Clinical Case Conference

Late-Onset Psychosis: Clinical, Research, and Ethical Considerations

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This is a case report of a patient with no history of psychiatric illness until the age of 60, at which time he presented with paranoid delusions and auditory hallucinations. Before then, he had been employed, full-time, for many years. In this report we discuss his initial presentation, history, and neuropsychological assessments, as well as treatment and course of illness. In the Discussion section, we outline diagnostic and prognostic implications, as well as treatment and ethical/legal considerations.

CASE PRESENTATION

Mr. A was a single, educated, unemployed, Caucasian man with no history of psychiatric symptoms until the age of 60. At that time, he presented with symptoms of paranoid delusions and auditory hallucinations, along with flat affect. Mr. A believed that his sister and several other people were conspiring against him and harassing him. He felt they were out to steal his money, lock him up, and physically injure him. He believed that his sister was "forcing" him to be hospitalized so that she could steal his money. He admitted to frequently hearing voices of people he knew. He would hear two, three, or even four voices at a time. The voices would sometimes converse with each other. The voices told him they would steal his money and his car and sometimes told him to hurt himself. He was admitted to a local hospital for 7 days. After discharge from that hospital, he was followed in a research clinic.

Mr. A's history was obtained from medical records, the patient himself, his sister, and physicians and medical staff involved in his care.

Background History

Mr. A was the youngest of four siblings. He had one brother and two sisters. His father and mother died of natural causes at 79 and 61 years of age, respectively. Mr. A was reported to be a "loner." He never married and had no children. He reported having several girlfriends in the past; his longest relationship lasted 10 years.

Mr. A earned a bachelor's degree in accounting. He joined the Army and was in active duty for 2 years but saw no combat and received an honorable discharge. He then worked as a hospital controller/accountant at a community hospital, his longest employment, for 14 years. Mr. A left this job because the administrative offices moved to a distant locale. He then worked at another community hospital as an accountant for nearly 6 years before being laid off because of budget cuts. He was unable to find a job for 1 year. He then worked for an advertising corporation for 2 years. His main hobby was collecting, restoring, and trading antique cars; he stopped this later because of financial constraints. He gambled on horse races, along with his friends, about once a week. He spent almost a year "studying" horse racing through books and papers at the local library. He won several thousand dollars through gambling; most of this money was spent on living expenses and paying off his usual bills. Mr. A stayed with his sister, to whom he paid rent, and provided child care, without compensation, for her daughter.

Limited information was available about Mr. A's family psychiatric history. His mother had been admitted to an inpatient psychiatric unit at the age of 22, but her diagnosis was unknown. She had remained in the hospital for nearly 3 months. There was no family history of dementia.

Initial Mental Status Examination

Mr. A presented to the emergency psychiatric unit at a local hospital as a pleasant and cooperative man who was balding and overweight. His clothing was dirty and stained. His voice was aprosodic. He was matter-of-fact but brief in recounting his psychotic symptoms. There were no obviously loose associations. He did have a rather elaborate, paranoid delusional system with delusions of thought insertion and of being harassed and talked about and a belief that he was being monitored by various electronic devices. He also believed that people were trying to control his thinking, although he would not allow it. He admitted to auditory hallucinations concerning his sister, i.e., voices saying that she was going to steal his car and money. His mood was dysphoric, and his affect was flat. He denied suicidal and homicidal ideation. He stated, however, that he would try to defend himself if someone continued to harass him. His orientation and memory were globally intact. Intellectual functioning was judged to be unimpaired. Insight and judgment were considered to be partial.

Medical Examination and History

Mr. A had mild essential hypertension and diabetes mellitus, which were

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Supported in part by NIMH grants MH-49671, MH-43695, MH-45131, and MH-19934 and by the Department of Veterans Affairs.

under good control with his weight loss program and required no further treatment. He had no history of cigarette smoking or substance abuse. He reported social drinking in the past. The results of a general physical examination at intake were reported to be within normal limits.

Subsequent detailed neurological examination (after Mr. A started neuroleptic treatment) revealed a decreased upward gaze. He had a positive snout reflex, decreased knee jerks, and absent ankle jerk bilaterally. He had rigidity on activation, a resting tremor, postural instability, and a slapping gait. He also had postural and intention tremors, sensory ataxia on tandem gait, decreased sensation of vibration in all extremities with decreased temperature sensation in peripheral upper extremities bilaterally, and graphesthesia. He did not demonstrate visual or hearing impairment.

