Aphasia Associated with Intracerebral Neoplasms

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INTRODUCTION

Consultation to neurosurgery and neurology by speech and language pathologists may involve the evaluation of patients with neoplasms. We undertook this study because we wanted to know more about how the various types of brain tumors affect language. Several studies of special value to the clinician interested in brain tumor syndromes are published (Miceli, Caltagirone, Gainotti, Masullo, Silveri, and Villa, 1981; Haas, Vogt, Schiemann, and Patzold, 1982; Holtzman, Rudel, and Goldensoh, 1978; Smirnov, 1977; Shuping, Toole, and Alexander, 1980; Rosse, 1983; Sandyk and Maloon, 1983). Most writers agree with Damasio (1979): "Tumor syndromes naturally vary with location and histological nature," but the degree and quality of variation is not completely understood (Rosenfield and Goree, 1975; Benson, 1979; Kertesz, 1979, 1981). There are several reasons for this. First, brain tumor cases have been grouped with stroke and trauma cases in many language studies (e.g., Varney, 1981; Basso, Capitani, and Zanobio, 1982). Second, the prevalence of brain tumor cases in reports of specific aphasia syndromes is low. For example, brain tumor is infrequently reported to be the cause of "thalamic aphasia" (Jonas, 1982). Conversely, brain tumor is a frequent cause of pure alexia (Greenblatt, 1973; Fincham, Nibbelink, and Aschenbrener, 1975; Cohen, Salanga, Hully, Steinberg, and Hardy, 1976; Vincent, Sadowsky, Saunders, and Reeves, 1977; Turgman, Goldhammer, and Braham, 1979; Van Buren, 1979), but results of these studies are probably not generalizable to broader aphasic syndromes. Third, language has been studied less extensively than other areas such as affect and cognition (Hochberg and Slotnick, 1980; Haaland and Delaney, 1981; Hom and Reitan, 1982; Golden, Moses, Coffman, Miller, and Strider, 1983). Except for Pettit, McNeil and Solomon (1978), and Burns and Boyle (1984), few studies of aphasia have been written from the point of view of the clinical aphasiologist.

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PROBLEM

The purpose of this paper is to describe, using the Western Aphasia Battery (WAB; Kertesz, 1979), the aphasia profiles of 30 patients with left hemisphere intracerebral neoplasms. Our specific questions were: 1) Is aphasia an early-presenting sign of brain tumor? 2) What is the severity of aphasia associated with brain tumor? 3) Is the severity of aphasia related to duration of symptoms or to type of tumor? 4) What types of aphasia are most frequently associated with brain tumors? and 5) Is type of aphasia related to site of tumor?

METHOD

Subjects. This was a retrospective study of speech/language pathology and medical records of patients hospitalized at Duke University and Durham Veterans Administration Medical Centers.

Thirty cases will be described in this paper. All had infiltrative intracerebral tumors in the left hemisphere. Twenty-nine subjects were right-handed; one was left-handed. Twenty-five were male, five were female. In our sample, ages ranged from 30 to 72 years with a mean age of 54.0. Mean educational level was 10 years, ranging from 2 to 20 years. Nine cases had metastatic brain tumors, 21 cases had gliomas, of which 17 were glioblastomas and 4 were other types of glioma.

We selected this patient sample following review of 84 records. Thirty-nine cases were excluded because of: 1) history of neurologic deficit unrelated to tumor, 2) bilateral or midline tumor locus, 3) right hemisphere tumor locus, or 4) extracerebral tumor, such as meningioma or schwannoma. Fifteen additional cases were excluded because we had administered a non-standardized memory and language protocol. Our inclusion criteria for this study were: 1) intracerebral tumor originating in the left hemisphere, 2) the availability of Western Aphasia Battery scores, and 3) data on lesion locus confirmed by CT scan. Patients were evaluated postoperatively in 23 of 30 cases and preoperatively in 7 cases.

