Surgical Management of Atlanto-axial Instability (AAI)

Eassy

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By

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Introduction

Atlanto-axial instability (AAI) is characterized by excessive movement at the junction between atlas C1 and axis C2 due to either a bony or ligamentous abnormality. The causes of AAI are varied. Sometimes results from trauma other cases occur secondary to an upper respiratory infection or infection following head and neck surgery. Another cause is rheumatoid arthritis (RA), with its predilection for the upper cervical spine. In addition, congenital anomalies, syndromes, or metabolic diseases can increase risk of AAI ***(Banit et al., 2005)***.

In traumatic situation, approximately 10% of cervical fractures involve some injuries to the AA level. Of injuries to the AA complex, only 16% produce neurologic deficit secondary to the wide spinal canal at this level ***(Banit, 2005)***.

Patients may present with symptoms ranging from mild local neck pain to myelopathy and vascular compromise. The variability of signs and symptoms may take diagnosis difficult and it is often discovered incidentally ***(Henderson and Henderson, 2006)***.

Various techniques of AAI fixation have been described in the literature. Since Gallie reported arthrodesis by using a posterior wiring and autogenous graft technique in 1939, various authors have reported modifications of Gallie produce in which

wires or interlaminar clamps are used. Magerl and Seeman introduced the technique for C12 transarticular screw fixation in 1979. The C1-2 transarticular screw procedure is technically demanding and requires precise intraoperative and radiological knowledge of vertebral anatomy to minimize risk of causing introgenic injury ***(Stokes and Bray, 2002)***.

Aim of the Work

The aim of this work is to revise the srugical anatomy and biomechanics of atlanto-axial complex, pathophusiology, etiology, clinical presentation and to stuudy and evaluate the different modalities of management of atlanto-axial instability with special focus on surgical management.

Embryology Of The Upper Cervical Region

Human spine development begins when the primitive streak forms at the day 15 of embryonic life. Before the primitive streak develops, the embryo is bilaminar disc consisting of the epiblast and hypoblast. The primitive streak consists of a thickened linear band of epiblast that appears caudally in dorsal aspect of the embryonic disc. It elongates by adding cells at the caudal end as the rostral end thickens to form the primitive knot. Concurrently, a depression (primitive groove) forms in the primitive streak that is continuous with a depression in the primitive knot. The rostrocaudal axis of the embryo is defined when the primitive streak develops ***(Shaffery et al,2005)***.

The occipital bone and the preotic region of the skull are of somitic origin ***(Shaffrey et al, 2005).*** The occipital bone is formed by the fusion of four vertebral sclerotome structures. This corresponds to the segmental nerves that group together to form the hypoglossal nerve. The caudal-most portion of the forth occipital sclerotome is termed the proatlas. The proatlas divides into a ventral-rostral segment and a corresponding dorsal-caudal portion. The dorsal-caudal portion forms the portion of the

posterior atlas arch as well as the lateral masses. The ventral component forms the anterior U-shaped margin of the foramen magnum as well as the occipital condyles, and a portion detaches to incorporate with the pleurocentrum of the atlas vertebrae ***(Davis et al, 1977)***. The core of the proatlas centrum is metamorphosed into the apical ligament of the dens. The alar and check ligaments, as well as the transverse ligament of the atlas, are derived from the proatlas as unossified tissue ***(VanGilder et al,1990).*** Ossification of the occipital bone proceeds as follows: the two occipital squamouos portions ossify from a single center, the clivus ossifies from a single center, the paired jugular tubercles ossify from individual centers, and the occipital condyles ossify from individual centers ***(Shaffrey et al, 2005).***

The first and second cervical vertebrae develop from the fourth occipital somite through the second cervical somite. Parts of the axis develop from the fourth occipital somite; the remaining parts of the axis develop from the first cervical somite. The first cervical somite develops into the ventral arch of the atlas. The dorsal arch of the atlas most likely arises from the proatlantal sclerotome. There is a contribution from the first cervical sclerotome to the midportion of the dens ***(Shaffrey et al, 2005).***

The major portion of the atlas vertebrae is formed from the first spinal sclerotome. This transitional vertebra is modified from the remaining spinal vertebrae in that the centrum of the sclerotome is separated to fuse with the axis body forming the odontoid process. The anterior arch of the atlas, formed from the hypocentrum of the first spinal sclerotome, is overly developed and forms the anterior ring of the atlas that encloses the odontoid process. At an early embryonic stage, a hypochordal bow is found in front of each vertebral segment and subsequently disappears, except for the part that forms the anterior arch of the atlas ***(Shapiro et al, 1976).*** The hypochordal bow of the proatlas itself may survive and join with the anterior arch of the atlas to form a variant structure. Thus, an abnormal articulation may appear to exist between the clivus, the anterior arch of the atlas, and the apical ligament of the odontoid process ***(VonTurklus et al, 1972).***

The axis, exclusive of the odontoid process, is derived from the second spinal sclerotome. The body of the axis originates from the first spinal sclerotome where the terminal portion of the odontoid arises from the proatlas (fig. 1). At birth, the odontoid is separated from the body of the axis by a cartilaginous band, which represents the vestigial disc referred to as neural central synchondrosis. It lies below the level of the superior articular facet

of the axis and does not represent the anatomical base of the dens. The neural central synchondrosis is present in nearly all children at age 3 years and is absent in most by age 6 years ***(Fielding et al, 1980)***. At birth, there should be recognizable odontoid process that has not yet fused to the base of the axis ***(Gehweiler et al, 1980).***

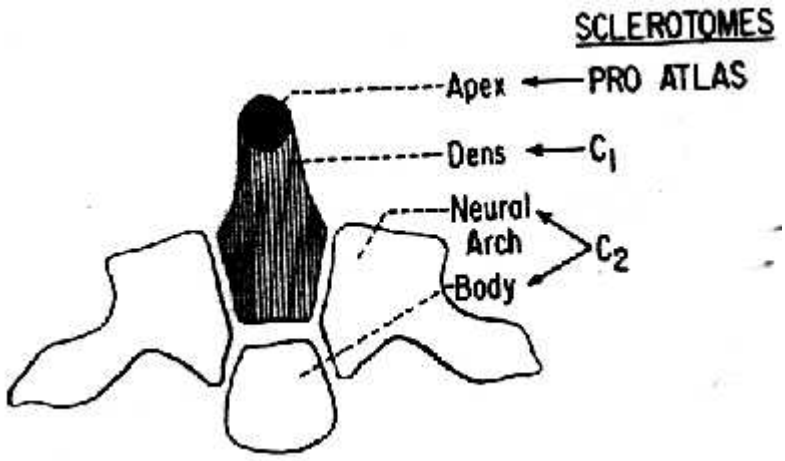

Fig. 1. Development of the axis***.(VanGilder et al, 1990)***

The tip of the odontoid process is not ossified and represents a separate ossification center, or ossiculum terminal, which usually appears at age 3 years and fuses with the body of the dens by age 12 years ***(Gaufin et al, 1975).*** It may occasionally fail to fuse with the odontoid process and is then called ossiculum terminal persistans. It has very little clinical significance ***(VanGilder et al, 1990).***