ScientificScholar®
Knowledge is power
Publisher of Scientific Journals

www.surgicalneurologyint.com

Surgical Neurology International

Editor-in-Chief: Nancy E. Epstein, MD, Clinical Professor of Neurological Surgery, School of Medicine, State U. of NY at Stony Brook.

SNI: Neuroanesthesia and Critical Care

Editor
Ramsis Ghaly, MD
Ghaly Neurosurgical Associates, Aurora, Illinois, USA

SNI
Open Access

Original Article

A prospective exploratory study to assess echocardiographic changes in patients with supratentorial tumors – Effect of craniotomy and tumor decompression

Nirupa Ramakumar[1], Priyanka Gupta[1], Rajnish Arora[2], Sanjay Agrawal[1]

Departments of [1]Anaesthesia and [2]Neurosurgery, All India Institute of Medical Sciences, Rishikesh, Uttarakhand, India.

E-mail: Nirupa Ramakumar - niru27r@gmail.com; Priyanka Gupta - priyanka.anaes@aiimsrishikesh.edu.in; Rajnish Arora - rajnish_19@yahoo.com; *Sanjay Agrawal - sanjay.anaes@aiimsrishikesh.edu.in

***Corresponding author:**
Sanjay Agrawal,
Department of Anaesthesia,
All India Institute of
Medical Sciences, Rishikesh,
Uttarakhand, India.

sanjay.anaes@aiimsrishikesh.edu.in

Received : 24 February 2023
Accepted : 07 April 2023
Published : 05 May 2023

DOI
10.25259/SNI_186_2023

Quick Response Code:

ABSTRACT

Background: Functional changes in the myocardium secondary to increased intracranial pressure (ICP) are studied sparingly. Direct echocardiographic changes in patients with supratentorial tumors have not been documented. The primary aim was to assess and compare the transthoracic echocardiography changes in patients with supratentorial tumors presenting with and without raised intracranial pressure for neurosurgery.

Methods: Patients were divided into two groups based on preoperative radiological and clinical evidence of midline shift of <6 mm without features of raised ICP (Group 1) or greater than 6mm with features of raised ICP (Group 2). Hemodynamic, echocardiographic, and optic nerve sheath diameter (ONSD) parameters were obtained during the preoperative period and 48 h after the surgery.

Results: Ninety patients were assessed, 88 were included for analysis. Two were excluded based on a poor echocardiographic window (1) and change in the operative plan (1). Demographic variables were comparable. About 27% of the patients in Group 2 had ejection fraction <55% and 21.2% had diastolic dysfunction in Group 2 in the preoperative period. There was a decrease in the number of patients with a left ventricular (LV) function <55% from 27% before surgery to 19% in the postoperative period in group 2. About 5.8% patients with moderate LV dysfunction in the preoperative period had normal LV function postoperatively. We found a positive correlation between ONSD parameters and radiological findings of raised intracranial pressure.

Conclusion: The study demonstrated that in patients with supratentorial tumors with ICP, cardiac dysfunction might be present in the preoperative period.

Keywords: Echocardiography, Intracranial hypertension, Left, Supratentorial neoplasms, Optic nerve sheath diameter, Ventricular dysfunction

INTRODUCTION

Brain-heart interactions in patients with brain insult hold a leading place in the developing field of organ cross-talk, with clinically significant implications throughout the perioperative period.[24] The maintenance of systemic and brain homeostasis is impacted by cardiac dysfunction, and

the consequent brain damage has a detrimental effect on the prognosis of neurosurgical patients. Many neurological problems, including aneurysmal subarachnoid hemorrhage (SAH), acute ischemic stroke (AIS), and intracerebral hemorrhage, have been linked to intricate heart-brain relationships.[19]

Although presumably less frequent, cardiac alterations following brain tumors are comparable to the site-specific harm brought on by brain lesions when the regions responsible for controlling circulatory function are injured.[2] Aberrant conduction, myocardial ischemia, abnormal contractility, or a combination of any of these, are all manifestations of cardiac dysfunction. Nearly 17% of patients of a-SAH reveal echocardiographic alterations, including reduced left ventricular (LV) function and regional wall motion abnormalities (RWMA), which are linked to poor functional outcomes. Numerous investigations have found that patients with traumatic brain injury (TBI) have cardiac abnormalities.[14,15] It has also been noted that patients with brain tumors have greater serum levels of N-terminal prohormone of brain natriuretic peptide.[31]

There is scant evidence to support the hypothesis that cerebral lesions with mass effect cause elevated intracranial pressure (ICP).[11] Although papilledema and clinical characteristics can be used to determine elevated ICP, patients with brain tumors do not have a quantitative association.[10] In cases when invasive assessment is not otherwise clinically required, optic nerve sheath diameter (ONSD) by ultrasonography (USG), a non-invasive ICP (nICP) measurement without a serious risk of side effects, could significantly increase our understanding of ICP dynamics.[30] Although the link between ICP and ONSD has been described by a number of authors with varied degrees of accuracy, no research has been done on the diagnostic efficacy of ONSD USG for intracranial midline shift.

Only a few case reports have investigated a persistent increase in ICP in primary brain tumors that causes cardiac dysfunction.[17,32,39] There is currently a paucity of studies investigating the effects of interventions intended to lower ICP on the restoration of cardiac dysfunction after brain tumor.