RESULTS

Early-presenting signs and symptoms documented by the admitting physician in the medical record were recorded. Of 13 early-presenting symptoms, aphasia was the most prevalent. Aphasia presented in 11 patients and usually occurred with at least one other symptom. Other symptoms present in 10 percent or more of our sample included mental status change, headache, motor deficit, personality change, seizure, and sensory deficit. Six other symptoms appeared in less than 10 percent of the cases. These were dysarthria, nausea and dizziness, insomnia, lethargy, loss of consciousness, and malaise.

The duration of symptoms before the medical diagnosis of brain tumor was recorded. The average duration from the onset of symptoms to medical diagnosis was approximately 9 months for our metastatic cases, 2-1/2 months for the glioblastoma cases, and 1/2 month for other glioma cases, for a group mean duration of 4-1/2 months. Our cases were heterogeneous with regard to aphasia severity, aphasia type, and lesion site.

Table 1. Mean Western Aphasia Battery aphasia quotients for three subgroups of patients with intracerebral neoplasms.

Subgroup	Western Aphasia Battery Aphasia Quotient
Metastatic Cases (N = 9)	x = 75.00 sd = 23.56 r = 22.2 - 95.2
Glioblastoma Cases (N = 17)	x = 61.35 sd = 25.18 r = 15.5 - 96.4
Other Gliomas (N = 4)	x = 75.28 sd = 17.49 r = 50.2 - 89.0
All Cases (N = 30)	x = 67.30 sd = 24.14 r = 15.5 - 96.4

Aphasia Severity. As shown in Table 1, aphasia severity ranged from mild to very severe, with aphasia quotients ranging from 15.5 to 96.4 (out of 100.0 possible). We found no apparent relationship between aphasia severity and duration of symptoms. In addition, the severity of aphasia bore no apparent relationship to type of tumor.

Aphasia Type. The analysis of aphasia type involved looking, first, at fluent versus nonfluent, and, second, the specific aphasia syndrome as derived from the Western Aphasia Battery aphasia profile. As shown in Table 2, our sample had 24 fluent aphasic patients and 6 nonfluent aphasic patients. All the aphasia syndromes -- anomic, conduction, Wernicke's, Broca's, transcortical sensory, transcortical motor and global aphasia -- are represented in our sample of 30 brain tumor patients. Of the fluent aphasia syndromes, 5 of 24 fluent aphasic patients had lesions confined to the frontal lobe, while only 13 of 24 had exclusively posterior lobe lesions. A similarly unexpected result was found for the nonfluent aphasic cases. Of 6 nonfluent patients, only 1 patient had an exclusively frontal lobe lesion, while 3 had exclusively posterior lobe lesions. These data offer objective support for the suggestion made by Kertesz (1979) that tumor patients differ from stroke patients in the presentation of their respective aphasias.

Lesion Site. We then looked at lesion site for the tumors in the left hemisphere. Six of our cases had frontal lesions; 6 had frontal-parietal lesions, 2 had frontal-temporal lesions, 3 had temporal lesions, 6 had temporal-parietal lesions, 5 had parietal lesions, and 2 had parietal-occipital lobe lesions. When we looked more closely at the distribution of the 2 major tumor types (glioblastoma or metastatic), the glioblastoma cases tended to favor posterior loci, while the metastatic cases tended to favor anterior loci.

SUMMARY

In summary, 30 cases of dominant hemisphere intracerebral neoplasms -- either metastatic or primary -- were evaluated using the Western Aphasia

Table 2. Classification of fluency and aphasia type for three subgroups of patients with intracerebral neoplasms.

