REVIEW

Vascular Foramina of C2 and its Odontoid Process: Application to a Better Understanding of Type II Odontoid Fractures

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ABSTRACT

The literature suggests that surgical treatment of type 2 fractures of the dens should be favored because poor vascular flow to its neck leads to low healing ability. The aim of this study was to investigate the presence of vascular foramina in the odontoid process to elucidate this potential relationship. Forty dry specimens of the C2 vertebra (35 adults and five children) were studied using 10x magnification. No specimen showed evidence of prior trauma or congenital malformation. The locations and concentrations of vascular foramina were identified. Vascular foramina are concentrated posteriorly and anteriorly at the base and neck of the odontoid process. However, most have 1-3 large foramina between the medialmost aspect of the superior articular facet and the base of the odontoid process, i.e. located along the lateral edge of the odontoid process at its base. These vascular foramina ranged in size from 0.25 to 2.2 mm (mean 1.1 mm). Anteriorly, they were primarily concentrated off the midline and inferior to a line connecting the superiormost aspects of the left and right superior articular facets. On the posterior aspect of the odontoid process, they were concentrated inferior to the lowest edge of the groove for the transverse ligament. There were no grossly identifiable foramina on the anterior facet of the dens or at the point of contact of the transverse ligament to this bony part in any specimen. No difference was identified between adult and child specimens. There is a concentration gradient of vascular foramina on the odontoid process, greatest at its base and decreasing toward its apex. The anterior facet and region of contact with the transverse ligament are devoid of grossly visible vascular foramina. Quantitative studies are needed to determine whether a lower quantity of cancellous bone in relation to a high gradient of vascular foramina decreases the surface area for bony fusion. Spine Scholar 1:124

INTRODUCTION

The odontoid process develops from two primary ossification centers and a secondary one. Synchondroses in the body, neural arches and the dens fuse at approximately three to six years of age. The most superior synchondrosis at the uppermost segment of the dens fuses by twelve years of age. The odontoid process projects superiorly from the body of the axis to articulate with the posterior segment of the anterior arch of the atlas (Akobo et al., 2015).

Reasons for nonunion of type 2 odontoid fractures have been debated (Schatszker et al., 1971; Schatzker et al., 1975; Althoff et al., 1977) and include vascular interruption to the odontoid, which could result from a limited vascular supply to the fracture site (Schatszker et al., 1971; Schatzker et al., 1975). This was attributed either to there being fewer vascular foramina between the body of the axis and neck of the dens, or injury to vessels reaching the odontoid process through accessory ligaments that are avulsed during trauma (Schatszker et al., 1971; Schatzker et al., 1975; Althoff et al., 1977). The passage of vessels to the dens as they course with the alar ligament has not been demonstrated in the apical ligament.

Blood supply

Most of the blood supply to the odontoid process arises from the posterior ascending branch of the vertebral artery; there is a less significant contribution from an anterior branch. The anterior and posterior ascending branches

merge and form a vessel arcade (Schiff et al., 1973; Kilmo et al., 2011; Cramer et al., 2013). The internal carotid artery (ICA) provides the blood supply to the anterior portion of the dens through superior horizontal arteries that rise from the ICA prior to entering the carotid canal. These arteries pierce the anterior atlanto-occipital membrane and cross to the supra-odontoid arterial confluence. A branch of the ascending pharyngeal artery rises from the external carotid artery (ECA) and joins the transverse anterior confluence formed by the anastomosing anterior and posterior ascending arteries as it passes through the hypoglossal canal (Althoff et al., 1977). Thus, the dens is perfused by a vast confluence of vessels.

Our aim was to quantify the vascular foramina and their relationship to other areas of the dens. In particular: the tip of the dens representing a type I odontoid fracture; the lower (subdental) subdental synchondrosis denoting a type II odontoid fracture; and through the body of C2 as would occur during a type III odontoid fracture.