With this background, this study was done to assess and compare the cardiac function using transthoracic echocardiography in patients with supratentorial tumor who underwent craniotomy and tumor excision, both before and after surgery (primary objective) and to find any correlation of ONSD USG with midline intracranial shift and cardiac parameters (secondary objective).

MATERIALS AND METHODS

This prospective observational study was conducted on 90 patients with supratentorial tumor of either sex, aged between the age group 18 and 60 years with revised cardiac risk index less than two scheduled for elective craniotomy and tumor excision over a period of 2 years. The patients were divided into two groups based on midline shift seen on the preoperative computed tomography (CT) scan/magnetic resonance imaging (a) Group 1: Midline shift of ≤6 mm and (b) Group 2: Midline shift of more than 6 mm. Patients with pregnancy, known case of cardiac illness, body mass index >35 kg/m^2, lesions in the prefrontal cortex, insula, hypothalamus, amygdala, hippocampus, and previous eye surgery and tumor involving optic nerve were excluded from the study. The Institutional Ethics Committee approval (AIIMS/IEC/20/504) was sought, and written informed consent was obtained from either patients or next of the kin. The study was registered with Clinical Trials Registry India (CTRI/2020/10/028313).

Radiological evaluation

Preoperative radiological variables including size, location and number of the tumor, laterality, presence, or absence of cerebral edema and midline shift was noted. The cerebral edema was graded as minimal (maximum thickness of edema was less than the radius of mass), moderate (between the radius and diameter of the mass), or severe (more than the diameter of the mass).

Cardiac evaluation

Transthoracic echocardiography was performed by NR using P21x, 1–5 MHz, phased array probe (Sonosite, Bothell, USA) at preoperative period (24 h) and postoperatively at 48 h. Chamber quantification was performed as per the guidelines of the American Society of Echocardiography.[16] The biplane summation-of-disks method was used for measuring 2D volume and ejection fraction (EF). The EF was graded as normal (EF-55–70%), mild dysfunction (EF-45–54%), moderate dysfunction (EF-35–44%) and severe dysfunction (<30%). Regional wall motion abnormality >1 was considered abnormal for analysis purpose. Diastolic function was graded based on the transmitral Doppler and principally the E/e' ratio obtained at the mitral annulus. E/e' ≥8 was taken as abnormal. E/A ratio <0.8 and >15 was taken abnormal. Right ventricular (RV) function was graded based on the Tricuspid Annulus Plane Systolic Excursion, the lateral tricuspid annular S' velocity on Tissue Harmonic Imaging. Blinded cardiologist unaware of patient clinical condition reviewed all echocardiograms.

ONSD

Patients' eyes were scanned in supine position using a high resolution 7-MHz linear array transducer (Sonosite, Inc., Bothell, WA, USA) on closed eyelids. The structure of the eyes was visualized to align with the optic nerve directly opposite the probe with the ONSD width perpendicular to the vertical

axis of the scanning plane. ONSD bilaterally was measured 3 mm behind the globe and an average of three readings from each eye for calculation. The ONSD measurements were classified as ipsilateral ONSD (craniotomy side, ONSD-Ipsilateral [ONSD-IP]) and contralateral ONSD (side opposite the craniectomy, ONSD-Contralateral [ONSD-CL]) according to the side of the lesion. Transorbital ONSD >5.5 mm was considered as indicative of raised ICP.[18]

Perioperative management

A written informed written consent was taken. All patients were kept nil per oral 6 h for solids and 2 h for clear liquids, premedicated with Tablet Ranitidine 150 mg early morning at 6 am. General anesthesia was given to all patients as per institutional protocol. Standard American Society of Anesthesiologist intraoperative monitors were applied. Anesthesia was induced with intravenous (i.v) propofol 1.5–2 mg/kg and fentanyl 2 μg/kg, while i.v Vecuronium 0.1 mg/kg was used to facilitate orotracheal intubation. Maintenance of Anesthesia was achieved with 50% Oxygen: Air mixture propofol infusion@ 100–150 μg/kg/min and fentanyl infusion @ 1 μg/kg/h and intermittent vecuronium boluses. Following surgical intervention, patients were shifted to the intensive care unit.

Statistical analysis

As it was an exploratory study, power analysis for Paired *t*-test was conducted in G-POWER to determine a sufficient sample size using an alpha of 0.05, a power of 0.80, an effect size of 0.3 (Using Cohen's Convention), and two tails. Based on the assumptions, the desired sample size total of 90 patients was taken**.** Categorical data were presented as percentages (%). Pearson's Chi-square test and Fishers exact test were used to evaluate differences between groups for categorized variables. Normally distributed data were presented as means and standard deviation, or 95% confidence intervals (CI). Student's *t*-test paired and unpaired and analysis of variance were used for comparison between various quantitative parameters. Pearson's correlation test was used for correlation between various quantitative parameters. All tests were performed at a 5% level of significance; thus, an association was significant if $P < 0.05$. Analysis was carried out using the Statistical Package for the Social Studies for Windows version 23.0 and online GraphPad software (Prism 5 for Windows) version 5.01.

RESULTS

Demographic and lesion characteristics

Ninety patients were assessed for eligibility; data of 88 were included for analysis [Figure 1]. Patients divided into – group 1 (36) while group 2 (52). Patients were relatively young (mean age 42 and 37 years in group 1 and group 2, respectively), and 53% were male. The groups were comparable in terms of demographic parameters and clinic-radiological parameters [Tables 1 and 2].

