The Seoul Journal of Medicine
Vol. 33, No. 3: 195-202, September 1992

A Study on Selective Cerebellar Degeneration Following β-Fluorethyl Acetate Poisoning†

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= Abstract = **This is to report clinical and radiological findings in 7 patients with selective cerebellar degeneration following β-fluoroethyl acetate intoxication. β-Fluoroethyl acetate, an ethyl ester of fluoroacetate which is a metabolic inhibitor of Krebs cycle is used as a rodenticide. Following initial stage of coma from intoxication, the patients woke up to show selective cerebellar dysfunction, often so severe as not to be able to sit or stand unsupported. They improved gradually over several months to years with variable degrees of residual cerebellar dysfunction. Gait disturbance and dysarthria were the most prominent and persistent factors of the cerebellar dysfunction, whereas mild nystagmus was rarely seen. Cognitive function was not impaired. Cerebellar atrophy became noticeable on CT and MRI 4 weeks after poisoning, and progressed over time even with clinical improvement. Cerebellar degeneration contrasts with pallidal degeneration following carbon monoxide poisoning. β-Fluoroethyl acetate may be selectively toxic to the cerebellum.**

Key Words; *β-fluoroethyl acetate, Fluoroacetate, Cerebellar degeneration*

INTRODUCTION

Sodium fluoroacetate(Compound 1080) is one of the most toxic chemicals ever produced, and its lack of taste and smell makes it an ideal pesticide. It was introduced as a rodenticide and reported on by Kalmbach in 1945. Even though it was very effective in controlling pests, its use became limited only to licensed personnel because of human fatalities and ecologic concern that the entire animal population of a poisoned area may be destroyed. It is so toxic that cats and dogs which eat dead rats killed by this poison will be killed. Monoacetin and others were tried for its poisoning, but it has no specific antidotes (Chenweth *et al.* 1951; Taitelman *et al.* 1983). Its use was banned in Korea in 1966. However, its ethyl ester form, β-fluoroethyl acetate (Fratol®) is still available to a general public as a very

Received July 1992, and in final form September 1992.

† This study was supported by Seoul City Boramae Hospital Research Fund.

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effective pest control agent with less than optimal regulation. It is available in a 30 ml bottle which contains 2% β-fluoroethyl acetate solution. Even though it is about one-tenth as toxic as sodium salt, toxicity is one of the highest. As a result, many human casualties are reported annually. One general hospital had 73 patients with Fratol[R] poisoning over 2 years when 529 patients were admitted because of drug ingestion (Kim *et al.* 1985).

Between 1984 and 1989, we saw 7 patients with neurological complications after Fratol[R] poisoning. They had a unique and stereotypical clinical syndrome of selective cerebellar dysfunction. Long-term neurologic complication has been reported in only two communications (Pridmore 1978; Trahes *et al.* 1983). Trahes *et al.* (1983) described a girl with cerebellar degeneration, but the details were lacking in the report. We would like to describe the details of our observations on this unique syndrome with emphasis on its interesting features. Fluoroacetate is an inhibitor of cellular respiration like carbon monoxide (CO) and cyanide. In contrast to global encephalopathy and basal ganglionic syndromes seen in CO and cyanide poisoning, cerebellar degeneration is the only neurologic syndrome we have encountered so far from β-fluoroethyl acetate poisoning. We will discuss the pathophysiology and significance of this syndrome.

MATERIALS AND METHODS

Between 1984 and 1989, we saw 7 patients at the Department of Neurology, Seoul National University Hospital and Affiliated City Hospital whose clinical presentation was acute onset cerebellar dysfunction after Fratol[R] poisoning. All poisoning was due to suicide attempts. Initial management of acute poisoning was mostly done by other hospitals in the local area. Three patients were seen many years after the initial poisoning, but four patients could be followed from the early stage. Patients were first seen by us from one day to 14 years after the poisoning. Follow-up ranged from 6 months to 6 years. Medical records were obtained and history was reviewed with the patients. Information was obtained about acute poisoning, amount ingested, initial condition and management, and later development of neurological problems and chronological follow up. The amount ingested was calculated based on the history. Fratol[R] comes in a 30 ml bottle of 2% solution, containing 0.6 gm of β-fluoroethyl acetate.