Neuropsychological Evaluations

As a research participant, Mr. A underwent annual comprehensive neuropsychological evaluations for the first 3 years of his illness. His baseline scores on cognitive screening tests, including the Mini-Mental State (1), Blessed Test of Information, Memory, and Concentration (2), and Mattis's Dementia Rating Scale (3), were 28, 0, and 142, respectively. These values were considered to be within normal limits for his age and education. At baseline, his WAIS-R full-scale IQ was 97, verbal IQ was 99, and performance IQ was 97.

Mr. A had scattered mildly to moderately impaired scores on a few tests within several neuropsychological ability areas (i.e., attention, learning, memory, and motor). His only consistently impaired performances were in the areas of fine motor coordination, speeded symbol copying, and general fund of information.

Magnetic Resonance Imagery (MRI)

Mr. A's MRI of the brain showed moderate central and mild cortical volume loss and a few scattered focal hyperintensities in the deep white matter of the cerebral hemispheres. No other abnormalities were noted.

Treatment and Course of Illness

Mr. A started regimens of haloperidol (10 mg b.i.d.) and benztropine mesylate (1 mg b.i.d.) during his initial visit at the local hospital. Subsequently, the dose of haloperidol was decreased to 10 mg/day. Mr. A experienced remission of most of his positive symptoms including auditory hallucinations and a decrease in paranoia, although he remained somewhat suspicious and guarded. He had an unchanging facial expression, paucity of expressive gestures, affective nonresponsivity, and poverty of speech.

Mr. A displayed signs of tardive dyskinesia (abnormal choreoathetoid jaw and foot movements) within a year of neuroleptic treatment. His appetite had decreased, and he had lost seven pounds. Because of these concerns, he was advised to decrease the haloperidol dose gradually to 2 mg. A few weeks later, Mr. A decided on his own to completely stop taking his medications. He reportedly did well without his medications for about 6 months.

At that time, Mr. A again developed psychotic symptoms, with a significant disturbance in his functioning in social relations and self-care. By the time he came to the clinic, he had been experiencing a full-blown recurrence of his hallucinations and delusions for at least several weeks. He began thinking that his sister and neighbors were plotting against him and were out to take his money and lock him up. Because of his paranoia, Mr. A refused to eat meals prepared by his sister and instead bought and cooked his own meals. He also believed that his drink was being poisoned. He feared that someone would set his car on fire if he were to be hospitalized. He reported auditory hallucinations of male and female voices, some familiar and some unfamiliar, threatening him. He reported that citizens band radios were waking him up. His paranoia became so intensified that he moved out of his sister's place and lived in a hotel for about 1 week. As he related his paranoid delusions, Mr. A became fearful and began incorporating the hotel staff into his delusional system. His thought process was difficult to follow and tangential. He admitted to homicidal ideation toward his "attackers." On examination, Mr. A was poorly groomed and malodorous, and, in contrast to his reported anxiety, fear, and worries, he had a restricted range of affect. He denied suicidal ideation. His speech was slow, with long intervals to respond. He reported that he was sleeping only 3 to 4 hours per night, a change from his usual 8 hours of sleep, because of anxious preoccupation with his paranoid delusions. Although he was guarded, he admitted that he had been sitting at home with a knife, ready to defend himself against his "attackers."

Mr. A was hospitalized. On admission, there was no evidence of sensory impairment, alcohol or other substance abuse, or neurological illness, such as stroke or seizure disorder. Results of laboratory tests including chemistry panel, CBC, vitamin B_{12} level, liver and thyroid functions, and serology were within normal limits, except for a slightly elevated cholesterol level.

Mr. A started a regimen of haloperidol, 6 mg/day. His hallucinations and delusions improved. Following discharge, he returned to baseline functioning with continued residual symptoms, including diminished sense of purpose, social drive, and emotional range, few interests, restricted affect, and poverty of speech. He was enrolled in a study of vitamin E to reduce symptoms of tardive dyskinesia, which had been evident in the orofacial region as well as limbs, on and off, for the previous couple of years. After 10 months in this study, Mr. A discontinued vitamin E because of lack of response.