Intraoperative and hemodynamic changes

Intraoperative events like hypotension were seen in 8 (22%) patients (group 1) and 18 patients (34%) (group 2) ($P = 0.21$). The heart rate was significantly higher in preoperative period as compared to postoperative period in group 2 ($P < 0.001$) [Table 3].

Echocardiographic changes

Echocardiographic changes between the groups are shown in [Tables 4a and b]. In the preoperative period, group 2 had more patients (27%) with systolic dysfunction as compared to 2.8% in group 1. ($P < 0.01$) In the postoperative period, also group 2 had more patients (19.2%) with systolic dysfunction as compared to 2.8% in group ($P < 0.02$). Only one patient had RWMA in group 2 which persisted postoperatively.

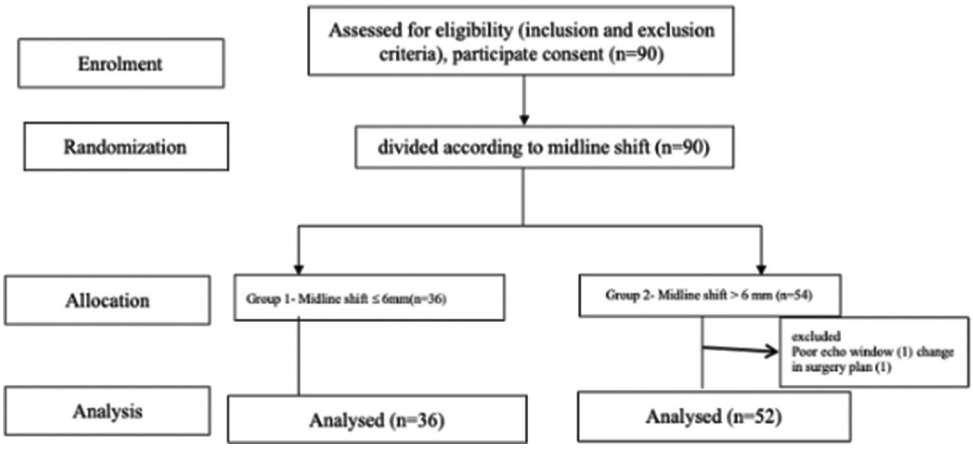

Figure 1: The consort diagram showing the recruitment of individuals for this study, n: number.

The echocardiographic changes before and after surgery in both the groups are shown in Table 3. There was a decrease in the number of patients with EF <55% from 27% before surgery to 19% in the postoperative period in group 2. About 5.8% patients with moderate LV dysfunction in the preoperative period had normal LV function postoperatively. Rest of the parameters related to chamber dimensions, volumes and LV diastolic function (LVDF) and RV systolic function showed no significant differences in the preoperative and postoperative period in both the groups. About 18% patients needed vasopressor use in group 2 and only 8% patients in group 1 postoperatively. About 5.6% and 7.7% in group 1 and group 2 underwent follow-up transthoracic echocardiography (TTE).

Table 1: Comparison of demographic and clinic radiological parameters between the groups.

Parameter	Groups		*P*-value
	Group 1 (*n*=36)	Group 2 (*n*=52)	
Age: Mean±SD	42.61±10.84	37.86±12.74	0.07
Gender: *n* (%)			
Male	21 (58.3)	26 (50.0)	0.44
Female	15 (41.7)	26 (50.0)	
ASA grade (%)			
1	32 (88.9)	38 (73.1)	0.07
2	4 (11.1)	14 (26.9)	
BMI (Mean±SD)	25.08±3.73	23.44±3.47	**0.04***
Preoperative GCS (Mean±SD)	14.94±0.23	14.71±0.57	**0.02***

ASA: American Society of Anesthesiologists (physical status), SD: Standard deviation. BMI: Body mass index, GCS: Glasgow Coma Scale Score, n: Number, **P*<0.05 as statistically significant

ONSD-USG changes

Intergroup and intragroup analysis of ONSD variables depicted in [Tables 5, 6a and b]. IL-ONSD and CL-ONSD reduced postoperatively in both the groups which were statistically significant.

The preoperative ONSD-IP and ONSD-CL value shows a correlation of 0.31 to mid-line shift (MLS) values which were found to be statistically significant ($P = 0.003$). The patients with ONSD-ipsilateral (IL) >5.5 mm had more perilesional edema (56% – moderate and 26% – severe) than in patients with ONSD < 5.5 mm. ($P = 0.024$). Nine (25%) patients with ONSD >5.5 mm in the preoperative period had LV diastolic dysfunction which was statistically significant ($P = 0.02$) [Figure 2] No statistically significant changes seen regarding systolic dysfunction and vasopressor use. No significant association was found between the postoperative TTE and ONSD findings.

DISCUSSION

Our study demonstrated that left ventricular ejection fraction (LVEF) is affected in patients with intracranial space occupying lesion and it may show improvement in few within first 24–48 h of tumor excision. Cross-talk between the brain pathology and the cardiac functions is focusing on wide range of cardiac-functional alterations secondary to brain pathology. Modest cardiac dysfunction secondary to intracranial pathology may have ramifications in the perioperative period. Association of increased ICP and cardiac dysfunction is sparsely reported.[14,17] This is, to our knowledge, the first prospective study on the reversibility of myocardial dysfunction and supratentorial tumors.

