An Outbreak of Peripheral Neuropathy in a Prison

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Abstract

Prisoners are at risk for both physical and psychological diseases. Here, we report an outbreak of peripheral neuropathy in a prison in northeast Thailand. Between July and December 2014, there were 88 male prisoners at Bueng Kan Provincial Prison in Bueng Kan, Thailand suffering from peripheral neuropathy out of a total of 1,464 prisoners (6.01%). The common age range was 20–39 years (58 patients; 65.91%). The three most common features were hyporeflexia/areflexia of the lower extremities (36 patients; 83.72%). On laboratory vitamin B1 deficiency was detected in 4/5 patients, positive rhinovirus polymerase chain reaction in 3/4 patients, positive *Mycoplasma pneumoniae* IgM in 1/12 patients, and positive urinary arsenic in 4/7 patients. A dT vaccination was given on October 14 during the outbreak. This was a large outbreak of peripheral neuropathy in male prisoners. There are several possible causes of this outbreak including vitamin B1 deficiency, dT vaccination, arsenic toxicity, rhinovirus, and *Mycoplasma* infection.

Introduction

Prisoners are at risk for both physical and psychological diseases such as hepatitis C virus infection, tuberculosis, and depression [1–3]. A study in Germany found epilepsy and peripheral nerve lesions to be the two most common neurological diseases among prisoners there [4]. Another report found that 679 out of 4,684 (14.5%) ex-Far Eastern prisoners of war suffered from neurological disease, optic atrophy, and peripheral neuropathy. In addition, it has been found that Parkinson’s disease may develop at a higher prevalence than in the general population, even several years after release [5].

In December, 2014, there was an outbreak of peripheral neuropathy at Bueng Kan Provincial Prison, located in Bueng Kan province in northeast Thailand. Twenty-nine prisoners were reported to suffer from muscle weakness in both legs and numbness in the hands and feet. There are several possible causes of peripheral neuropathy including vitamin B1 deficiency, vaccination, infections, or toxins such as arsenic [6–9]. Prisoners are a vulnerable population and at are risk for nutritional deficiency. Additionally, a few months prior to the outbreak, there was an outbreak of diphtheria all over Thailand. Thus, all prisoners had received diphtheria and tetanus vaccinations prior to the outbreak. A previous study showed that peripheral neuropathy in prisoners at a Bangkok Central Prison had been due to arsenic-contaminated water [8]. This study aimed to evaluate the prevalence and causes of the peripheral neuropathy outbreak at this provincial prison.

Methods

This study was conducted at Bueng Kan Provincial Prison in Bueng Kan, Thailand. The inclusion criteria were that subjects had experienced weakness or paresthesia between July and December 2014. The clinical diagnosis of peripheral neuropathy was made based on the presence of one or more of the following: weakness of the arms and/or legs, paresthesia of
any part of the body, autonomic symptoms such as constipation, urinary incontinence, or erectile dysfunction. Electromyography was used as a confirmatory test.

Laboratory workups were performed on both symptomatic and asymptomatic subjects, at the subjects’ voluntary discretion. These workups aimed to find possible causes of peripheral neuropathy, such as viral infection or toxic substances, and included a throat swab, a rectal swab, a blood sample, a urine sample, a cerebrospinal fluid sample, and a measurement of vitamin B1 levels. All workups were analyzed at the Department of Laboratory Medicine at Bangkok’s Chulalongkorn Hospital. An environmental survey was also performed to evaluate the sanitary conditions of the prison, the environment, and the prisoners’ nutritional/food data.

**Results**

There were 88 male patients who were diagnosed as suffering from peripheral neuropathy out of a total of 1,464 prisoners (6.01%). The common age range was 20–39 years (58 patients; 65.91%). Most patients (59 patients; 67.04%) lived in sleeping units 5 and 6 (Table 1). The three most common features were hyporeflexia/areflexia of the lower extremities (36 patients; 83.72%), sensory deficit (34 patients; 78.81%), and motor weakness (31 patients; 72.09%), as shown in Table 2.

The laboratory workups found various abnormalities in prisoners suffering from peripheral neuropathy. According to electromyography results, 5 out of 6 patients (83%) had sensorimotor axonal polyneuropathy. Vitamin B1 levels were low in 4 out of 5 male prisoners with peripheral neuropathy, while only 2 out of 5 male prisoners without peripheral neuropathy had low vitamin B1 levels (Table 3). Other workup results showed that 75% of prisoners with peripheral neuropathy were positive for rhinovirus (according to a throat swab), 8% were positive for *Mycoplasma pneumoniae* IgM, and 57% had high urinary arsenic levels (Table 3).

