EKG Changes After Spinal Subarachnoid Hemorrhage

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Abstract:
Marked morphological electrocardiographical changes occurred in a patient whose subarachnoid hemorrhage originated in the lower spinal canal. Thus, the electrocardiogram can show the changes well known for intracranial subarachnoid hemorrhage even when the source of bleeding is in the lower spinal canal.

Additional Key Words: electrocardiogram, subarachnoid bleeding

It is well known that intracranial subarachnoid hemorrhage may be associated with a variety of electrocardiographical morphological changes.1-10 Specifically, those changes most frequently mentioned are: large T waves of either normal or abnormal polarity, S-T segment elevation or depression, Q-T interval changes, and abnormal U waves. The mechanisms underlying these EKG changes still need to be clarified. Review articles on spinal subarachnoid hemorrhage in general, or those dealing with spinal cord tumors with hemorrhage or spinal vascular malformations in particular, do not comment on electrocardiographical abnormalities.11-16 We wish to report a case of spinal subarachnoid hemorrhage associated with EKG morphological changes.

Case Report
The present illness of this 22-year-old Mexican-American female dates back to infancy. She was first admitted to the Denver General Hospital at three and one-half months of age in January, 1950. Up to that time she had developed normally. In particular, movements of the extremities were intact. She was admitted because of colicky-type pain, fever and constipation. Shortly thereafter she went into urinary retention, became paraplegic and areflexic in her lower extremities, and had a T-8 sensory level. A myelogram demonstrated an LI-2 level block and she underwent an LI-2 laminectomy. Operative findings included an encapsulated hemorrhagic intramedullary tumor mass with proximal extension. Microscopic examination was nondiagnostic, reported as "hemorrhagic necrotic material."

The patient was discharged from the hospital with a flaccid paraplegia and a nonfunctioning bladder. During childhood she became able to ambulate with her legs in braces using a swing-through gait. An ileal loop urinary diversion procedure for urinary difficulty was performed in her early teens; bowel emptying was controlled daily by digital removal.

At age 22 she could walk using Canadian crutches and a swing-through gait. She supported herself mainly on her right leg and even could do so unbraced. However, she still used long leg braces. She had no movement below her knees but some poor to fair movement of her hip group muscles. She was wasted, flaccid and anesthetic below L-2.

Her second Denver General Hospital admission occurred on June 4, 1972, at age 22. She had the acute onset of severe low back pain followed by a constricting feeling around her lower rib cage as if a binder were being tightened. She then felt a "rush" of heat and developed an intense head and neck ache. All this occurred while she was seated in a wheelchair doing wheelchair square dancing. The entire episode evolved over a matter of a few minutes. The patient was immediately taken to a local clinic on the premises. Her BP was 154/80 with an "irregular pulse." She was given nasal oxygen and 10 mg of morphine sulfate intramuscularly. An EKG taken within 30 minutes of the onset of the episode revealed significant diffuse ST segment depression especially in the inferior leads and across the precordium (Fig. 1).

On arrival at the Denver General Hospital, she was afebrile, and had a flaccid paraplegia with a sensory level of anesthesia below T-10 and decreased pin prick sensation to T-8. There was marked nuchal rigidity. Examination of the back revealed the old laminectomy scar, and a lower thoracic rotatory scoliosis. An ileostomy stoma was present on the right side of her abdominal wall and rectal tone was nil. Cardiovascular examination was normal. Cranial nerves, mentation, and upper extremity functions were all intact. Initial laboratory work revealed a hematocrit of 38, white blood cells of 19,000 with 78% polymorphonuclear, 20% lymphocytes, arterial pH of 7.48, PO2 of 27 mm Hg, and PO2 of 75 mm Hg. Electrolytes were normal.

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FIGURE 1

Initial electrocardiogram one-half hour after onset of spinal subarachnoid hemorrhage (V₆ not taken). There is diffuse ST-T depression.

except for a potassium of 3.1 mEq/liter, which returned to normal within 36 hours. EKG on admission, within five hours postonset, still showed ST segment depression in the inferior leads but no longer in the precordial leads.

Thoracic-lumbar spine films (fig. 2) showed a calcified mass inside the spinal canal at the T-12 vertebrae level posteriorly. CSF at the time of myelography was bloody with an xanthochromic supernatant, having more than 100,000 red blood cells and 6,200 white blood cells with a differential of 75% polymorphonuclear, 25% lymphocytes, a protein of 4,200 mg% and a glucose of 8 mg%. Using only ½ cc of dye, a limited myelogram revealed a complete block at the upper margin of L-2. Laminectomy, T-11 through L-2, revealed a large calcified intradural hematoma in which were enmeshed roots of the cauda equina. In order to control brisk hemorrhage, the dural sac had to be ligated. The final histological report on the intradural calcified mass revealed only "recent and old blood clot with organization, fibrosis and ossification."

The patient had a benign postoperative course and remained with a flaccid paraplegia and a T-8 sensory level at the time of her discharge to a rehabilitation center. Her in-hospital BP ranged from 80/60 to 130/80, with 110/70 being the most frequent reading. Follow-up electrocardiograms during her hospitalization revealed a variety of changes consisting of prolonged QT interval, large broad T waves, ST elevations, and U waves (fig. 3). By discharge 15 days postspinal subarachnoid hemorrhage, her electrocardiogram still showed slight ST segment elevation in the inferior and precordial leads with decreasingly prominent T waves.

Discussion

The etiology of the morphological electrocardiographical changes that may be seen in intracranial subarachnoid bleeding are debated. Altered autonomic nervous system tone secondary to the hemorrhage, direct cortical and/or subcortical stimulation-irritation, or electrolyte-metabolic abnormalities have all been discussed. Most authorities appear to favor the autonomic dysfunction theory yielding cardiac changes reflecting themselves electrically. Recently it has been shown that experimental trauma to the cervical spinal cord also may yield electrocardiographical changes thought secondary to autonomic imbalance.

Our patient had the rapid onset (in about one-half hour) of EKG morphological changes following her spinal subarachnoid hemorrhage from a source in the region of the cauda equina. The sequence of low back pain followed by feelings of a heat "rush" and meningeal irritation, with systolic hypertension, suggests that the spinal subarachnoid hemorrhage was followed by some autonomic dysautonomia. This autonomic component may help explain etiologically the marked electrocardiographical morphological abnormalities which appeared acutely and gradually diminished. It appears unlikely that either her slightly low serum potassium in the first two hospital days or the minor blood gas abnormalities on admission played a significant role in the EKG
changes. In any event, the EKG abnormalities described herein are identical with those seen after intracranial subarachnoid hemorrhage, which suggests that they may share the same etiological mechanisms.

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**FIGURE 3**

Representative leads of electrocardiograms obtained during hospitalization. One notes large upright T waves, ST segment elevation, QT interval changes and prominent U waves.


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