Table 2: Preoperative tumor profile and radiological parameters. Values are number (proportion).

Preoperative tumor profile	Group 1 (*n*=36) *n* (%)	Group 2 (*n*=52) *n* (%)	Total	
Glioma	17 (47)	37 (71.2)	54 (61.3)	
Meningioma	15 (41)	12 (23.1)	27 (30.6)	
Sellar and suprasellar tumor	3 (8.3)	2 (3.8)	5 (5.6)	
Pineal tumor	0 (0.0)	1 (1.9)	1 (1.1)	
Radiological parameters				
Parameter	**Group 1 (*n*=36) (%)**	**Group 2 (*n*=52) *n* (%)**	**Total**	***P*-value**
Laterality				
Right	18 (50.0)	23 (44.2)	41 (46.6)	0.59
Left	18 (50.0)	29 (55.8)	47 (53.4)	
Perilesional edema#				
Minimal	12 (33.3)	8 (15.4)	20 (22.7)	0.06
Moderate	20 (55.6)	30 (57.7)	50 (56.8)	
Large	4 (11.1)	14 (26.9)	18 (20.5)	

#Perilesional edema - <5 mm: Mild, 5–10 mm: Moderate, >10 mm: Severe. Minimal, moderate or large if its maximum thickness was less than the radius of mass, between the radius and diameter and more than the diameter, n: number

Preoperative hypertension, hyperlipidaemia, increasing age, and asymptomatic coronary artery disease can cause diastolic dysfunction. Intraoperative direct effects of anesthetics on cardiac inotropy, lusitropy, and peripheral vasodilatation cannot be underestimated in unmasking any subclinical systolic cardiac dysfunction. The absence of a regional wall motion abnormality rules out systolic failure due to an acute myocardial infarction. Increased sympathetic surge with resultant increased levels of circulating endogenous catecholamines consequent to increased ICP is described in the literature.[22] Incidence of patients in TBI cohort with reduced LVEF has been found in the range of 12–22%.[6,25] Only one study found that no patients had global LV dysfunction (95% CI, 0–0.07).[34] The average LVEF was 65 ± 4% like in our study. This contradictory finding was attributed to likelihood of lower levels of catecholamines in TBI and the younger age group in their cohort.

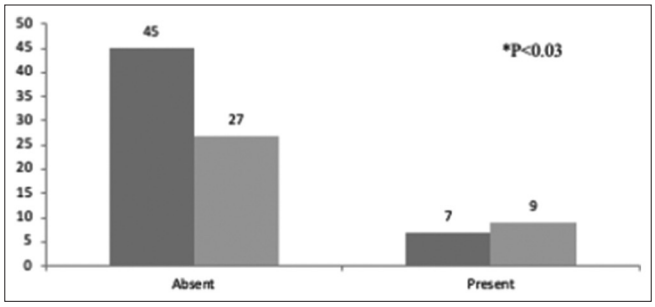

Figure 2: Association of optic nerve sheath diameter (ONSD) and left ventricular diastolic dysfunction (LV) diastolic dysfunction. Dark grey bar- ONSD <5.5 mm, light grey bar- ≥5.5 mm. **P*<0.05 as statistically significant.

To determine diastolic function, we employed the streamlined measurement technique recommended by Greenstein and Mayo under the aegis of the American College of Chest Physicians, employing e and E/e' using echocardiography.[7] The most updated guideline given in 2016 for evaluating LVDF by Echocardiography is challenging for anesthesiologists to apply at the patient's bedside.[23] It is also essential to note that the American Society of Echocardiography/European Association of Cardiovascular Imaging algorithm has yet to receive any additional validation and relies on expert consensus. The E/e' ratio was utilized by us, with values >8 suggesting diastolic dysfunction. In this simplified method, the diastolic function is a binary concept: Present or absent.

In our study, diastolic dysfunction was present in three patient (8.3%) in patients with midline shift of <6 mm and in 11 (21.2) % of patients with MLS of >6 mm preoperatively. Chronic sympathetic activity and myocardial norepinephrine discharge maybe postulated as a cause of LV diastolic dysfunction. Chronic hypertension could be the cause of diastolic dysfunction in the group with MLS <6 mm. Grassi *et al*., in their study, discovered a correlation between elevated sympathetic nerve activity as detected by microneurography and the existence of asymptomatic LV diastolic failure.[5] Studies on TBI and SAH patient population have found greater frequency of diastolic dysfunction.[2,12]

Group 2 patients (increased ICP) had a higher heart rate in the preoperative period than group 1 ($P < 0.05$). It can be because ICP returned to normal after the tumor removal. This finding contrasts the classic Cushing's reflex causing bradycardia primarily found in these patients. The Koszewicz *et al*. study, which examined the profile of autonomic dysfunctions in 30 patients with the primary

Table 3: Comparison of echocardiographic and hemodynamic changes in preoperative period and postoperative period (48 h) in both the groups.

