and radiological parameters were used in calculating the CIS, including the GCS motor response, pupillary status, and cisternal appearance on CT imaging. Each parameter is scored individually, with a total score ranging from 0 to 10.

TABLE 2. Interpretation of the CIS

Total Score	Recommended Clinical Action
9–10	Conservative management & close neurological monitoring
8	Decision individualized based on overall clinical status
3–7	Cisternostomy indicated if diffuse axonal injury is not present
0–2	Cisternostomy generally not beneficial

Recommended clinical actions based on the total CIS, as derived from summing the component scores in Table 1. Decision-making depends on score thresholds and the presence or absence of diffuse axonal injury.

continuous ICP monitoring in the intensive care unit (ICU). The bone flap can be replaced after dura closure if the brain becomes lax and pulsatile, a common occurrence following BC. Bone flap replacement was performed without difficulty in this case (Fig. 1D–F). If the bone flap cannot be replaced intraoperatively due to persistent brain swelling despite placement of a functioning BCD, cranioplasty should be scheduled as early as feasible, once the acute phase has resolved and cerebral edema has subsided sufficiently in follow-up CT scans. Early cranioplasty during the initial hospitalization can streamline patient care by avoiding the need for readmission, while also offering potential benefits such as improved neuromotor and cognitive recovery through timely restoration of CSF dynamics and intracerebral hemodynamics. Nonetheless, neurosurgeons should remain vigilant for an increased risk of hydrocephalus associated with early intervention.[16,17]

Ensuring the proper function of the BCD at all times in an ICU is crucial. The drain is positioned 10–15 cm above the tragus and kept open. It is typically maintained for 3–5 days, depending on the reversal of brain edema, which is assessed through cranial CT on the 1st postoperative day and subsequent days if needed. The ICP sensor may be removed 24–48 hours after BCD removal, provided that ICP remains within normal limits and the patient's neurological status and CT findings are stable. In the acute phase, if the BCD is obstructed and ICP rises, a careful sterile flush may help restore function. However, if this fails and pathological ICP persists, timely revision surgery with placement of a new BCD may be necessary to curb progressive brain swelling and correct the situation. As with an EVD, strict adherence to sterile handling protocols is crucial to reduce the risk of infection. Figure 1D–F shows the patient's 1st postoperative day CT scans, demonstrating a reversed midline shift following hematoma removal and the placement of the BCD in the interpeduncular cistern. The prepontine cisterns appear patent again in Fig. 1F. Small ischemic zones as well as subdural hematoma in the left frontal and temporal areas are also seen (Fig. 1D and E). The reappearance of the basal cisterns and neurological improvement are key indicators guiding the duration of the BCD placement.

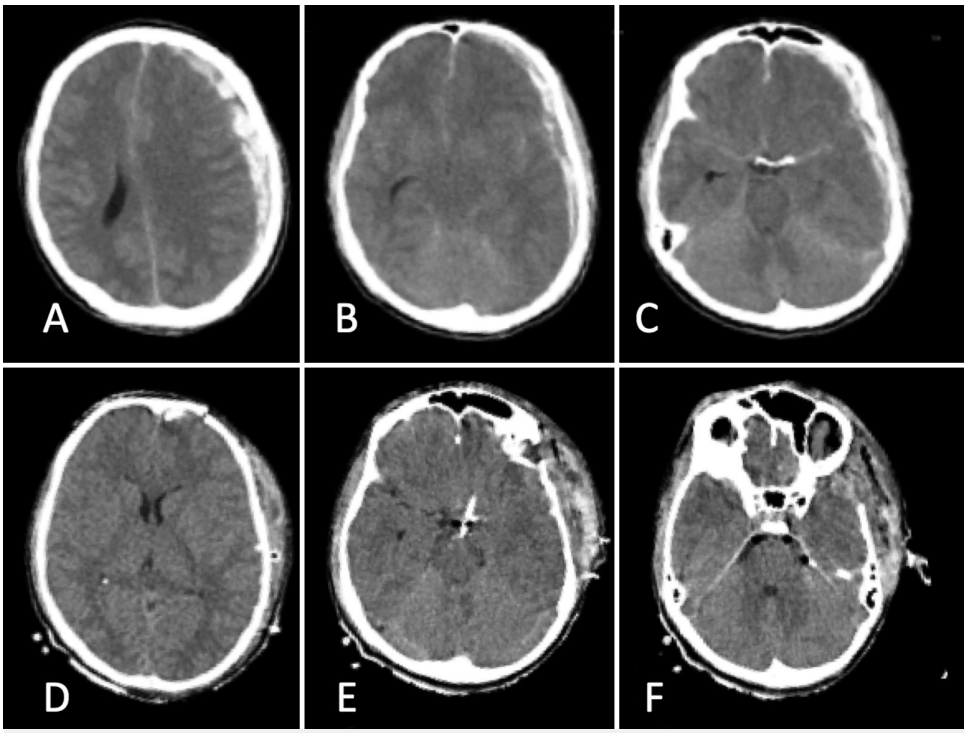

FIG. 1. A–C: Preoperative axial CT scans showing a space-occupying left ASDH, traumatic SAH in the left sylvian fissure, and minor left frontotemporal contusions (B and C). Notice the significant midline shift and severe global brain edema. The basal cisterns are obliterated and a subtle white cerebellum sign is evident. **D–F:** Axial CT scans obtained on the 1st postoperative day. Note the lack of midline shift and the patent basal cisterns. In image E, the tip of the BCD in the interpeduncular cistern following BC is seen. In image F, the prepontine cisterns appear patent again. The bone flap could be replaced despite the severity of the TBI, as seen in this scan.

