Medical Journal of the
Islamic Republic of Iran

Volume 9
Number 4
Winter 1374
February 1996

Case Reports

NEUROGENIC HYPERTENSION: REPORT OF FIVE CASES TO HIGHLIGHT THE CONTROVERSY

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ABSTRACT

The entity of "neurogenic hypertension" is defined as arterial hypertension caused explicitly by derangement of the intricate network of the central nervous system.

Among 193 cases of cranial rhizopathies operated on between 1984 and 1995 at this center, 5 cases of established arterial hypertension with concomitant rhizopathies also underwent ventrolateral medullary decompression. The elevated blood pressure showed an incredible decline, reaching normal values after vascular decompression. The systolic blood pressure however showed a much greater fall than the diastolic component.

An endeavour is made to throw light on the cases operated on with special emphasis on the central nervous system as an etiological factor to explain the cause of essential hypertension.

Keywords: Neurogenic Hypertension; Neurovascular Decompression; Cranial Rhizopathies.

MJIRI, Vol. 9, No. 4, 351-354, 1996.

INTRODUCTION

Arterial hypertension, defined as a blood pressure (BP) exceeding 160/90 according to the American Heart Association, stands as the major nominator of mortality in industrialized societies.[4]

Regarding hyper/hypoactive dysfunction of the cranial nerves, Dandy, at the dawn of the century in 1932, explained the causal relationship between trigeminal neuralgia (TN) and 5th nerve root vascular compression.[2] Gardner and Sava postulated the same mechanism for hemifacial spasm (HFS).[2] Tenth nerve compression as a cause of neurogenic hypertension (NH) was subsequently explained by Jannetta,[9] who reported 53 cases of EH treated by vascular decompression.[7]

Four of our patients had TN and one had subarachnoid hemorrhage (SAH). They constituted 2.5% (5/193) of our neurovascular decompressions (NVD). The patient with SAH and hypertension was operated after magnetic resonance imaging (MRI) revealed an abnormal vessel in the left ventrolateral medullary cistern.

This study was performed on 5 cases of cranial rhizopathies in an attempt to clarify the role of the central nervous system (CNS) in the genesis of NH.

PATIENTS AND METHODS

Of 193 cases of cranial rhizopathies, i.e., 153 cases of TN, 33 with HFS, 6 with spasmodic torticollis and one with

vertigo, 5 cases with NH have undergone surgery at our center since 1984.[18] Among the NH cases, 4 had left-sided TN and one had SAH. Only one had a familial history of high BP. A brain MRI of the case with SAH showed an abnormal vascular loop at the left medullary cistern (Fig. 1). All patients had been receiving antihypertensive treatment but of no avail. Surgical intervention was initiated on the grounds of ameliorating the pain caused by the coexisting cranial nerve dysfunction.

Secondary causes for hypertension had been ruled out preoperatively and patients were allowed to take antihypertensives till the morning of the scheduled date for surgery.

Underneuroleptanesthesiaconductedinsupineleftlateral position, a standard retromastoid craniectomy was performed, the brainstem exposed, the incriminating artery visualized and the 5th & 10th nerves decompressed by autologous muscle tissue (Fig. 2).

The BP levels which were monitored intraoperatively and followed every four hours postoperatively, showed no rise to necessitate medication (Fig. 3). Characteristics of the patients with NH are summarized in Table I.

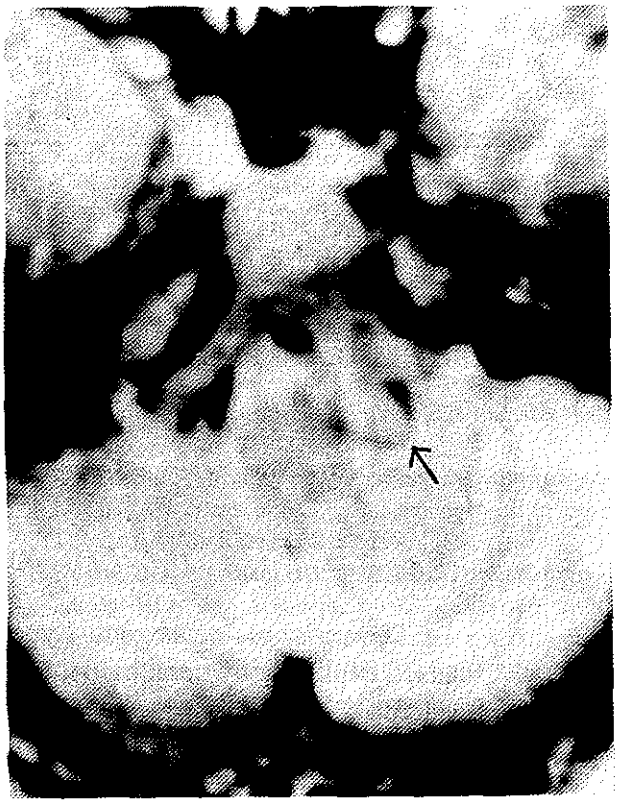

Fig. 1. MRI showing the abnormal vessel in left lateral medulla (arrow).