RESULTS

Ages ranged between 18-51 years (mean 31). All were women except for one. Amount of ingestion varied from 0.6-1.8 gm (mean 1.2 gm). No one suffered hypoxemia, seizures or other metabolic derangements in the acute stage. From review of the histories and follow-up, we were able to divide the clinical course into three phases: acute phase, when patients are suffering from stupor and coma most likely due to acute toxicity of fluoroacetate; recovery phase, when patients are waking up from initial state of decreased mental status, and manifesting symptoms and signs of cerebellar dysfunction with resolution with time; and lastly chronic phase, when they are lefted with persistent cerebellar dysfunction. However, one patient continued to show small improvements even after many years. Cerebellar dysfunction was most prominent by history and examination immediately upon recovery from coma. All patients were "shaky", unable to sit or stand, severely tremulous so that they could not feed themselves, and hard to understand because of dysarthria. They improved over months to several years and then plateaued. On examination, the most prominent neurological findings were gait ataxia and dysarthria. Limb ataxia tended to be moderate, and rare nystagmus was mild, if present. There was no rigidity or bradykinesia. They tended to be hypotonic. Spasticity could be seen in the acute phase and immediately after swakening, but disappeared as they were followed up. Upon recovery from acute poisoning, they were able to return to work as

housewives or to school even with severe motor disability. Cognitive deterioration was not a factor in disability by history. Minimental status examinations were normal.

Illustrative cases are presented in the case records.

CASE RECORDS

Case 1 (SNUH #15588869-0) is a 51 year old woman, who ingested 1.2 gm of β-fluoroethyl acetate (2 bottles of FratolR) for suicidal purpose at age 39. She was unconscious for 18 days, and was discharged 7 days after recovery from coma. On discharge, she had to crawl because she was not even able to sit. It was impossible for her to communicate verbally because of dysarthria. After 6 months, she was able to walk alone with severe ataxis. When first seen by us in March, 1984 at age 46 (7 years after drug ingestion), she had dysarthria, unsteady gait, markedly impaired rapid alternating movement and severe dysmetria. Dysarthria was characterized by slow, explosive, staccato and scanning nature and a laborious effort to pronounce each syllable correctly and modulate the rhythm. There was a mild gaze dependent nystagmus in the horizontal directions. Cognitive function was normal. Muscle tone was normal. DTRs were slightly increased, and toes were downgoing. Sensory examination was normal. She continued to show a slow improvement. Currently, she is ambulatory with mildly impaired tandem gait. She continues to have severe dysarthria. Nystagmus is not seen. The CT scan showed diffuse severe cerebellar atrophy.

Case 4 (YCH #25975) was a 35 year-old woman, who was unconscious for 7 days after taking 1.2 gm of β-fluoroethyl acetate. On awakening, she was too dysarthric to be intelligible, and was unable to sit unaided. A month later, she had severe cerebellar dysarthria, mild dysphagia, marked intention tremor, hypotonia and generally increased reflexes with down-going toes. Cognitive function was normal. Eye movements were normal without nystagmus. She was not able to sit unaided. Three months after, she was able to sit unaided, but had difficulty in standing even with assistance. She was able to walk a few steps with assistance 6 months after. Ten months later, she was barely able to walk with a side rail. She continues to have dysarthria and dysphagia even though much improved. Limb ataxia was still moderately impaired. The CT scan done 3 days later after drug ingestion showed obliteration of the quadrigeminal and prepontine cisterns, suggesting cerebellar swelling. Follow-up CT scan done 6 months later showed widening of quadrigeminal and prepontine cisterns, and mild cerebellar atrophy. The MRI scan done a year later showed severe cerebellar atrophy.