The first peripheral neuropathy patient was reported on July 4, 2014. Later, there were two peaks of patients being reported, one in the week of September 28 to October 4 and the other in the week of November 11–15, with a higher number of patients being reported in the latter period. There was a total of 41 patients diagnosed in November. The dT vaccination was given on October 14 (Fig. 1).

Various environmental aspects of the prison were also taken into account. There are a total of 1,464 prisoners in the prison, 1,285 of whom are male and 133 of whom are female. The interior space of the prison has the capacity to hold 1,350 prisoners. There are six sleeping units with a total capacity of 571. Water for general use is ground water without standard water chlorination, while filtered tap water is used for drinking. With regard to food, breakfast and dinner consist of sticky rice with one side dish and lunch consists of a rice dish. Fruits are occasionally served. Meals provided for the prisoners contain between 0.70 and 2.03 mg of vitamin B1 per day, or 1.35 mg on average.
Discussion

This was a large outbreak of peripheral neuropathy in male prisoners. There are several possible causes of this outbreak including vitamin B1 deficiency, dT vaccination, arsenic toxicity, rhinovirus, and Mycoplasma infection (Table 3).

Vitamin B1 is a common cause of peripheral neuropathy in prisoners or ex-prisoners of war. Peripheral neuropathy without cardiac involvement may be the only clinical manifestation apparent in an outbreak, as was the case in this study [10]. A report from Cuba found that sensory deficit was the most common symptom (78.95%), the reported rate of which was comparable to that found in this study (78.81%). Although hyporeflexia was the most common finding (83.72%), it is a physical sign, not a symptom that patients complain about (Table 1). The low vitamin B1 levels in prisoners and prison diets being low in vitamin B1-containing foods act as supportive evidence for vitamin B1 deficiency as a cause of the outbreak. The percentage of male prisoners with vitamin B1 deficiency was higher than that of normal male prisoners (80 vs. 40%), as shown in Table 3. Though the average amount of vitamin B1 in meals served at the prison was higher than the recommended requirements (1.35 mg vs. 1.2 mg for males), vitamin B1 deficiency may occur due to low meat intake or to meals being served with white rice as opposed to other types. Polished or white rice contains lower amounts of vitamin B1 than brown rice. As has previously been reported, males are typically predominant in prison settings, in which the male:female ratio can be as high as 18:1 [11]. In addition, populations from northeast Thailand tend to have lower vitamin B1 levels than those from other parts of the country [12]. Up to 88% of laborers from the northeast were found to suffer from thiamine deficiency [12]. Vitamin B1 deficiency may not explain all aspects of this outbreak, such as the two peaks in the number of reported cases, as shown in Figure 1. There may, thus, be other contributing causes of this outbreak.

Rhinovirus may be another contributing factor. Between 20 and 75% of the laboratory tests in this study were positive for rhinovirus (Table 3). Two previous reports showed that enterovirus may be associated with neuropathy [13, 14]. For example, polio-like acute flaccid paralysis was found to be followed by an enterovirus D68 outbreak in Cleveland, USA [13]. It has also been shown that neural degeneration or dysfunction may occur due to cough-induced plasticity in the neural pathways [14]. Additionally, crowded sleeping units may be an aggravating factor for viral infection and other infectious agents such as Mycoplasma or tuberculosis [15, 16]. There have been reports of outbreaks of adenovirus serotype 14p1 and an influenza A/Fujian/411/2002 (H3N2)-like virus in prisons in the UK and Australia. Even though M. pneumoniae may be associated with acute motor axonal neuropathy [17], the evidence for acute M. pneumoniae as a cause of the outbreak in this survey was low (8%), as shown in Table 3.

Diphtheria and tetanus vaccines have been shown to be associated with neuropathy and Guillain-Barré syndrome [6, 18]. These vaccinations cannot explain the first peak of this outbreak (Fig. 1), which occurred prior to the mass dT vaccination on October 14. However, it may have been a contributing factor in the second peak.