There was no decline in Mr. A's cognitive performance (including memory) over the course of repeated testing over 3 years, except for a word list learning task (California Verbal Learning Test), in which his learning performance declined from the average to the mildly to moderately impaired range. Given that a similar but milder decline was seen on story learning, it appears that Mr. A experienced an isolated, mild decrease in verbal learning ability. There was no known neurological cause for such an isolated deficit. His IQ ranged from 100 to 112. His Mini-Mental State scores remained at 29, and Dementia Rating Scale scores ranged from 135 to 140 (all within the normal range).

Mr. A's sister asked him to move out. He had difficulty accepting this situation and came up with a number of excuses as to why he could not move out on his own. He refused assistance from a clinic social worker in finding a board-and-care home or other living arrangements.

During a subsequent routine physical examination, a nodule was discovered on Mr. A's prostate. A laboratory evaluation revealed an elevated prostate-specific antigen count. No further workup could be done, however, because of his refusal. Mr. A was educated about the seriousness of possible prostate cancer and its treatability, if

diagnosed early, and was encouraged to get treatment for this condition, but he continued to refuse treatment, probably because of underlying paranoia. He was alerted about possible consequences of refusing treatment, and his informed consent not to receive treatment was obtained. At the time of his decision, Mr. A was considered to have the legally mandated decision-making capacity for deciding not to be treated.

During the latest medication visit, Mr. A was fairly cooperative with the interviewer but made poor eye contact. Moderate orofacial and limb tardive dyskinesia and mild parkinsonism and akathisia were present. His speech was monotonic. His affect was flat, but his mood was slightly anxious. He did not report hallucinations or delusions but appeared guarded and suspicious. Other staff working with Mr. A described him as being unwilling to discuss his symptoms, probably because of a fear of being hospitalized again. He stated that he was doing well and had adequate sleep (about 6 hours per night, with some middle insomnia), appetite, and energy.

Mr. A's medications at this time included haloperidol, 2 mg at bedtime, for psychotic symptoms, trazodone, 25 mg at bedtime, for insomnia, and levobunolol, 0.5% ophthalmic solution, for glaucoma. Mr. A asserted that he was taking his medications regularly. Although he had been informed several times about the benefits of switching to a newer atypical antipsychotic medication, he continued to refuse this medication because he feared that it would not be helpful for him and that his auditory hallucinations would return if he switched to a different medication. He also refused to participate in individual or group psychotherapy and dropped out of research but attended the medication clinic regularly.

DISCUSSION

In summary, Mr. A had no history of psychiatric illness until the age of 60. He presented with paranoid delusions and auditory hallucinations. Limited family psychiatric history was available; his mother had been admitted to an inpatient psychiatric unit at the age of 22, but her diagnosis was unknown. Results of medical examination and history were unremarkable. Neuropsychological performance did not suggest dementia. Moderate central and mild cortical volume loss was noted on the MRI. Although Mr. A had tried several psychotropic medications, he felt he benefited most from haloperidol. He developed tardive dyskinesia within a year of treatment.

Diagnostic Implications

Mr. A presented at the local hospital with psychotic symptoms. His initial symptom presentation was atypical, in that at age 60, he had no history of psychiatric illness, no known history of substance abuse, and no obvious medical illness. A number of evaluations, including history, physical examination, and laboratory tests, were conducted to rule out specific diagnoses. The differential diagnosis included 1) psychotic disorder due to a general medical condition (metabolic or other medical encephalopathy), 2) delirium, 3) psychosis secondary to substance abuse/dependence, 4) dementia with delusions and hallucinations, 5) mood disorder with psychotic features, 6) delusional disorder, 7) psychosis not otherwise specified or schizophreniform disorder, 8) brief reactive psychosis, and 9) schizophrenia with onset before versus after age 45.

1. Psychotic disorder due to a general medical condition (metabolic or other medical encephalopathy). Late age at onset and no past psychiatric history would point in favor of this diagnosis. According to history, physical examination, and laboratory results, however, there was no evidence of medical conditions known to cause psychiatric symptoms. The few neurological abnormalities identified were generally consistent with age. The MRI findings were nonspecific.