	Group 1 (*n*=36)			Group 2 (*n*=52)		
	Preop	**Postop**	***P*-value**	**Preop**	**Postop**	***P*-value**
TTE variables						
LVIDs (Mean±SD	2.01±0.73	2.08±0.73	0.29	2.01±0.59	2.01±0.65	0.65
LVIDd (Mean±SD)	4.23±1.3	4.37±1.06	0.30	4.04±0.65	4.07±0.99	0.11
ESV (Mean±SD)	45.08±6	44.77±8.13	0.8	44.57±7.75	46.50±10.09	0.11
EDV (Mean±SD)	99.77±14	101.80±14.57	0.13	91.69±18.48	94.63±16.981	0.08
EF (Mean±SD)	63.50±8.43	64.66±7.58	0.06	60.71±9.81	62.25±9.35	**0.006***
TAPSE (Mean±SD)	1.95±0.26	1.93±0.25	0.62	1.90±0.37	1.99±0.30	0.1
E/A (Mean±SD)	1.53±0.40	1.56±0.19	0.64	1.63±0.40	1.59±0.28	0.43
E/e'(Mean±SD)	7.70±3.02	6.79±1.50	0.06	8.10±3.15	7.71±2.9	0.45
Hemodynamic variables						
Heart rate (Mean±SD)	77.91±11.0	77.50±9.33	0.71	81.80±14.18	77.09±12.86	**<0.001***
MAP (Mean±SD)	92.66±6.39	93.34±6.54	0.51	93.07±7.00	93.27±6.92	0.83

LVIDd: Left ventricular internal diameter end in diastole and LVIDs: Left ventricular internal diameter end in systole, ESV: End systolic volume, EDV: End diastolic volume, E: Early diastolic mitral inflow velocity; e': Early diastolic mitral annulus velocity, A: Mitral peak velocity of late filling, TAPSE: Tricuspid annular plane systolic excursion, EF: Ejection fraction, MAP: Mean arterial pressure, SD: Standard deviation. Preop: Preoperative period, Postop: Postoperative period at 48 hours, n: Number, **P*<0.05 as statistically significant

brain tumors, is consistent with this conclusion. The subjects had increased heart rates and elevated blood pressure with low heart rate variability.[13] ICP and heart rate (HR) variability were explored concerning one another by Fedriga *et al*. The study showed sympathetic nervous system hyperactivity in patients with the primary brain tumors.[4] Schmidt *et al*. showed that sympathetic activity increased significantly by 17% with ICP rise from 8 to 15 mmHg.[33]

There was statistically significant improvement in EF in the postoperative period at 48 h in few patients. This concurs with study by Srinivasaiah and Praveen *et al*. who found significant improvement in systolic dysfunction after surgery.[26,36] As opposed to their studies, mean ejection fraction (>60%) in our results was higher. We observed that the chamber dimensions and diastolic dysfunction present preoperatively persisted postsurgery. The concentric remodeling and decreased LV compliance could have persisted in the postoperative period. The possible reason may be attributed to time needed for the reversibility to be achieved. We measured these changes at 48 h postoperatively by which time reversibility might not have been achieved. In most of the other studies with significant improvement in cardiac function, the echocardiography was done at 1-week postsurgery and comprised of acute brain injury patients with significantly increased ICP-induced cardiac changes.

Interestingly, ONSD values in our patients resemble those found in patients with elevated ICP. Montorfano *et al*. demonstrated ONSD value of 5.82 mm (95% CI 5.58–6.06 mm) in patients with elevated ICP.[20] Several studies evaluated sonographic ONSD assessments in neurocritical care cohorts with various central nervous diseases, including intracerebral hemorrhage, ischemic stroke, brain tumor, and SAH.[3,21,27] In agreement with our findings, these studies reported a good correlation between ONSD and ICP. However, ICP values were considerably lower in Moretti *et al*.[21] and Rajajee *et al*. study[27] and higher in Geeraerts's *et al*. study[6] than in our investigation. Dubourg *et al*. noted that ONSD >5.00–5.70 mm had a raised ICP >20 mm with a sensitivity of 90% (95% CI: 80–95%) and a specificity of 85% (95% CI: 73–93%).[3]

There was a significant decrease in ONSD- IP and ONSD-CL after the surgery in both the groups ($P < 0.001$). These results are consistent with these studies.[9,35,37] The cutoff values for raised ICP was 4.28 ± 0.28 mm in Kalim *et al*. study.[9] There was significant regression at 12 and 24 h compared with preoperative values in the postoperative period. Wang *et al*. found strong correlation between ONSD and ICP values (r of 0.798 [95% CI, 0.71–0.86]).[37]

A recently published study by Bäuerle *et al*. revealed that the ultrasonic measurement of ONSD could not accurately estimate ICP in patients with SAH.[1] They attributed the impaired reversibility of the optic nerve sheath to massive ICP exposure up to 45 mm Hg and obturation of arachnoid trabecular

Table 4a: Comparison of the preoperative echocardiography variables between the two groups.

Parameters	Group 1 (*n*=36) (%)	Group 2 (*n*=52) (%)	*P*-value
LV systolic dysfunction			
Mild (45–54%)	1 (2.8)	11 (21.2)	**0.01***
Moderate (30–44%)	0 (0.0)	3 (5.8)	
LVDF			
Present	3 (8.3)	11 (21.2)	0.84
Absent	33 (91.6)	41 (78.8)	
RWMA			
Present	0 (0.0)	1 (1.1)	0.43

LVDF: Left ventricular diastolic dysfunction, RWMA: Regional wall motion abnormality, LV: Left ventricular. n: number, *P<0.05 as statistically significant

Table 4b: Comparison of the postoperative echocardiography variables between the two groups.