Subgroup	Fluent/Nonfluent	Aphasia Type	
Metastatic Cases (N = 9)	8 Fluent	Anomic	5
	1 Nonfluent	Conduction	0
		Wernicke's	2
		TSA*	1
		Broca's	0
		TMA**	1
		Global	0
Glioblastoma Cases (N = 17)	12 Fluent	Anomic	7
	5 Nonfluent	Conduction	2
		Wernicke's	2
		TSA	1
		Broca's	3
		TMA	0
		Global	2
Other Gliomas (N = 4)	4 Fluent	Anomic	3
	0 Nonfluent	Conduction	0
		Wernicke's	1
		TSA	0
		Broca's	0
		TMA	0
		Global	0
All Cases (N = 30)	24 Fluent	Anomic	15
	6 Nonfluent	Conduction	2
		Wernicke's	5
		TSA	2
		Broca's	3
*Transcortical sensory aphasia		TMA	1
**Transcortical motor aphasia		Global	2

Battery, and records were reviewed retrospectively. Our findings were as follows: 1) Approximately one-third of our sample presented aphasia before the medical diagnosis was made, 2) all patients had aphasia at the time of the language evaluation, and aphasia ranged from mild to very severe, 3) aphasia severity in our cases was related neither to duration of symptoms nor type of tumor, 4) most patients (24 of 30) had a "fluent aphasia," but were heterogeneous regarding specific types of aphasia, and 5) type of aphasia--nonfluent or fluent--was not always related to anterior or posterior lesion locus, as predicted from the classical model of the aphasias associated with focal lesions (i.e., stroke) in the dominant hemisphere. Some of our nonfluent aphasia cases had tumors originating in posterior areas; some of our fluent aphasia cases had tumors originating in the anterior left hemisphere.

CONCLUSIONS

We derive 3 conclusions from our data. First, aphasia associated with intracerebral neoplasms is heterogeneous with regard to degree of deficit and type of aphasia. Second, although metastatic tumors tended to favor anterior loci, and glioblastomas tended to favor posterior loci, type of aphasia did not appear to be related directly to the type of neoplasm. Third, aphasia due to neoplastic lesion may not offer the same localizing value as aphasia subsequent to stroke.

As our basic understanding of the language disorder associated with brain tumor improves, it is likely that our ability to offer meaningful prognostic statements, treatment, and couseling will also improve. It is to this end that further study of patients with brain tumors is necessary.

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DISCUSSION

Q: You mentioned that about one-third of your patients presented with aphasia as an early symptom. Do you have any information about the behaviors shown, or was this just noted in the medical record?

A: This information was derived from the physician's admission history in the medical record.

Q: From your experience, do you have any impressions about what we should look for as an early symptom?

A: In general, I think we should look for a progressive onset as well as memory and personality changes co-occurring with aphasia.

Q: I think more specific language characteristics in your follow-up study would be informative.

A: I agree.

Q: All your subjects were typed, but it is my understanding that you didn't see any of the subjects because it was a retrospective study.

A: The majority of patients were originally seen by me, my co-authors, or by other Western Aphasia Battery-trained speech and language pathologists, and all files were then studied retrospectively.

Q: Of the patients you personally saw, what were your impressions of the language disorder? Was the language disorder typical of what you see in CVA patients or did you notice anything different?

A: Some patients in this series were similar to left hemisphere stroke patients, others were not. I'm not sure that I could immediately differentiate a stroke from a tumor patient on initial evaluation. The differential characteristics between aphasia associated with stroke versus brain tumor certainly deserves study, but this was not the specific purpose of our study.

Q: Last year I presented a single subject who had a head injury and with whom I used the Boston Diagnostic Aphasia Exam (BDAE). He typed as a transcortical sensory aphasic on the BDAE, but if you met this person you just knew that this wasn't an appropriate label. I'm concerned with our using labels when behaviorally there are some differences from the aphasias as classically defined.

A: I agree that there are often other behaviors in addition to the aphasia that may complicate the aphasia profile.

Q: Did you look at or find any relationship between the fluency-nonfluency dimension and the severity of aphasia?

A: No, we did not.

Q: Were the tumors restricted to the cortex?

A: No. The infomation on lesion site and extent was taken from the neuro-radiology report. We anticipate a more refined analysis of the CT scan data by our co-author, Dr. Massey, who is a neurologist.