2. Delirium. Mr. A's physical examination yielded largely negative results. He had no fever and no medical conditions associated with delirium.

3. Psychosis secondary to substance abuse/dependence. There was no history of substance abuse. Laboratory results showed no evidence of acute drug or alcohol intoxication or liver disease.

4. Dementia with delusions and hallucinations. Points in favor of this diagnosis would include late age at onset, scattered abnormalities on neurological and neuropsychological examination, and moderate central and mild cortical volume loss with multiple areas of focal hyperintensities on the MRI. There are, however, several points against this diagnosis including an absence of marked memory disturbances, dysphasia, and significant visuoconstructional deficits on neurological examination, no decrement in cognitive performance over the follow-up period, and global neuropsychological performance within normal limits. On clinical mental status examination, Mr. A did not demonstrate any evidence of dementia, such as word-finding difficulties or inability to retain newly learned information over time. Neurological findings did not fit a clear pattern. Although not conclusive, the MRI results, along with history and mental status findings, pointed away from dementia as the cause of Mr. A's current psychotic symptoms. However, the possibility of a very early dementing process could not be ruled out.

5. Mood disorder with psychotic features. Mr. A never met the criteria for a manic or hypomanic episode; he never had a distinct period of abnormally and persistently elevated, expansive, or irritable mood lasting at least 4 days. He did not have inflated self-esteem or grandiosity, a decreased need for sleep (in fact, on numerous occasions he reported sleep disturbances and the need for more sleep), pressure to keep talking (he was usually quite guarded), flight of ideas, distractibility, increased goal-directed activity, or excessive involvement in pleasurable activities with a high potential for painful consequences. He also did not meet the criteria for major depressive episodes (i.e., for a period of at least 2 weeks, there was either depressed mood or loss of interest or pleasure in nearly all activities). In addition, he did not have psychomotor agitation or retardation, fatigue or loss of energy, feelings of worthlessness or excessive or inappropriate guilt, diminished ability to think or concentrate, or indecisiveness nearly every day or recurrent thoughts of death, suicidal ideation, or expansive or irritable mood. Mr. A's affect was typically described as flat. There was no history of marked mood fluctuations.

In terms of family history, the reason for Mr. A's mother's past psychiatric hospitalization was unknown and therefore was not helpful for evaluating the possibility of a mood disorder.

6. Delusional disorder. Late-onset psychosis with paranoid delusions may seem to favor the diagnosis of delusional disorder. Bizarre delusions, including thought insertion, and prominent auditory hallucinations would, however, rule out this condition.

7. Psychosis not otherwise specified or schizophreniform disorder. Mr. A could have been given a provisional di-

agnosis of psychosis not otherwise specified or schizophreniform disorder at the initial presentation, but the follow-up showed persistence of some symptoms of schizophrenia.

8. Brief reactive psychosis. When questioned about the onset of his symptoms, Mr. A denied having any recent stressors in his life.

9. Schizophrenia. Mr. A met all DSM-IV criteria for schizophrenia. One issue was whether he might have had onset of illness before age 45. Pointers against this possibility include a lack of prodromal symptoms, functional decline, or history of treatment before the age of 60. Mr. A had had a stable vocational history, and he had been able to function successfully. Although he was always somewhat of a loner, he had been able to sustain some relationships.

At follow-up, Mr. A was comprehensively reevaluated as part of his research participation. Baseline neuropsychological testing (including assessments of learning and memory) did not reveal deficits consistent with dementia, and his isolated cognitive deficits did not fit a pattern characteristic of a known neurological disorder. He did not experience any significant mood changes. Although Mr. A discontinued research participation after 4 years, he continued to be followed clinically. Earlier repeated neuropsychological evaluations were generally in the average range and showed no evidence of a progressive decline. Mr. A's cognitive strengths and weaknesses did not suggest any common neurological illness but were consistent with the pattern commonly seen in schizophrenia (4). He continued to take haloperidol, and he experienced a significant improvement in positive symptoms. Given Mr. A's symptoms and course of illness, his diagnosis would be late-onset schizophrenia.

At the initial presentation, Mr. A's clinical subtype was paranoid. The clinical picture does change over time, however, especially with neuroleptic treatment. In the present case, the alternative subtype diagnosis at follow-up would have been either undifferentiated or residual.

