

The First Reported Case of Second Impact Syndrome: A Reexamination of Dr Fekete's Case Report from 1968

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Curr Res Concussion 2017;4:e55–e57.

Abstract

Background In the December 28, 1968 edition of the Canadian Medical Association Journal, Dr John Fekete described a 16-year-old hockey player with fatal cerebral edema following a brain impact while actively symptomatic of a concussion incurred 4 days ago. This case has been described as a “possible” case of second impact syndrome, an entity that was named in 1984 and purportedly first described by Schneider in 1973.

Method An audit of material in the public register of Dr Fekete's case was undertaken. Information sources included the newspaper report of the death, transcripts from a coroner's inquest held 12 days later, including the autopsy report, genealogical data available online, and available internet resources.

Results There was clear documentation of concussive symptomatology following an initial head injury and evidence of a medical assessment, thus fulfilling the “definite” clinical criteria for second impact syndrome as proposed. After 4 days of ongoing concussive symptomatology, a dramatic, rapid neurological deterioration took place following an apparently unremarkable body contact and fall on the ice while playing hockey. His primary brain pathology included cerebral edema.

Conclusion Cerebral edema may follow primary or secondary head injury, the latter comprising second impact syndrome. Dr Fekete's case, as described in the December 28, 1968 edition of the Canadian Medical Association Journal, should be recognized as the first description of this condition.

Keywords

- ▶ second impact syndrome
- ▶ brain concussion
- ▶ child

Second impact is a syndrome where an athlete sustains a brain injury (concussion or worse) and while symptomatic of the first injury, sustains a second brain injury, which is associated with catastrophic deterioration with diffuse cerebral swelling.^{1,2} It has been subsequently posited to either “not exist” or “has been overdiagnosed,” as reported cases are often lacking in clinical detail and the primary symptoms are suspicious for a recall bias in the face of the outcome of the secondary injury.^{3,4} Fortunately, if second impact syndrome exists, it is remarkably uncommon.⁵

Nearly 50 years ago, Dr John Fekete, then a regional pathologist, described, in the December 28, 1968 edition of the

Canadian Medical Association Journal, a 16-year-old youth with two such injuries with a fatal outcome, with the conclusion, “It is emphasized that a minor brain concussion with loss of consciousness may set the stage for subsequent lethal brain damage, even in the absence of skull fracture or epidural hematoma.”⁶

In a 1998 review of cases of putative second impact syndrome (SIS),⁴ the authors concluded that no case fulfilled all their criteria for SIS and that Dr Fekete's case was one of only five cases that fulfilled their criteria for “probable” SIS, as there was no evidence of a medical review after the witnessed first

received
September 23, 2016
accepted after revision
August 24, 2017

DOI <https://doi.org/10.1055/s-0037-1607223>.
ISSN 2368-0539.

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impact. The original article itself provided limited clinical information surrounding the nature of the initial injury, as it was reported primarily from a neuropathological perspective.

In the face of current medical literature, two questions persist about this report: first, was this a case of second impact syndrome? and second, if it was, should it not be correctly recognized as the inaugural report of this syndrome?

Methods

An audit of material in the public register of Dr Fekete's case was undertaken to resolve these issues. Information sources included the newspaper report of the death, transcripts from a coroner's inquest held 12 days later (including the autopsy report), genealogical data available online, and available internet resources.

Results

A coroner's inquest was held 12 days after the death on the basis that "there may have been more than one accident involved" (in his death).

A witness testified that she had seen the deceased skating backward quickly without a helmet at a public skate when he fell suddenly on the ice, striking his occiput, 4 days before he died. He did not move on the ice for 4 to 5 minutes and required two-person assistance to leave the ice surface. His mother reported that he said he "felt alright" but that he had a bump on his head when she came to see him in the rink manager's room. He stayed off the ice subsequently and requested to go home later that night. Once home, he reported a headache that had been unresponsive to three 325 mg acetylsalicylic acid (ASA) tablets and requested additional analgesic.

A classmate reported him telling her, "I've got a slight concussion, you know," and of an ongoing headache during the day of the second injury. She further reported him borrowing approximately 9 to 21 ASA tablets (325 mg) from her locker over the week. A teacher reported the deceased requesting a delay in a literature test on the basis of headache the day prior to the second injury and that "he would be going to the doctor on Thursday" (the day of the second injury). She further reported that he functioned normally in her class.

On the evening of the second impact (4 days after the initial impact with apparent ongoing concussive symptomatology), the scheduled opponent for the deceased's hockey team did not arrive, and a call went out to assemble a pickup team. During that game (with the deceased wearing a Cooper hockey helmet, likely the SK10 model), there was a collision with a player (much bigger than he), and the deceased fell to the ice, striking his left temple (reported as ~8:15 p.m.).