Materials and Methods

Forty dry specimens of the C2 vertebra (35 adults and five children) were examined by 65x magnification (Carson zOrb digital USB microscope, Ronkonkoma, NY). No specimen showed evidence of prior trauma or congenital malformation. The locations and concentrations of vascular foramina were identified and the foramina were documented and photographed, especially around the dens.

Results

Vascular foramina are concentrated posteriorly and anteriorly at the base and neck of the odontoid process. However, most were found to have 1-3 large foramina between the medial most aspect of the superior articular facet and the base of the odontoid process, i.e., located along the lateral edge of the odontoid process at its base (Figs. 1-4).

Figure 1: Anterior view of C2

Figure 2: Zoomed in view of Figure 1 noting the many vascular foramina

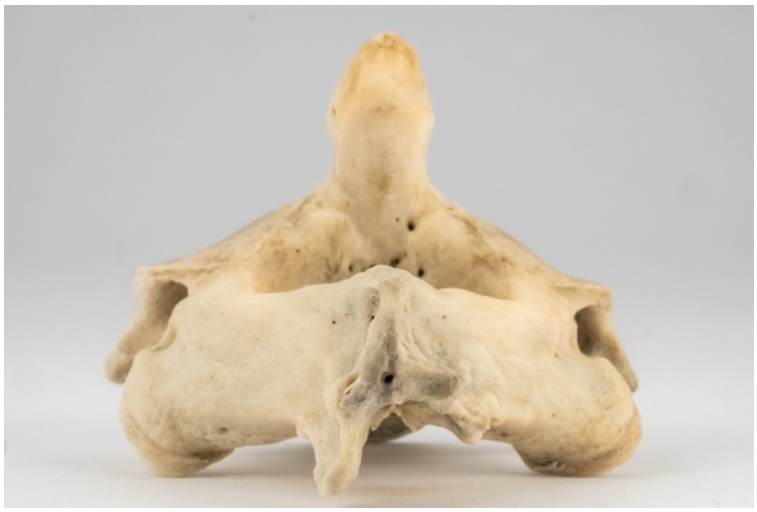

Figure 3: Posterior view of C2 noting vascular foramina entering the odontoid at its neck on the right and inferior to this nearer the level of the synchondrosis.

Figure 4: Lateral view of C2 noting vascular foramina toward apex of the odontoid process

Some specimens were also found to have small foramina near the apex of the odontoid process (Fig 5). These vascular foramina ranged in size from 0.25 to 2.2 mm (mean 1.1 mm). Anteriorly, they were primarily concentrated off the midline and inferior to a line connecting the superior most aspects of the left and right superior articular facets. On the posterior aspect of the odontoid process, they were concentrated inferior to the lowest edge of the groove for the transverse ligament. No grossly identifiable foramina were identified on the anterior facet of the dens or at the point of contact of the transverse ligament to this bony part in any specimen. In the pediatric specimens, no variability of the primary or secondary synchondroses was noted. No difference was identified between the adult and child specimens. In general, the larger foramina were located at the neck of the dens.

Figure 5: Posterior apex of odointoid process demonstrating small vascular foramina when magnified.

DISCUSSION

Our results revealed no significant difference in the number of vascular foramina in the three sites studied characterizing type I, II and III odontoid fractures. They were concentrated anteriorly and posteriorly at the base and neck of the dens, with 1-3 large ones between the medial most aspect of the superior articular facet and the base of the dens, i.e. along the lateral edge of the base of the odontoid process. The vascular foramina ranged in size from 0.25 to 2.2 mm (mean 1.1 mm). Anteriorly, they were primarily concentrated off the midline and inferior to a line connecting the superior most aspects of the left and right superior articular facets. On the posterior aspect of the odontoid process, they were concentrated inferior to the lowest edge of the groove for the transverse ligament. No grossly identifiable foramina were identified on the anterior facet of the dens or at the point of contact of the transverse ligament to this bony part in any specimen. There was no lack of vascular foramina along the site of the subdental synchondrosis that could suggest a deficient vascular supply. However, in view of the location and larger size of the vascular foramina in relation to the rest of the axis, the lower amount of cancellous bone surface area in this region could be a factor in nonunion of type 2 odontoid fractures.