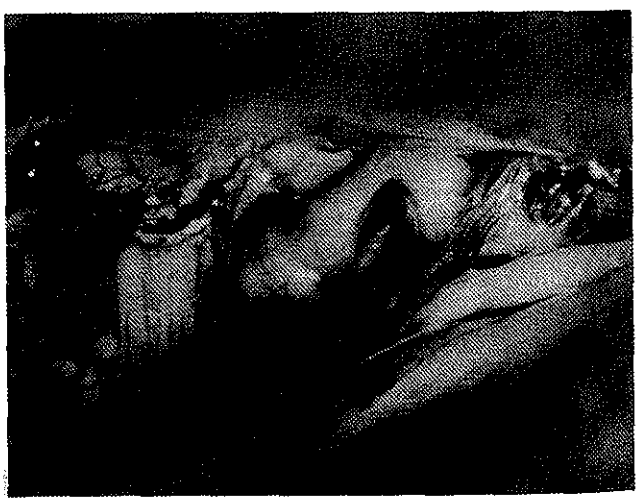

Fig. 2. Intraoperative photomicrograph demonstrating the abnormal vascular loop decompressed by muscle tissue.

RESULTS

The BP showed striking changes in the early postoperative period, as depicted in the graphs. During neurovascular decompression, transient fluctuations in BP were noted which could safely be attributed to manipulation of the vagus nerve. A gradual reduction in BP was evident postoperatively, finally reaching a steady state (Fig. 3). In one patient (case IV) with TN and arterial hypertension and no evidence of vascular compression of the 10th nerve, the BP remained high in the postoperative pain-free period. Therefore NH in this particular case could not have been due to stressful pain.

The average follow-up was one year (6 to 24 months). All patients except case IV remained normotensive, and facial pain resolved in all.

Using the" paired t-test", the changes in BP were shown to be statistically significant for systolic and diastolic values ($P<0.05$). Table II demonstrates the calculated t's.

DISCUSSION

Essential hypertension forms the bulk of hypertensive cases.[20] Lately however, the entity of neurogenic hypertension caused by ventrolateral medullary compression has been ushered into forming a percentage of EH cases.[7]

Disorders such as TN, HFS, tinnitus and vertigo are classified as hyperactive cranial nerve dysfunctions, while facial hypoesthesia, Bell's palsy and sensorineural hearing loss are known as hypoactive dysfunction syndromes.[10]

Although many seem to be benefitted from the clinical effects of NVD,[9,18] as yet the concept of neurovascular compression for some remains controversial in functional neurosurgery.[1]

The significant drop in our patient's BP levels could be explained on the basis of Jannetta's neurovascular

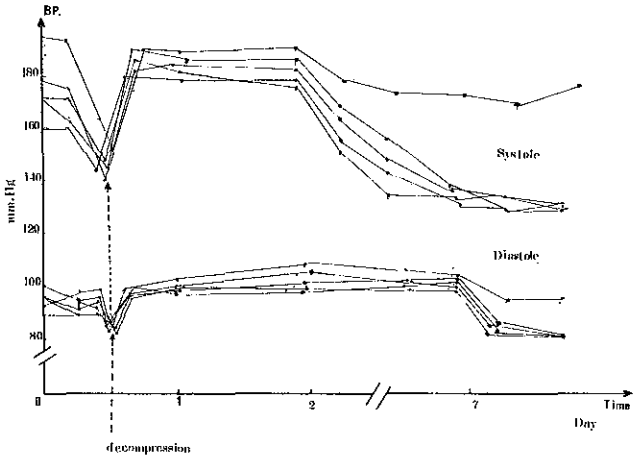

Fig. 3. Graph showing the BP changes of cases I to V.

compression hypothesis.[18] In our patients, the most common associated cranial rhizopathy was TN[19] but in Jannetta's series it was HFS.[7] The most common offending vessels were the vertebral artery and PICA. Only 2.5% of our NVDs constituted NH, but this figure was 7.5% in Jannetta's study.[9]

Jannetta and Segal produced an experimental form of chronic hypertension in baboons by pulsatile compression of the left ventrolateral medulla.[8,17] Many experiments produced chronic labile hypertension in the cat by lesions of the nucleus tractus solitarius.[15]

In a retrospective angiographic study, Kleinberg showed an abnormal vascular loop in the left ventrolateral medulla of hypertensive patients.[11] Naraghi et al., in a postmortem microanatomical study showed an abnormal vascular loop in the left ventrolateral medulla of hypertensive people dying of some coexisting disease, although brainstem histology was not significantly abnormal.[14] Despite near normal histology of the brainstem in NH, it has been proposed that a central abnormality is necessary for neurovascular compression to become symptomatic.[13]

The central network of BP regulation is depicted in Fig.