Clinical and radiological findings are summarized in the Table 1. The patients were young adults. The predominance of females probably reflects different means of suicide in different sexes. The range of dosage of β-fluoroethyl acetate was 600 mg to 1800 mg with a mean of 1200 mg. This is close to the lethal dose of β-fluoroethyl acetate is about ten times that of fluoroacetate which is 2-5 mg/Kg in humans, and average weight is 60 Kg). All of

Table 1. Summary of cinical and radiological findings

Case No.	1	2	3	4	5	6	7
Age/Sex	51/F	30/F	25/F	35/F	23/F	18/M	36/F
Amount ingested(gm)	1.2	1.8	1.2	1.2	0.6	1.2	1.2
Coma duration (days)	18	7	12	7	13	20	7
Time needed to walk unsupported	6mo	3wk	6mo	imposs	6mo	12mo	imposs*
Initial evaluation	7yr	1wk	4yr	1mo	1d	4wk	14yr
Follow-up	5yr	6mo	6mo	1yr	6mo	6yr	6mo
Dysarthria	++	+	+++	++	++	++	+++
Ataxic gait	+	+	+	+++	++	+	+++
Cerebellar atrophy	++	+	+++	++	++	+++	++

+: mild ++: moderate +++: severe

*: unable to walk even with support

them had decreased levels of consciousness for more than 7 days (range 7-20 days with a mean of 12 days). Patients typically came in for gait ataxia. As other symptoms, walking difficulty was the most severe right after recovery from coma, and gradually improved so that patients were usually able to walk 6 months after without support. One patient (case 4) continued to have walking difficulty without support after one year on last follow-up, and another patient (case 7) continued not to be able to walk with support even 15 years after poisoning. Dysarthria and gait abnormality in the Table 1 are from the last follow-up examination. Four patients had serial CT or MRI scans as they were followed from the early stage. The scans showed swelling of posterior fossa in the first week. Cerebellar atrophy progressed on follow-up. Patients improved clinically when cerebellar atrophy progressed on the brain scans (See case 4).

DISCUSSION

The pharmacologic properties of fluoroacetate have been studied extensively (Liebecq and Peters 1949; Cheonweth 1949). The toxicity is known to be produced by formation of fluorocitrate with oxaloacetate. Fluorocitrate in turn blocks aconitase in Krebs cycle, thus inhibiting generation of energy. The concept of "lethal synthesis" that a substance becomes "lethal" only were it has been transformed by the actions of tissue enzymes was first contributed by Peters (1952) in his Croonian lecture. The presence and importance of hypocalcemia are still being debated (Taitelman *et al.* 1983). Szerb and Issekutz (1987) reported that fluoroacetate can increase the stimulation-induced overflow of glutamate by inhibiting energy production of glia thus blocking reuptake of released glutamate. Animal experiments showed variable pharmacological actions in different species (Chenoweth 1949). LD_{50} varied from 0.06 mg/Kg in dogs to well over 500 mg/Kg in South African clawed toads. A lethal dose of sodium fluoroacetate in humans is 2-5 mg/Kg. The qualitative character of toxicity was also quite variable. Rabbits suffered mainly cardiac toxicity with ventricular fibrillation; rats, respiratory depression; dogs, central nervous system with epileptiform convulsions; and rhesus monkeys, mixed toxicity of heart and central nervous system. Herbivorous animals tended to manifest cardiac toxicity; carnivorous animals, per primum central nervous system convulsions and depression; and omnivorous species, both caridac and central nervous system toxicity.

Limited reports outside Korea suggest that humans mainly suffer from cardiac and CNS toxicity (Gajdusek and Luther 1950; Reigart *et al.* 1975). Seizures were a very common manifestation. In reports from Korea, the pattern was different from what was reported elsewhere (Kim *et al.* 1985; Yu *et al.* 1986). In one series of 73 patients (Kim *et al.* 1985), 65.6% of patients had mental status changes on admission, however, seizures were seen in only 27.4%. EKG changes were not common (16.5%), and ventricular fibrillation was seen in only 4.5%. Fatalities were due to acute respiratory failure in 7 and acute renal failure in 5. In another series of 30 patients (Yu *et al.* 1986), mental state changes were seen in 46.7%, and seizures occurred in 16.6%. The mortality of fluoroacetate poisoning is high, and the reports of survivors are rare Gajdusek and Luther 1950; Reigart *et al.* 1975; Pridmore 1978; Trahes *et al.* 1983). However, survivors are commonplace in Korea because of the high incidence of poisoning due to availability and the reduced toxicity of the ethyl ester form (mortality 15.4% in Kim *et al.* 1985). All of our patients with cerebellar degeneration were in coma for many days (7-20 days). They received general supportive care for drug ingestion. Some were treated with monoacetin and ethanol. None of them had seizures. Two of them were intubated, but none suffered hypoxemia. As patients woke up from coma, they showed prominent cerebellar dysfunction. This was maximal on awakening from coma, and gradually improved over a period of several months to years. One patient

(Case 1) was seen seven years after poisoning, and still continued to show small but significant improvement on follow up.