Parameters	Group 1 (*n*=36) (%)	Group 2 (*n*=52) (%)	*P*-value
LV systolic dysfunction			
Mild (45–54%)	1 (2.8)	10 (19.2)	**0.02***
Moderate (30–44%)	0 (0.0)	0 (0.0)	
LVDF			
Present	2 (5.5)	10 (19.9)	0.5
Absent	34 (94.4)	42 (80.1)	
RWMA			
Present	0 (0.0)	1 (1.1)	0.43

LVDF: Left ventricular diastolic dysfunction, RWMA: Regional wall motion abnormality, LV: Left ventricular, n: Number, *P<0.05 as statistically significant

Table 5: Intragroup analysis of ONSD variables at different times of observation.

Parameter	Group 1		*P*-value	Group 2		*P*-value
	Preoperative	Postoperative (48 h)		Preoperative	Postoperative (48 h)	
	Mean ± SD	Mean ± SD		Mean ± SD	Mean ± SD	
ONSD IP (mm)	5.06 ± 1.1	4.53 ± 0.92	**<0.001**	5.87 ± 1.08	5.06 ± 0.94	**<0.001***
ONSD CL (mm)	4.63 ± 0.92	4.28 ± 0.62	**0.002**	4.97 ± 0.78	4.54 ± 0.69	**<0.001***

ONSD: Optic nerve sheath diameter, IP: Ipsilateral, CL: Contralateral, SD: Standard deviation. n: Number, *P<0.05 as statistically significant

Table 6a: Comparison of ONSD in the two groups in the preoperative period.

Parameter	Group 1 (*n* = 36) (Mean ± SD)	Group 2 (*n* = 52) (Mean ± SD)	*P*-value
Preoperative ONSD IP	5.05 ± 1.10	5.87 ± 1.08	**0.001***
Preoperative ONSD CL	4.63 ± 0.91	4.97 ± 0.77	0.06

ONSD: Optic nerve sheath diameter, IP: Ipsilateral, CL: Contralateral, SD: Standard deviation. n: Number, **P*<0.05 as statistically significant.

Table 6b: Comparison of ONSD in the two groups in the postoperative period.

Parameter	Group 1 (*n*=36) (Mean±SD)	Group 2 (*n*=52) (Mean±SD)	*P*-value
Postoperative ONSD IP (48 h)	4.53±0.92	5.06±0.93	**0.01***
Postoperative ONSD CL (48 h)	4.28±0.62	4.54±0.69	0.07

ONSD: Optic nerve sheath diameter, IP: Ipsilateral, CL: Contralateral, SD: Standard deviation, n: Number, **P*<0.05 as statistically significant

structures with subarachnoid blood. Wu *et al*. measured ONSD in the supine position once a day for 3 consecutive days starting on the day of admission. These results suggest that the changes in the ONSD and ICP values are not closely correlated after dynamic observation.[38] The reasons for the unsuitability of ONSD for dynamically monitoring ICP were first, selection bias as most patients had intracranial emorrhage and second, that difference in the ONSD measurements for each eye was not taken. Ragauskas *et al*. study show that the TCD-based ICP measurement method has a better diagnostic reliability than the ONSD method due to high age dependent variations of 4.9 mm in the latter.[28]

We found positive correlation between ONSD and radiological findings of raised ICP. Robba *et al*. demonstrated that ONSD was the best non-invasive way to measure ICP and recommended the following formula to do so: nICP ONSD = 5.00 × ONSD–13.92 mmHg.[29] However, no predictive models have been ever achieved. Therefore, a normal CT scan does not exclude a raised ICP. Our study found that 40% of patients had ONSD ≥5.5 mm. This higher percentage might be due to more patients with high ICP and interobserver variability. We observed that raised ONSD-IP findings preoperatively had significant relation with LV diastolic dysfunction. The pathophysiological effects of increased ICP on cerebral venous system can explain this association.

Cardiovascular disease is the second largest cause of death in a retrospective study patients with brain tumors, 30–50% comprised patients with meningioma.[8] Therefore, it seems prudent to identify patients with any cardiac changes for better perioperative outcomes. Our findings have significant ramifications for the perioperative management of brain tumor patients. First, our findings suggest the complexity of the cardiac response to raised ICP. The enhanced sympathetic response in these patients and the local/systemic effect of the tumor are thought to contribute to these changes. Second, changes to systolic and diastolic function occur to a variable degree. These results show that a uniform strategy for fluid administration and vasopressor selection is risky; hence, a comprehensive resuscitation plan should be uniquely adapted to the patient and incorporate an echocardiographic assessment of the heart into any therapeutic decisions. TTE is a complex, operator-independent tool. We need to interpret the echo findings in the context of the signs and symptoms of the patient. A comprehensive approach incorporating clinical, biochemical, and echocardiographic findings is advised when treating a patient with cardiac dysfunction.

Our study had few limitations. The study included a small sample size, long-term effects at 6 months/1 year were not evaluated, biochemical markers of cardiac dysfunction, or catecholamine levels measuring the sympathetic activity were not observed, and quantitative measurement of ICP was not done. We also did not have previous transthoracic echocardiography reports of the patients as it is not routinely advised when they visit the preanesthetic clinic. Prospective multicenter randomized trials are essential to evaluate the impact of intraoperative TTE. Further studies are required to investigate the temporal course of the changes in the echocardiographic variables in chronically raised ICP scenarios and to see the long-term effects at 6 months/1 year.

