An Unusual Presentation of Bee Sting: Internal Jugular Vein–Sigmoid Sinus Thrombosis

Asuman Çelikbilek1, Ugur Yıldırım2, Aylin Okur2, Esef Bolat3, Mehmet Adam4, Tugay Atalay5

Bee stings are commonly encountered worldwide, in the rural population in particular. We present the case of a young woman who was admitted with a complaint of severe persistent headache after a bee sting. Her routine laboratory, serologic, genetic, and coagulation tests were normal. Inflammatory, autoimmune, tumor, and hepatitis markers were negative. Magnetic resonance imaging showed a filling defect in the right sigmoid sinus and internal jugular vein. Lumbar puncture revealed an opening pressure of 250 mmH2O. Based on the diagnosis of internal jugular vein–sigmoid sinus thrombosis, the patient was treated with anticoagulation therapy resulting in a complete recovery within two weeks.

Keywords: Bee sting, internal jugular vein, sigmoid sinus, thrombosis

INTRODUCTION

Bee stings are one of the commonly encountered insect bites in the world, in the rural population in particular (1). Various manifestations after bee stings, from slight local reactions to severe life-threatening events, have been described (2). However, neurological involvement is rare. Here we present the case of a young woman who developed internal jugular vein–sigmoid sinus thrombosis following a bee sting with no other etiology.

CASE REPORT

A 29-year-old woman from a rural area was admitted to our neurology outpatient clinic because of a 1-week history of severe persistent headache after a bee sting. Her headache started 6 h after the bee sting and gradually improved. From her past medical history, it was found that she had no allergic reactions, drug use, or chronic disease. On physical examination, she was afebrile and conscious, and her vital signs were normal. The site of the sting was on the anterior chest wall above the sternal margin with a little erythema and was 1×1 cm in size without any tenderness (Figure 1). On neurological examination, cranial nerves were intact and upper and lower meningeal signs were negative. On funduscopic examination, a complete papilledema accompanied by peripheral visual field defect was detected. Visual acuity was normal. Laboratory tests including complete blood count and renal, liver, and thyroid function tests were within normal ranges. Electrocardiogram and echocardiography were normal. Brain computed tomography (CT) scan revealed diffuse edema in both hemispheres, whereas contrast-enhanced venous phase of axial CT scan revealed a filling defect and nonvisualization of the internal jugular vein on the right side (Figure 2). Magnetic resonance angiography images confirmed the internal jugular vein–sigmoid sinus thrombosis without venous infarction (Figure 3a, b). Lumbar puncture was performed demonstrating a clear cerebrospinal fluid with no pleocytosis. Opening pressure was 250 mmH2O. In view of the clinical and radiological findings, a diagnosis of internal jugular vein–sigmoid sinus thrombosis was made. Regarding etiology, inflammatory, autoimmune, tumor, and hepatitis markers were negative. Serologic and coagulation tests and predisposing genetic factors were normal. Systemic heparinization was immediately started. Intravenous mannitol (400 cc/day) and oral acetazolamide (250 mg/day) were prescribed to manage the increased intracranial pressure. After 3 days, the medications were replaced with warfarin sodium (5 mg/day) in accordance with the optimization of international normalized ratio. Her headache completely resolved within 5 days. On follow-up, the visual field defect recovered, and she was discharged in good health within 2 weeks.

DISCUSSION

Allergic reactions are most commonly seen after a bee sting (2). Immunological responses vary according to an individual’s immune system. Classical local reactions such as sudden burning, pain, itching, redness, and local swelling are common. Systemic toxic effects and anaphylaxis appear due to neurotoxic, hemorrhagic, and hemolytic toxin effects of bee venom (3). Accordingly, vasculitis, cardiac arrhythmia, renal failure, and neurological and...
Coagulation disorders may occur (3). Although neurological involvement is rare, a varied spectrum of neurological manifestations, such as convulsions, subarachnoid hemorrhages, parkinsonism, cerebral infarction, coma, and encephalitis, are reported in literature (4-8). In general, bee venom contains a wide array of amines, peptides, enzymes, and other vasoactive and inflammatory mediators responsible for local and systemic reactions (2, 3). A major allergen is phospholipase A2, which acts as a cytotoxin and an indirect cytolysin and that also has neurotoxic activity by blocking neurotransmission in the neural synapse (3). Neurological complications after bee stings generally develop from within a few hours to weeks (4). The exact pathogenic mechanism of central neurological findings is still unclear. The proposed mechanism indicates possible neurotoxic effects of the venom, including enzymes such as hyaluronidases and phospholipases, allowing the venom to spread (3, 4).

Figure 1. The site of the bee sting was the anterior chest wall above the sternal margin with a little erythema and was 1×1 cm in size.

Figure 2. A contrast-enhanced venous phase of axial computed tomography scan revealed a filling defect and nonvisualization of the right internal jugular vein.

Figure 3. (a) A coronal 3D magnetic resonance angiography image revealed the thrombosis with no signal in the right sigmoid sinus and internal jugular vein. (b) An axial T1-weighted image showed the hyperintense thrombus in the right sigmoid sinus and internal jugular vein.
In literature, there are only two cases where patients presented with cerebral sinus thrombosis as a complication of bee sting till date (9, 10). Senthilkumaran et al. (9) reported a farmer who was stung by a bee on the left side of the upper lip, where a delay in the diagnosis enhanced the spread of infection; this was linked to the thrombophlebitis of the facial vein due to the thrombogenic substances in the venom. In another paper, a young boy in a rural population, which is particularly vulnerable to bee stings as they are encountered during field work, was reported to have bilateral cavernous sinus thrombosis with bilateral cerebral infarcts as a rare combination (10). Cavernous sinus thrombosis might have developed due to the spread of acute local inflammation caused by bee venom (10). Vasoactive and inflammatory mediators causing vasoconstriction, platelet aggregation, and retrograde stimulation of the superior cervical ganglion leading to occlusion of the terminal internal carotid artery have been postulated (10). Our patient had right-sided internal jugular vein–sigmoid sinus thrombosis, an unusual presentation of bee stings, with no other etiology. Her persistent headache completely resolved with anticoagulation treatment within 5 days. We suggested a similar hypothesis where neurological symptoms might be provoked by vasoactive, proinflammatory amines leading to vasoconstriction and platelet aggregation and possibly predisposing to systemic thrombogenic responses, although allergic reactions are not necessarily present (3, 4).

**CONCLUSION**

To our knowledge, this is the first report of internal jugular vein–sigmoid sinus thrombosis following a bee sting in the central Anatolia region. We report this case in view of its rarity as an unusual complication following a bee sting in a normal healthy person and to make clinicians consider this entity in the differential diagnosis of cerebral sinus thrombosis.

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