The initial responder (with whom he had collided) found him to be immediately unresponsive, with difficulty in breathing, his jaw tightly closed, and then he started vomiting repeatedly.

The first medical responder, a local GP arrived at approximately 8:30 p.m., found him "badly hurt" and unresponsive. He was transferred to the local hospital, where "his condition grew worse"—his pupils were later reported as fixed and dilated. A transfer was discussed with the regional neuro-

surgeon in St. John (1 hour 55 minutes away by Google maps⁷), and the deceased was ultimately transferred to the regional hospital in Fredericton (42 minutes away by Google maps⁷). The weather was clear that night with no snow and wind.⁸ He probably died during that transfer at approximately 10:00 pm.

If there was a genetic propensity to brain swelling following trauma, a genealogical review traced the deceased's lineage back four to five generations to primarily Irish and also Scottish immigrants to New Brunswick, Canada in the mid-1800s.

While the postmortem findings were extensively reported in Dr Fekete's original report, the summary of the autopsy is as follows: Normal development and well-nourished male, weight at 25th percentile and height at the 10th percentile. Recent hemorrhage in left temporalis muscle and left orbit. The brain was edematous, weighing 1,600 g (+5.3 SD⁹) with subarachnoid hemorrhage, both cerebral and spinal; a right occipital lobe contusion 1 × 2 cm; and hemorrhage within the brainstem—cerebral peduncles and pons. The larynx and trachea showed widespread petechial hemorrhage and the lungs showed edema and congestion with large hemorrhagic areas. A remote kyphoscoliosis surgical repair was present.

Discussion

McCrary and Berkovic proposed diagnostic criteria for second impact syndrome,³ consisting of:

1. Medical review after a witnessed first impact.
2. Documentation of ongoing symptoms following the first impact up to the time of the second impact.
3. Witnessed second head impact with a subsequent rapid cerebral deterioration.
4. Neuropathological or neuroimaging evidence of cerebral swelling without significant intracranial hematoma or other cause for edema.

They have previously concluded that the above-mentioned case was a "probable" case of SIS on the basis that there was no medical review following the first impact. From the coroner's inquest, we have indirect evidence that the deceased was aware that he had a concussion, that he was experiencing ongoing symptomatology, and that he consulted his doctor earlier in the day he died. Using the above diagnostic criteria, this description fulfills definite criteria for SIS.

Cerebral edema may follow a primary or a secondary head injury, the latter comprising second impact syndrome. Similar mechanisms may be involved. Within the concussion literature, the existence of second impact syndrome has been questioned,¹⁰ preferring the simplicity of diffuse cerebral edema following a primary head injury. These same articles reference case series of primarily children with dramatic cerebral edema developing acutely in the context of a head injury, most notably the series by Snoek¹¹ and Bruce.¹² Unfortunately, these case series either did not include adequate details as to the mechanism of injury to ensure a "minor" injury or clearly included children with severe head injury (by Glasgow coma scale). Within pediatrics, short height, witnessed falls have been

associated with death, and a subset of these have contusion with brain edema.^{13,14} Fortunately, these incidents are rare, estimated at less than 1 in a million children less than six per year¹⁵ and approximately 1 per 100,000 children brought to an emergency department following a playground equipment fall.¹⁶ It is known that mutations within the CACNA1A gene predispose individuals to familial hemiplegic migraine and delayed cerebral edema following minor head trauma;^{17,18} however, this gene abnormality is exceedingly rare within the population,¹⁹ and therefore unlikely to have accounted for these cases of diffuse cerebral edema.

Would the outcome of this case have been different if the physician who reviewed him earlier in the day was practicing using current guidelines? I suspect that the answer is "likely." Practicing consistent with the consensus statement from the fourth International Conference on Concussion in Sport²⁰ would have seen the physician recommend physical and cognitive rest until the acute symptoms resolved and then a graded program of exertion prior to medical clearance and return to play.

Second impact syndrome was named by Saunders and Harbaugh in the *Journal of the American Medical Association* in 1984.²¹ Reviews of this condition^{1,2} erroneously attributed its first description to Schneider in 1973,²² 5 years after Dr Fekete's report. As one of many Canadian physicians who manage individuals with concussive symptomatology, I believe that it is time that Dr Fekete's foundational work is recognized.

Financial Disclosure

The author has no financial relationships relevant to this article to disclose.

Conflict of Interest

The author has no conflict of interest to disclose.

Acknowledgment

The author would like to thank M.E. Gordon, DipPOT, for providing the genealogy of the deceased.

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