Late-Onset Schizophrenia

Late-onset schizophrenia is predominantly of the paranoid subtype. According to DSM-III-R, the onset of symptoms, including prodromal symptoms, must be after the age of 45. DSM-IV, however, does not specify the term "late onset," nor does it set an upper age limit for the diagnosis of schizophrenia. Often, the most notable symptom of late-onset schizophrenia is a bizarre, persecutory delusion. A number of patients experience systematized delusions of physical or mental influence. Auditory hallucinations are the next most prominent psychotic symptom. Schneiderian first-rank symptoms as well as mood symptoms may be seen in patients with late-onset schizophrenia. A review of the literature suggests that 13% of all hospitalized patients with schizophrenia had onset of illness in their 40s, 7% had onset in their 50s, and only 3% first presented after age 60 (5). Most studies report that late-onset schizophrenia is two to 10 times more common in women than in men (6–9). The higher prevalence of late-onset schizophrenia among women may result from postmenopausal neuroendocrine changes, psychosocial stressors, or unknown neurobiological factors. Most studies suggest a chronic course for late-onset schizophrenia (5, 10–12).

Reported similarities between late-onset schizophrenia and the typical early-onset schizophrenia include severity of positive symptoms, chronicity of course, sensory impairment, family history of schizophrenia, early childhood maladjustment, number of minor physical anomalies, increased mortality, overall pattern of neuropsychological impairment, nonspecific brain imaging abnormalities, and qualitative response to neuroleptics (13–16). In comparison to patients with early-onset schizophrenia, those with late-onset schizophrenia have a higher female-to-male ratio, less severe negative symptoms, less severe impairment in learning and abstraction, and a more intact semantic network. In adolescence and early adulthood, patients with late-onset schizophrenia also have had better premorbid functioning. In addition, patients with late-onset schizophrenia may have a larger thalamus, as shown on MRI, and seem to need lower doses of neuroleptics than age-comparable patients with early-onset schizophrenia. In summary, it appears that although there is a similar predisposition and probably similar brain lesions in patients with early-onset and late-onset schizophrenia, patients with late-onset may have a less severe form and a neurobiologically distinct subtype of the illness (15). Discontinuation of neuroleptics tends to exacerbate psychotic symptoms. Infrequently, spontaneous remissions may occur.

Late-onset schizophrenia is still a controversial entity. Beginning with the Kraepelinian notion of dementia praecox (17) and recently exemplified by DSM-III, the conventional wisdom limits the age at onset of schizophrenia to adolescence and young adulthood. It is postulated that negative symptoms, personality and cognitive deterioration, and social dysfunction typically associated with schizophrenia are typically lacking in patients with later onset of the illness. For this reason, various terms such as paraphrenia, late paraphrenia, and paranoia have been employed to describe late-onset paranoid psychotic disorders. One problem with such nosology, however, is that these terms have been used by different authors with varying definitions and do not have consistent clinical criteria (5).

Psychosis at any age can be due to any one of multiple causes, including substance use, specific brain disorders (e.g., tumors), and other general medical conditions. In later life, the likelihood of new-onset psychosis being secondary to identifiable brain disorders increases, and these must first be ruled out. Nonetheless, there is a proportion of patients, as illustrated by Mr. A, whose late-onset psychotic disorder is similar to that of patients with early-onset schizophrenia, particularly the paranoid type, in terms of symptoms, course, and treatment response. Furthermore, neuropsychological and brain imaging findings, as well as follow-up data, do not support alternative diagnoses. It is worth noting that paranoid schizophrenia at any age is characterized by relative paucity of negative symptoms, absence of pronounced deterioration, and better prognosis than other clinical subtypes such as disorganized schizophrenia (18). We believe that on the basis of the overall clinical picture, the most appropriate diagnosis for individuals such as Mr. A is late-onset schizophrenia. Making a distinct categorization of late-onset schizophrenia and continuing research on this topic are necessary to learn more about the factors associated with delayed onset of schizophrenia.