Q: There are numerous case studies showing that lesions deep to the left temporal lobe, especially those impinging upon the caudate nucleus and the anterior limb of the internal capsule can cause nonfluency.

A: Yes, we intend to look at depth of lesion in further detail.

Q: Did you classify your patients before surgery?

A: Most of our patients were evaluated postoperatively.

Q: I mention this because sometimes neurosurgeons will enter the brain in a different place from actually right above the lesion site, so that in fact these patients may have had brain damage to a part of the brain other than what is seen on their CT scan. Were the CT scans before or after surgery?

A: The 2 CT scans shown in our paper were postoperative scans.

Q: Was postoperative CT information used in all cases?

A: That was not well controlled in all cases. That is the other aspect of the CT scan analysis that we're doing now.

Q: Might not a better title for your paper be something like "Speech and Language Characteristics of patients with Intracranial Neoplasms?" The Western Aphasia Battery (WAB) classifies everybody, as we learned the other day. A lot of your patients were classified as anomic on the test. Since naming is a problem for all kinds of patients we see, not just aphasic patients, and you're only talking about the WAB Aphasia Quotient here, it is hard for me to agree that all these patients should be called aphasic patients. I wonder if you can look back at your files and look at things beyond the aphasia quotient.

A: I have a couple of comments. First, this was a select group of brain tumor patients that was referred because of apparent language deficits. Many patients were given the WAB (all patients reported here); others were given a broader nonstandardized battery. If we believed the WAB would not allow us to document all the deficits, we used our nonstandardized protocol. The WAB was given not just to achieve a profile but because we believed it to be sufficiently descriptive of the patients' deficits. Second, whether or not you agree with the classification system, our data do show that tumor patients show a variety of aphasia types in addition to anomic aphasia that have not been reported previously.

C: I think the question as to the appropriateness of currently available aphasia batteries for tumor patients is an important one. The same question arises in studies of "aphasia" in the dementias. Systematic comparisons of standardized and nonstandardized language tests will, we hope, lead to the development of new batteries specific to special patient populations. We started with the WAB in this study of brain tumor patients because this is part of our standard diagnostic protocol.

Q: I have some questions from a different orientation. First, in terms of duration of illness on these individuals, you said that it was duration from the time of diagnosis in the metastatic cases. Was that diagnosis of the primary lesion or the metastasis?

A: When we did not have information regarding the onset of symptoms from the metastasis, we used diagnosis of primary tumor.

Q: What other treatments were these patients receiving at Duke? As I recall, aren't they very much involved with the chemotherapy and radiotherapy programs? My guess is that these people were receiving a lot of other medications and many of them may well have been radiated to the brain. If you're out beyond six months, part of the effect may have nothing to do with the tumor but with radionecrosis.

A: We did not look systematically at medical treatment, but several patients did have all three treatments -- surgery, radiation, and chemotherapy. The patient we discussed that had the nonfluent aphasia and posterior lesion was studied before his medical treatment was begun.

Q: My next comment relates to what was said before in terms of the depth of lesion. I think your slide showing cortical relationships to these lesions is very deceiving because of the 2 cases you showed, one was very deep and went to the midline. To say that the tumor involves a single lobe area is really very misleading. Particularly in the glioblastomas, which are noted for being very invasive, you may not see the extent of these tumors on a CT scan.

A: We appreciate that point.

Q: Most or all of these patients have had surgery, is that right?

A: Yes.

Q: How confident are you that their aphasia was the same after surgery as it was before surgery?

A: Only 5 of our subjects had both pre- and postoperative evaluations. I suspect that there were changes in language performance pre- and postoperatively.

Q: What you may have is a study of patients post-op for removal of tumors, and not a study of the effect of the tumor on language function.
A: Yes, we agree.

Q: What other psychometric tests did these patients have such as the WAIS or Halstead-Reitan? What other cognitive problems did they have? It's hard to believe that they were just aphasic.
A: To my knowledge, the patients in our series did not routinely undergo neuropsychological evaluations. We did not review the medical chart for this information.