There are some limitations to this study. First, not all patients were given all laboratory workups, as these were performed on a voluntary basis. Second, the amounts of vitamin B1 provided in meals was calculated based on estimates, not actual measurement. Finally, other possible causes of neuropathy were not fully investigated, such as hepatitis or HIV infection.
Conclusion

There are several possible contributing factors to the peripheral neuropathy outbreak in the prison. Vitamin B1 deficiency is a potential and treatable cause. Further evaluations of the prisoners may be needed.

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Statement of Ethics

The authors have no ethical conflicts to disclose.

Disclosure Statement

The authors declare no conflicts of interest.

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Fig. 1. Epidemic curve in Bueng Kan Prison, Bueng Kan province, Thailand. December 2014: n = 88.
Table 1. Baseline characteristics of patients with peripheral neuropathy during the outbreak

| Variables                  | n (%)          |
|----------------------------|----------------|
| Sex                        |                |
| Male                       | 88 (100)       |
| Age, years                 |                |
| 10–19                      | 1 (1.14)       |
| 20–29                      | 30 (34.09)     |
| 30–39                      | 28 (31.82)     |
| 40–49                      | 16 (18.18)     |
| 50–59                      | 10 (11.36)     |
| 60 and over                | 3 (3.41)       |
| Sleeping unit              |                |
| 1                          | 5 (5.68)       |
| 2                          | 9 (10.23)      |
| 3                          | 9 (10.23)      |
| 4                          | 4 (4.55)       |
| 5                          | 30 (34.09)     |
| 6                          | 29 (32.95)     |
| Hospital unit              |                |
|                            | 2 (2.27)       |

Table 2. Clinical manifestations of patients with peripheral neuropathy (n = 43) during the outbreak

| Signs and symptoms                  | n  | %    |
|-------------------------------------|----|------|
| Hyporeflexia/areflexia of lower extremity | 36 | 83.72|
| Sensory deficit                     | 34 | 78.81|
| Motor weakness                      | 31 | 72.09|
| Edema of lower extremity            | 15 | 34.88|
| Hyporeflexia/areflexia of upper extremity | 13 | 30.23|
| Erectile dysfunction                | 5  | 11.62|
| Constipation                        | 4  | 9.30 |
| Dysphagia                           | 3  | 6.98 |
| Leg cramp                           | 2  | 4.65 |
| Urinary retention                   | 1  | 2.32 |
Table 3. Laboratory findings of patients with/without peripheral polyneuropathy during the outbreak

| Specimens/tests | Variables                                      | Tested, n | Positive, n (%) |
|-----------------|------------------------------------------------|-----------|-----------------|
| Throat swab     | Respiratory viruses 16 type PCR<sup>a</sup>    | 4         | 3 (75%)<sup>a</sup> |
|                 | *Mycoplasma pneumoniae*                        | 7         | 7 (100%)        |
|                 | Enterovirus PCR<sup>b</sup>                   | 10        | 2 (20%)<sup>a</sup> |
| Serum           | Herpes simplex IgM                            | 12        | 2 (17%)         |
|                 | *Mycoplasma pneumoniae* IgM                   | 12        | 1 (8%)          |
|                 | *Mycoplasma pneumoniae* IgG                   | 12        | 8 (67%)         |
| Blood           | Lead                                           | 7         | 0               |
| Urine           | Arsenic                                        | 7         | 4 (57%)         |
| Stool           | Rhinovirus PCR                                 | 10        | 2 (20%)         |
| Vitamin B<sub>1</sub> | Male patients (prisoners)                    | 5         | 4 (80%)         |
|                 | Normal male prisoners                          | 5         | 2 (40%)         |
|                 | Normal female prisoners                        | 5         | 4 (80%)         |
|                 | Normal wardens                                 | 5         | 4 (80%)         |
| Electromyogram  | Sensorimotor axonal polyneuropathy            | 6         | 5 (83%)         |
| MRI             | Brain, spinal cord                             | 2         | 0               |

MRI, magnetic resonance imaging; PCR, polymerase chain reaction. <sup>a</sup>Positive for rhinovirus. <sup>b</sup>Enteroviruses family PCR for genetic locations: VP3/1, VP4/2, and 5’UTR long covering poliovirus (human enterovirus C serotypes PV-1, PV-2, PV-3), human coxsackievirus, human enterovirus 70 (EV-70), EV-71, and human rhinovirus A, but not Picornaviridae. <sup>c</sup>Vitamin B1 level tested by high-performance liquid chromatography and erythrocyte transketolase activity.