CONCLUSION

Our study demonstrated that in patients with supratentorial tumors with features of increased ICP, cardiac dysfunction maybe present in the preoperative period; hence, 2D-echocardiography must be included in preoperative assessment for patients presenting for craniotomy and tumor excision.

Acknowledgment

I would like to thank the anesthesia technical team for the help extended throughout the study period.

Declaration of patient consent

The Institutional Review Board (IRB) permission obtained for the study.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

REFERENCES

1. Bäuerle J, Niesen WD, Egger K, Buttler KJ, Reinhard M. Enlarged optic nerve sheath in aneurysmal subarachnoid hemorrhage despite normal intracranial pressure. J Neuroimaging 2016;26:194-6.
2. Cuisinier A, Maufrais C, Payen JF, Nottin S, Walther G, Bouzat P. Myocardial function at the early phase of traumatic brain injury: A prospective controlled study. Scand J Trauma Resusc Emerg Med 2016;24:129.
3. Dubourg J, Javouhey E, Geeraerts T, Messerer M, Kassai B. Ultrasonography of optic nerve sheath diameter for detection of raised intracranial pressure: A systematic review and meta-analysis. Intensive Care Med 2011;37:1059-68.
4. Fedriga M, Czigler A, Nasr N, Zeiler FA, Park S, Donnelly J, *et al*. Autonomic nervous system activity during refractory rise in intracranial pressure. J Neurotrauma 2021;38:1662-9.
5. Grassi G, Quarti-Trevano F, Seravalle G, Arenare F, Volpe M, Furiani S, *et al*. Early sympathetic activation in the initial clinical stages of chronic renal failure. Hypertension 2011;57:846-51.
6. Geeraerts T, Launey Y, Martin L, Pottecher J, Vigué B, Duranteau J, *et al*. Ultrasonography of the optic nerve sheath may be useful for detecting raised intracranial pressure after severe brain injury. Intensive Care Med 2007;33:1704-11.
7. Greenstein YY, Mayo PH. Evaluation of left ventricular diastolic function by the intensivist. Chest 2018;153:723-32.
8. Jin K, Brennan PM, Poon MT, Sudlow CL, Figueroa JD. Raised cardiovascular disease mortality after central nervous system tumor diagnosis: Analysis of 171,926 patients from UK and USA. Neurooncol Adv 2021;3:vdab136.
9. Kalim Z, Siddiqui OA, Nadeem A, Hasan M, Rashid H. Assessment of optic nerve sheath diameter and its postoperative regression among patients undergoing brain tumor resection in a tertiary care center. J Neurosci Rural Pract 2022;13:270-5.
10. Kazdal H, Kanat A, Findik H, Sen A, Ozdemir B, Batcik OE, *et al*. Transorbital ultrasonographic measurement of optic nerve sheath diameter for intracranial midline shift in patients with head trauma. World Neurosurg 2016;85:292-7.
11. Kienzler JC, Zakelis R, Marbacher S, Bäbler S, Schwyzer L, Remonda E, *et al*. Changing the paradigm of intracranial hypertension in brain tumor patients: A study based on non-invasive ICP measurements. BMC Neurol 2020;20:268.
12. Kopelnik A, Fisher L, Miss JC, Banki N, Tung P, Lawton MT, *et al*. Prevalence and implications of diastolic dysfunction after subarachnoid hemorrhage. Neurocrit Care 2005;3:132-8.
13. Koszewicz M, Michalak S, Bilinska M, Budrewicz S, Zaborowski M, Slotwinski K, *et al*. Profile of autonomic dysfunctions in patients with primary brain tumor and possible autoimmunity. Clin Neurol Neurosurg 2016;151:51-4.
14. Krishnamoorthy V, Prathep S, Sharma D, Gibbons E, Vavilala MS. Association between electrocardiographic findings and cardiac dysfunction in adult isolated traumatic brain injury. Indian J Crit Care Med 2014;18:570-4.
15. Krishnamoorthy V, Chaikittisilpa N, Lee J, Mackensen GB, Gibbons EF, Laskowitz D, *et al*. Speckle tracking analysis of left ventricular systolic function following traumatic brain injury: A pilot prospective observational cohort study. J Neurosurg Anesthesiol 2020;32:156-61.
16. Lang RM, Badano LP, Mor-Avi V, Afilalo J, Armstrong A, Ernande L, *et al*. Recommendations for cardiac chamber quantification by echocardiography in adults: An update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. J Am Soc Echocardiogr 2015;28:1-39.e14.
17. Lechowicz-Glogowska E, Rojek A, Duszynska W. The irreversible neurogenic stress cardiomyopathy during large supratentorial brain tumor resection. Neurocrit Care 2019;31:587-91.
18. Lee SH, Kim HS, Yun SJ. Optic nerve sheath diameter measurement for predicting raised intracranial pressure in adult patients with severe traumatic brain injury: A meta-analysis. J Crit Care 2020;56:182-7.
19. Mazzeo AT, Micalizzi A, Mascia L, Scicolone A, Siracusano L. Brain-heart crosstalk: The many faces of stress-related cardiomyopathy syndromes in anaesthesia and intensive care. Br J Anaesth 2014;112:803-15.
20. Montorfano L, Yu Q, Bordes SJ, Sivanushanthan S, Rosenthal RJ, Montorfano M. Mean value of B-mode optic nerve sheath diameter as an indicator of increased intracranial pressure: A systematic review and meta-analysis. Ultrasound J 2021;13:35.
21. Moretti R, Pizzi B, Cassini F, Vivaldi N. Reliability of optic nerve ultrasound for the evaluation of patients with spontaneous intracranial hemorrhage. Neurocrit Care 2009;11:406-10.
22. Mrozek S, Gobin J, Constantin JM, Fourcade O, Geeraerts T. Crosstalk between brain, lung and heart in critical care. Anaesth Crit Care Pain Med 2020;39:519-30.
23. Nagueh SF, Smiseth OA, Appleton CP, Byrd BF 3rd, Dokainish H, Edvardsen T, *et al*. Recommendations for the evaluation of left ventricular diastolic function by echocardiography: An update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. J Am Soc Echocardiogr 2016;29:277-314.
24. Prasad Hrishi AP, Lionel KR, Prathapadas U. Head rules over the heart: Cardiac manifestations of cerebral disorders. Indian J Crit Care Med 2019;23:329-35.
25. Prathep S, Sharma D, Hallman M, Joffe A, Krishnamoorthy V, Mackensen GB, *et al*. Preliminary report on cardiac dysfunction after isolated traumatic brain injury. Crit Care Med 2014;42:142-7.
26. Praveen R, Jayant A, Mahajan S, Jangra K, Panda NB, Grover VK, *et al*. Perioperative cardiovascular changes in patients with traumatic brain injury: A prospective observational study. Surg Neurol Int 2021;12:174.
27. Rajajee V, Fletcher JJ, Rochlen LR, Jacobs TL. Comparison of accuracy of optic nerve ultrasound for the detection of intracranial hypertension in the setting of acutely fluctuating