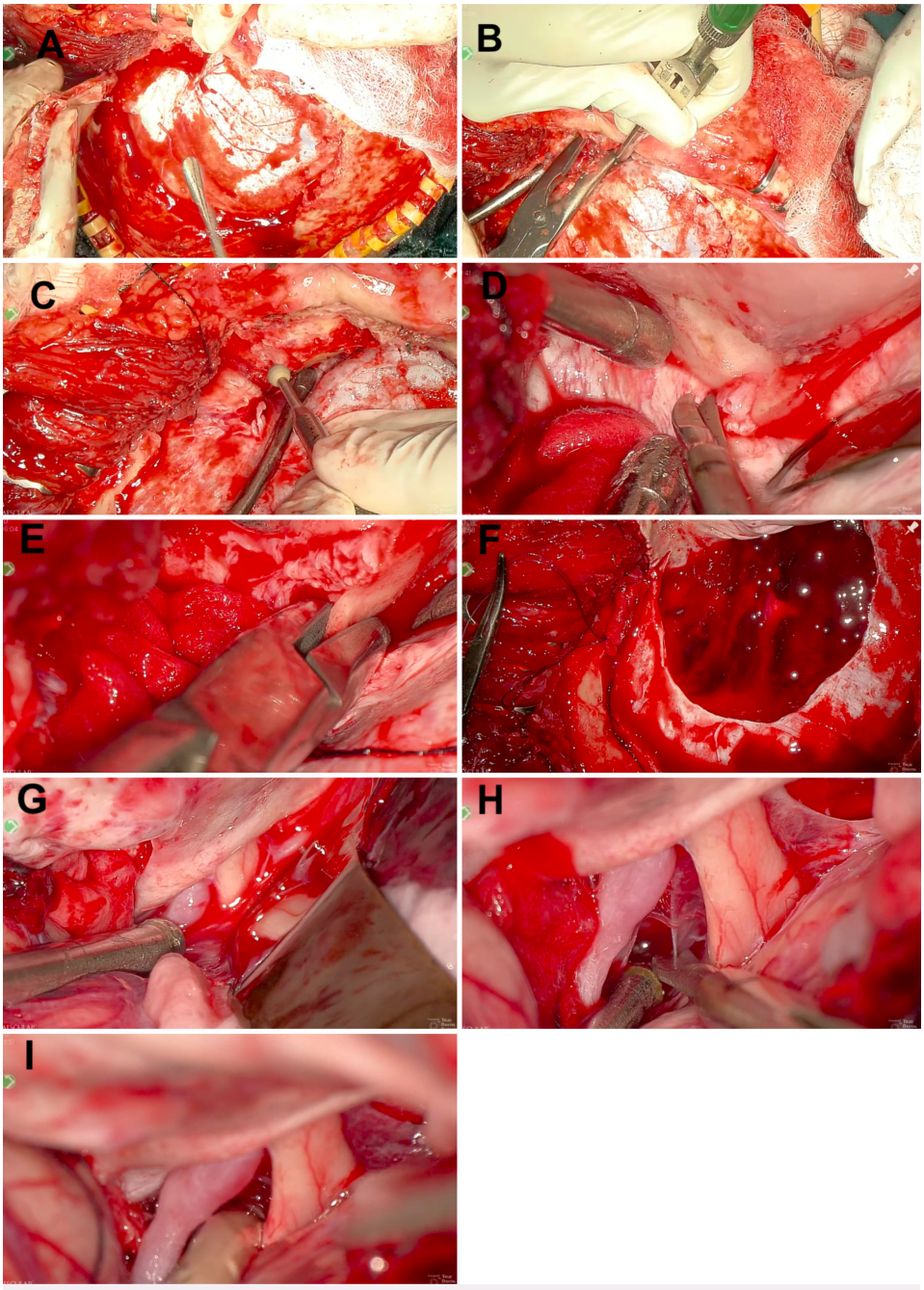

FIG. 2. A: Left pterional craniotomy. **B:** Initial stages of the sphenoid ridge removal with a Luer. **C:** Latter stages of the sphenoid ridge removal; a diamond drill was used. **D:** Cutting of the OMB. **E:** Removal of the lateral ACP after the OMB is cut. **F:** The initial smaller dural opening to access the basal cisterns is shown. **G:** Intradural step of advancing to the basal cisterns. After removal of the ASDH clot at the dural opening, advance toward the arachnoid membrane surrounding the opticocarotid window under gentle and dynamic retraction of the frontal lobe. The suction may be used for dynamic retraction of the temporal lobe. **H:** The mesencephalic leaf of the Liliequist membrane is carefully incised, ensuring that no perforating arteries are damaged. **I:** Final step of BCD insertion through the opticocarotid window.