Dysarthria was very severe and characteristic. Initially nearly all of them were not able to speak or make intelligible sounds. Dysarthria fitted very well with the description and characteristics of cerebellar dysarthria (Gilman *et al.* 1981). Speech was slow, explosive, staccato, scanning, harsh, poor in melody and accent. They tried with laborious effort to articulate each syllable correctly and smooth the rhythm without success and with great frustration. The best analogy would be "an unpracticed student laboriously reading lines of poetry, making special effort to decipher the thythmic structure of the verse" (Gilman *et al.* 1981). But this student does not have a good mental note to follow, and our patients know exactly what to do and simply can not do it. Gait ataxia was another disabling difficulty. Nearly all were not even able to sit unaided initially, and took many months to stand without help and walk. On testing they had severe motor incoordination and unsteadiness. Dysarthria and ataxic gait were the most severe, and remained the longest. Limb incoordination was moderately impaired also. In contrast, nystagmus was only minimal if present, and disappeared earlier than other signs.

Localization of speech function in the cerebellum is controversial, and is well discussed in Gilman *et al.* (1981). In his classic study of patients with gunshot and shrapnel injuries to the cerebellum, Holmes(1917) concluded that the vermal portion of the cerebellum was important in dysarthria. However, Brown(1959) observed that dysarthria developed at about the same time in the progression of degenerative cerebellar disease as limb ataxia rather than gait ataxia. This observation moved the locus for speech regulation out of the vermis into the lateral cerebellar hemispheres. Further observations (Amici *et al.* 1976; Lechtenberg and Gilman 1985) provided support that the cerebellar hemispheres, especially the left side, are important in speech regulation. Gait ataxia is caused by midline lesion especially in the anterosuperior part, best exemplified in alcoholic cerebellar degeneration (Victor *et al.* 1959). Many parts of the cerebellum participate in ocular motor control, and ocular motor abnormalities sometimes can not be easily localized to a spepcific cerebellar region. Based on clinical data of severe dysarthria, gait ataxia, and limb incoordination, it is difficult to localize the main pathology to a specific area, and seems more likely that there is generalized cerebellar damage. Minor, if any, nystagmus was a very interesting, consistent observation in every patient. We are not sure this will help to further localize the lesion.

Even though the patients were seen over a 6 year period, the initial times of drug ingestion span over 16 years. There has been no study on systematic follow-up of survivors from Fratol^{R} poisoning. As far as our experiences go, this unique cerebellar degeneration is the only pattern of neurologic syndrome from β-fluoroethyl acetate poisoning. We were struck by the rather uniform pattern of selective cerebellar dysfunction and lack of other expected patterns of hypoxic damage such as global enceophalopathy and basal ganglionic syndrome that we have so often encountered in CO (Jeon and Lee 1989) and cyanide poisoning (personal observation). As the pathophysiology of this poisoning is inhibition of cellular respiration, not unlike carbon monoxide or cyanide poisoning, it is not surprising to find a report of a patient with global encephalopathy (Pridmore 1978). However, this patient had a prolonged ventricular asystole for 10 minutes which might have damaged the brain in an independent way from fluoroacetate and complicated the picture. He was left with dementia, epilepsy, tetraplegia, increased tone with cogwheel rigidity, and cortical blindness. It is possible that the cerebellar dysfunction might have been overshadowed by rigidity and spasticity.