vs stable intracranial pressure: Post-hoc analysis of data from a prospective, blinded single center study. Crit Care 2012;16:R79.
28. Ragauskas A, Bartusis L, Piper I, Zakelis R, Matijosaitis V, Petrikonis K, *et al*. Improved diagnostic value of a TCD-based non-invasive ICP measurement method compared with the sonographic ONSD method for detecting elevated intracranial pressure. Neurol Res 2014;36:607-14.
29. Robba C, Cardim D, Tajsic T, Pietersen J, Bulman M, Donnelly J, *et al*. Ultrasound non-invasive measurement of intracranial pressure in neurointensive care: A prospective observational study. PLoS Med 2017;14:e1002356.
30. Robba C, Santori G, Czosnyka M, Corradi F, Bragazzi N, Padayachy L, *et al*. Optic nerve sheath diameter measured sonographically as non-invasive estimator of intracranial pressure: A systematic review and meta-analysis. Intensive Care Med 2018;44:1284-94.
31. Ruggieri F, Gemma M, Calvi MR, Nicelli E, Agarossi A, Beretta L. Perioperative serum brain natriuretic peptide and cardiac troponin in elective intracranial surgery. Neurocrit Care 2012;17:395-400.
32. Santos S, Castro M, Raimundo A, Ribeiro A. Anaesthetic management of large meningioma excision complicated by Takotsubo and posterior reversible encephalopathy. BMJ Case Rep 2022;15:e246690.
33. Schmidt EA, Despas F, Traon AP, Czosnyka Z, Pickard JD, Rahmouni K, *et al*. Intracranial pressure is a determinant of sympathetic activity. Front Physiol 2018;9:11.
34. Serri K, El Rayes M, Giraldeau G, Williamson D, Bernard F. Traumatic brain injury is not associated with significant myocardial dysfunction: An observational pilot study. Scand J Trauma Resusc Emerg Med 2016;24:31.
35. Shirodkar CG, Rao SM, Mutkule DP, Harde YR, Venkategowda PM, Mahesh MU. Optic nerve sheath diameter as a marker for evaluation and prognostication of intracranial pressure in Indian patients. Indian J Crit Care Med 2014;18:728-34.
36. Srinivasaiah B, Muthuchellappan R, Sesha UR. A prospective observational study of electrocardiographic and echocardiographic changes in traumatic brain injury-effect of surgical decompression. Br J Neurosurg 2022:1-6.
37. Wang LJ, Chen LM, Chen Y, Bao LY, Zheng NN, Wang YZ, *et al*. Ultrasonography assessments of optic nerve sheath diameter as a non-invasive and dynamic method of detecting changes in intracranial pressure. JAMA Ophthalmol 2018;136:250-6.
38. Wu GB, Tian J, Liu XB, Wang ZY, Guo JY. Can optic nerve sheath diameter assessment be used as a non-invasive tool to dynamically monitor intracranial pressure? J Integr Neurosci 2022;21:54.
39. Yogendranathan N, Herath HM, Pahalagamage SP, Kulatunga A. Electrocardiographic changes mimicking acute coronary syndrome in a large intracranial tumour: A diagnostic dilemma. BMC Cardiovasc Disord 2017;17:91.

How to cite this article: Ramakumar N, Gupta P, Arora R, Agrawal S. A prospective exploratory study to assess echocardiographic changes in patients with supratentorial tumors – Effect of craniotomy and tumor decompression. Surg Neurol Int 2023;14:166.