This patient showed a remarkable speed of recovery. He was extubated on the 2nd postoperative day, and the BCD was removed on the 3rd postoperative day. Mobilization and physiotherapy were initiated on the same day he was extubated. By the 7th postoperative day, he was walking and climbing stairs with assistance. In the initial days postextubation, he exhibited signs of psycho-organic syndrome with aggression and screaming tendencies, which resolved quickly. He was discharged on the 13th postoperative day with mild motor

aphasia, characterized by word-finding difficulty, and slight psychomotor retardation. At the 2-week follow-up, his wound had healed perfectly and his behavior was prompt and adequate. The motor aphasia had improved quite significantly. On realizing the rapid and substantial neurological improvement, the patient expressed deep appreciation for the benefits gained from BC.

Informed Consent

Surgery was performed as an emergency surgery on a patient with severe TBI (GCS score 5).

Discussion

Interestingly, three meta-analyses were published in close succession between September and November 2024.[18–20] These meta-analyses highlighted both potential benefits and current limitations of BC in the management of severe TBI. The studies reported that BC, when combined with DC, is associated with a significant reduction in overall mortality,[18] a shorter ICU stay,[19] and reduced mechanical ventilation duration.[18,20] Additionally, BC has been linked to decreased brain herniation, midline shift reversal, and improved ICP control, as well as a lower requirement for osmotherapy.[19] However, several criticisms remain. One major concern is the low quality and heterogeneity of available studies, making it difficult to draw definitive conclusions.[19] Additionally, the lack of high-quality randomized controlled trials has been emphasized, with calls for more rigorous research.[18] The technical complexity of BC also poses a challenge, requiring advanced microsurgical skills and an in-depth understanding of basal cistern anatomy, which may limit its widespread adoption.Furthermore, safety concerns and uncertainties regarding long-term outcomes reinforce the need for larger, multicenter studies to fully assess the role of BC in TBI management.[20]

Observations

Even though the initial CT scans showed significant brain edema and midline shift, the bone flap could be replaced after BCD insertion, consistent with studies reporting that BC reduces the need for DC in severe TBI.[1–5] Keeping the bone flap back spares the need for a later cranioplasty, thereby completely avoiding its associated surgical risks and costs.

It is well established that blood components released during intracerebral hemorrhage and subarachnoid hemorrhage (SAH) trigger significant inflammatory responses in the brain.[21,22] BCD in SAH facilitates the clearance of blood and its toxic degradation products, leading to reduced cerebral vasospasm and delayed cerebral ischemia as well as improved outcomes.[23–26] Likewise, in severe TBI, blood clearance from the CSF via a BCD may help mitigate inflammatory responses in the brain, thereby reducing the extent of secondary brain injury. This may explain the speedy recovery of the patient characterized by early extubation, rapid mobilization, and near-complete neurological recovery within weeks.

There are some open questions that remain to be addressed by the neurosurgical community. One is regarding the optimal indication for BC due to the wide variety of injury mechanisms. The evidence from the growing number of patients undergoing BC in countries like India and China, where the cause of severe TBI is mainly road traffic accidents, suggests that BC may be safe and effective in many cases, including scooter crashes, which are on the rise and represent an emerging public health problem.[27] Continued research will further refine our understanding of whether certain traumatic mechanisms respond more favorably to BC than others. Another aspect is the lack of clarity on how to approach the disrupted autoregulation. Small et al. demonstrated that cerebral perfusion pressure can play a role as a surrogate measure for cerebral blood flow, but they also drew attention to the constraints of existing clinical management, largely due to the absence of systems capable of continuously capturing physiological variables to inform timely interventions.[28] The above, together with the shortcomings of relying on ICP monitoring only, represents the strongest arguments in favor of a multimodal monitoring strategy including technologies such as near-infrared spectroscopy.[29]

Lessons

Despite presenting with a low GCSP score of 3 and imaging features indicative of impending brainstem herniation, the patient made a striking neurological recovery postoperatively at a remarkable pace. This example case offered the opportunity to discuss the relevant surgical anatomy and the technical steps to perform BC safely and effectively. The course of this young patient highlights the potential of BC as an effective alternative or adjunct to traditional DC for patients with severe TBI, underscoring the need for larger, multicenter studies to further validate its efficacy.

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Disclosures

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

Author Contributions

Conception and design: Sharafudeen, Sakurada, Ganau, Kurita. Acquisition of data: Sharafudeen, Ueno, Cherian. Analysis and interpretation of data: Sharafudeen, Ganau, Cherian. Drafting the article: Sharafudeen, Ganau. Critically revising the article: Ganau, Cherian. Reviewed submitted version of manuscript: Jbarah, Ganau. Approved the final version of the manuscript on behalf of all authors: Sharafudeen. Statistical analysis: Sharafudeen. Administrative/technical/material support: Cherian. Study supervision: Suzuki, Ganau, Kurita.

Supplemental Information

Previous Presentations

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