Treatment Considerations

Pharmacological. Mr. A was treated with an antipsychotic (haloperidol). Because of the potential side effects associated with neuroleptic use, informed consent was obtained and documented in his medical chart. At the

time of his first symptoms, the only atypical antipsychotic medication available was clozapine, which, in the elderly, is recommended only for severe, refractory cases (19). The starting dose of haloperidol was 10 mg b.i.d.; this would generally be considered too high for a person with late-onset schizophrenia. The dose was later reduced to 10 mg/day and subsequently to 6 mg/day. His last maintenance dose was 2 mg at bedtime. Given the sensitivity of older patients to side effects, it is critical to prescribe antipsychotics in doses that are considerably lower than those used in younger adults (19).

Mr. A had a partial response to haloperidol; his auditory hallucinations remitted and his delusions improved, although mild paranoia persisted, along with some residual symptoms. He also developed parkinsonism, akathisia, and, within a year of treatment, tardive dyskinesia. Older patients are more likely to develop tardive dyskinesia even with lower doses (20, 21). Vitamin E did not improve his tardive dyskinesia.

Despite recommendations from his treating physician to switch to an atypical antipsychotic medication, Mr. A continued to take haloperidol. He was given information about the risks and benefits associated with typical and atypical antipsychotics, but he was unwilling to change medicines because of fears that his positive psychotic symptoms would return.

Psychosocial. Mr. A did not follow up with recommendations to participate in individual or group therapy that could improve his coping skills. Possible reasons for his refusal include the existence of better than average social supports, along with a lack of awareness of his limitations and of the benefit he might derive from therapy, the fact that he was always a loner, and his continued underlying suspiciousness toward others. It appeared that Mr. A was overly dependent on his older sister in terms of housing, financial, and social support. It would be important to develop alternative resources for him so that he would be prepared if his sister carried through with her plans to live without him.

Ethical/Legal Considerations

The two main ethical/legal issues in Mr. A's care were his continuing tardive dyskinesia and the prostate nodule that remained undiagnosed.

Mr. A was informed about the risk of tardive dyskinesia when he started neuroleptic medication and continued to be reeducated about his options (i.e., switching to an atypical antipsychotic medication) at follow-up visits. He was, however, less concerned with medication side effects and more attuned to the fact that his medication was keeping his symptoms under control.

The other ethical/legal issue that affected Mr. A's care was his unwillingness to pursue evaluation and treatment of the prostate nodule. His physicians continued to educate Mr. A about the potential risks of letting this condition go untreated. His unwillingness to consent to evaluation and treatment might be attributed to his paranoia. Evidence from his clinical and neuropsychological evaluations demonstrated that he was capable of understanding the risks, benefits, and alternatives to treatment. Thus, he was entitled to make his own health care choices. Nonetheless, it is important to continue to provide Mr. A with education about his condition and to monitor his mental status to determine whether he remains competent and free of the influence of delusions.

Prognostic Implications

A comprehensive assessment of Mr. A revealed several good prognostic indicators (18). These include later onset of schizophrenia, paranoid subtype, relatively good premorbid adjustment with a supportive social network, treatment initiated early in the course of illness, only mildly impaired neuropsychological functioning, stable living situation (at least for the time being), and neuroleptic medication compliance. Negative prognostic indicators that cloud the picture include persistence of some symptoms, male gender, development of tardive dyskinesia, some treatment noncompliance, impaired insight into his needs and deficits, and the undiagnosed prostatic nodule.

Mr. A should continue to receive antipsychotic medication, with careful monitoring of side effects. As he ages, Mr. A's dose requirement and tolerance are likely to decline, whereas his risk of and sensitivity to side effects may increase.

In terms of suicide risk, Mr. A is now past the phase of the greatest risk (5 to 10 years after diagnosis of schizophrenia). The fact that he does not have command hallucinations further decreases his risk. In addition, he denies suicidal ideation and has never attempted suicide in the past. On the other hand, as a single, elderly white man, Mr. A is in the demographic group with the highest risk of suicide (22).

Research has shown that outpatients with schizophrenia are not necessarily at greater risk of developing dementia (23), but, of course, this risk will increase with age, as it does for the population at large. Thus far, however, repeated neuropsychological and clinical assessments have found Mr. A's cognitive functioning to be stable.

In terms of his psychosocial functioning, it is important to make long-term plans for Mr. A's future care needs. Previous plans to move Mr. A to a board-and-care facility failed because of a lack of follow-through and support from Mr. A's sister. It is important to enlist her support and to make plans while his functioning is still stable.

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