Cerebellar degeneration has been reported secondary to phenytoin toxicity (Ghatak *et al.* 1976), alcoholism (Victor *et al.* 1959), hyper-

pyrexia (Lee *et al.* 1989), Ara-C (Winkelman and Hines 1983), and as a paraneoplastic syndrome (Brain *et al.* 1951). One case of cerebellar degeneration secondary to fluoroacetate poisoning was reported by Trahes *et al.* (1983). The patient was a 15-year old who attempted suicide by fluoroacetate. She became comatose, and later developed seizures. On awakening after 2 weeks, she had severe cerebellar dysfunction. Details of cerebellar dysfunction are not mentioned. During the following 18 months, she had complaints of memory disturbances and depressive behavior with later resolution. Moderate cerebellar ataxia remained. Diffuse brain atrophy was seen in the CT scans done 2 weeks and 18 months after drug ingestion without interval change. No mention was given to cerebellar atrophy. In our series, cerebellar atrophy was more selective and observed only in the late stage after months in the follow-up scans. Swelling of the cerebellum was seen in CT scans done in the acute phase, which later changed into atrophy. Atrophy of the cerebral hemispheres was mild, if any.

Selective involvement of the cerebellum is quite intringuing. Mettler and Sax(1972) were able to produce selective cerebellar degeneration in Rhesus monkeys by intravenous infusion of azide. They suggested that the selectivity could be explained by hemodynamics and vascular anatomy. However, other poisonings that would cause similar hemodynamic changes as CO and cyanide produce a quite different pattern of selectivity. All these poisons are inhibitors of cellular respiration (CO has a more important action of oxygen delivery inhibition), and cause cardiac depression and hypotension. It has been repeatedly shown in humans and animals that CO and cyanide produce selective degeneration of basal ganglia, cerebral white matter or both. Not a single case of selective cerebellar degeneration from CO poisoning has been reported to our knowledge. Cerebellar Purkinje cells are more susceptible to hyperthermia than other neurons (Malamud *et al.* 1946). Lee *et al.* (1989) reported a case of cerebellar degeneration in neuroleptic malignant syndrome, and reviewed cases of cerebellar degeneration in other conditions of hyperpyrexia such as heat stroke, fever therapy, and post-thyroidectomy. It may be suspected that our patients were somehow hyperthermic at the time of intoxication, or became hyperthermic as a result of intoxication, thus developing selective cerebellar damage. As fluoroacetate is a metabolic inhibitor, it is unlikely to cause hyperthermia. If hyperthermia is contributing to this selectivity, we would expect that cerebellar degeneration be seen in CO poisoning, because CO poisoning in Korea occurs because of "Ondol" which is a way of heating rooms. Virtually all CO poisoning patients were left for hours in hot poorly-ventilated rooms before being found. One interesting aspect of fluoroacetate is that it is rather selective in inhibiting glia rather than neurons (Clarke *et al.* 1970; Quastel 1974). Fluoroacetate is more readily taken up by glia through a Na+-dependent transport system like acetate (Gonad and Quastel 1966). Thus it inhibits Krebs cycle preferentially in the glia. Characteristically it does not decrease the overall consumption of oxygen in brain slices because it blocks only the smaller energy cycle in glia (Clarke *et al.* 1970; Lahiri and Quastel 1963; Quastel 1974). Neither does it affect Ca++-dependent release of glutamate by electrical stimulation in neurons (Szerb and Issekutz 1987). Whether it plays a role in selective cerebellar damage is not clear.

The question of selective neuronal vulnerability or pathoclisis has been an interesting one, but has still not been answered. It is interesting to note that fluoroacetate, CO, cyanide, and axide are mitochondrial toxins, and produce rather selective damage to the CNS. Fluoroacetate inhibits Krebs cycle; and CO, cyanide, and azide inhibit cytochrome C oxidase, the last step in the respiratory chain. Inconsistency in patterns between sites of enzyme inhibition and pathology stands out. Cyanide and azide poisoning which inhibit the same enzyme cytochrome C oxidase produce a different

pathology of basal ganglia and cerebellar degeneration respectively. In contrast, fluoroacetate and azide which inhibit different enzymes produce the same cerebellar degeneration. Many patterns of CNS involvement are observed in mitochondrial encephalomyopathy, and do not follow any specific patterns in relation to enzyme defects (Zeviani *et al.* 1989). Acute poisoning will not be a good model for human mitochondrial encephalomyopathy as it reflects more acute and probably near complete blocking of mitochondrial function with cardiorespiratory depression. However, studies of chronic poisoning with these toxins may give some insights into selective neuronal vulnerability and human mitochondrial encephalomyopathy.

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