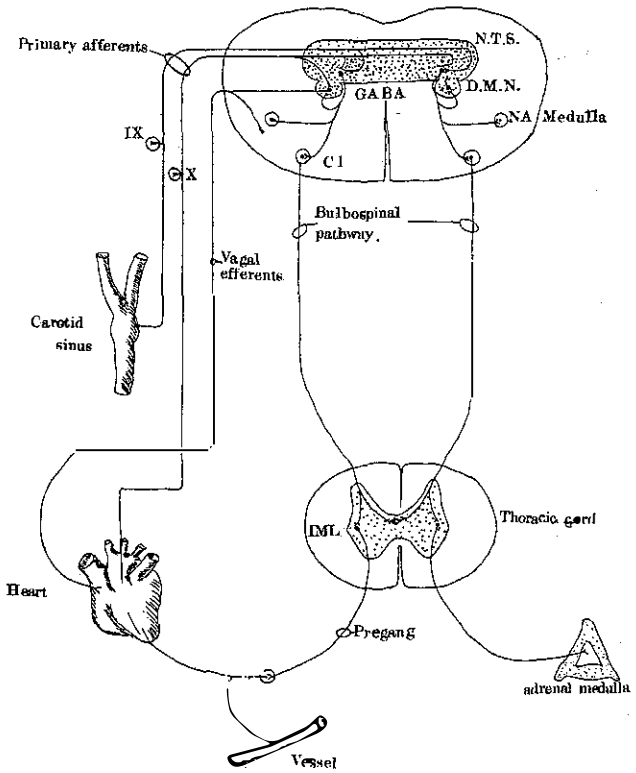

Fig. 4. The central reflex arc responsible for BP regulation.

4. NH can be produced by disturbing the components of the central reflex arc responsible for BP regulation, either peripherally by baroreceptor deafferentation[12,21] or centrally by neurovascular compression[7,8] and other lesions at the medullary level.[5,6,16]

In order to regulate BP, signals transmitted to the nucleus tractus solitarius via the 9th and 10th cranial nerves stimulate neurons which secrete GABA and inhibit the C1 cell group of the vasomotor center in the ventrolateral medulla.[21]

Theoretically, neurovascular compression of the medulla can disturb this reflex arc; this hypothesis is confirmed by our operative results, which show a significant reduction in

Table I. Perioperative patient characteristics and blood pressure recordings.

Case	Age/Sex	Complaints	Duration (year)	BP Preop.	BP Postop.	Offending vessel 5th n.	Offending vessel 10th n.
1	51/Male	L.$V_{2,3}$TN	4	170/100	140/90	SCA[1]	PICA[2]
2	57/Fem	L.$V_{1,2}$TN	2	190/120	120/80	SCA	BA[3]
3	65/Male	L.V_3TN	4	160/100	100/60	SCA	PICA
4	50/Fem	L.&R.V_3TN	5	160/100	160/100	L.Vein R.SCA	None
5	65/Male	IVH+SAH	10	200/120	120/80	None	VA[4]

1- Superior cerebellar artery
2- Posterior inferior cerebellar artery
3- Basilar artery
4- Vertebral artery.

$V_{1,2,3}$ = Trigeminal nerve (1st, 2nd, 3rd Branch).
L = Left
R= Right
TN = Trigeminal neuralgia.

Table II. Blood pressure comparisons pre- and postoperatively.

BP. Preop	BP. Postop	dif. systolic	dif. diastolic
170/100	140/90	30	10
140/120	120/80	70	40
160/100	100/60	60	40
160/100	160/100	0	0
200/120	120/80	80	40
Mean			
174/108	128/82	48	26

$t = \frac{\bar{d}}{\sqrt{\frac{s^2}{n}}}$ t systolic = 3.28 t diastolic=2.98 (df= 4)

BP after NVD.

Due to the satisfactory results of NVD of the 5th and 7th cranial nerves, its application has gained widespread popularity in different neurosurgical centers. Presently, 10th nerve decompression has been suggested as a form of treatment for NH in selected cases.

Further studies on larger series of patients with NH are needed in order to determine the criteria for the selection of patients for this apparently favoured and seemingly correct surgical procedure.

ACKNOWLEDGEMENTS

We are indebted to Dr. N. Saffarian who helped us by providing the available literature.

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