Type 2 Anderson and D'Alonzo odontoid fractures are classified as unstable fractures occurring at the base of the neck of the dens (Anderson et al., 1974). They are the most common cervical spine fractures in patients over 65 years old, with a non-union rate of up to 85% without surgical intervention (Ryan et al., 1992; Sasso et al., 2001). The most common clinical manifestations include neck pain, weakness of the extremities, upper limb wasting, neck tilt, neck movement restriction, and transient loss of consciousness (Kirankumar et al., 2005). High energy trauma is typical in younger patients while geriatric patients develop type 2 dens fractures because of falls from standing height (Müller et al., 1999). In octogenarians, this injury is associated with high morbidity, one study calculating it at 41% at one year irrespective of intervention (Graffeo et al., 2016).

In a retrospective cohort study of 223 patients with type 2 dens fractures, the mortality rates among acutely hospitalized octogenarians was 12.5% in the surgical group and 15% in the non-surgical cohort (Smith et al., 2008). In patients younger than 45-55 years and for anteriorly displaced fractures, conservative management in the form of a rigid cervical collar or Halo immobilization is as effective as surgery (Nourbakhsh et al., 2009). Mortality and complication rates are lower in patients with less than 50% odontoid displacement who are treated with a cervical collar and early immobilization (Molinari et al., 2013). Type 2 dens fractures have a neurological deficit incidence of 9.6-13%. The incidence is similar between patients under and over 70 years of age. Most neurological symptoms are related to posterior dislocation of the dens (Müller et al., 1999; Patel et al., 2012). Patients with neurological deficits as a result of a type 2 dens fractures are 4.72 times more likely to die than patients who are neurologically intact (Patel et al., 2012).

In a study of 133 patients with type 2 dens fractures presenting to a level 1 trauma center, 30% had fractures with chronic features on imaging. Among these patients, 17% presented with acute or chronic injuries (Kepler et al., 2014). Most patients do not present acutely with neurological symptoms (Omeis et al., 2009).

Older age, anterior displacement of more than 4mm, posterior displacement, angulation, and late presentation are known contributors towards non-union (Govender et al., 2000). Initial conservative treatment has a 2.92 times higher risk of failure than early surgical treatment. Male sex has also been associated with treatment failure (Fehlings et al., 2013).

Operative treatment has been found cost effective in patients aged 65 to 84 when a measure of quality-adjusted life years (QALY) of $100,000/QALY is used as a benchmark, but more costly and less effective in patients aged over 84 years (Barlow et al., 2016). Chapman et al. found that among 322 patients over 65 with type 2 dens fractures, those treated

operatively had a 30-day survival advantage. Non-operative patients showed a trend toward higher mortality at maximal follow-up.

A systematic review of type 2 and 3 dens fractures revealed that the most common complication in the operative group was related to instrumentation placement, whereas in the conservative treatment group the most common complication was device-related ulceration (Huybregts et al., 2013).

A biomechanical study used cadaveric heads with cervical spines to show that specimens that underwent an impact and subsequent deceleration of the upper forehead while the torso had forward movement were more likely to sustain type 2 or 3 odontoid fractures (Ivancic, 2014). The dens can direct the kinetic energy to the upper cervical spine as the orientation of the force vector creates the fracture line (Benzel et al., 1994). This high-tension location and the poorer density of cancellous bone have been proposed as explanations for the high non-union rate of type 2 dens fractures (Heggeness et al., 1993).

In a study of 183 patients who underwent conservative management of type 2 dens fractures, union was achieved in 54%. Eighteen patients were subjected to selective vertebral angiography, 10 with acute fractures and eight with nonunion (Govender et al., 2000). The primary perfusion vessel, the posterior ascending branch from the vertebral artery, was demonstrated in all cases. Studies have revealed no histological evidence of avascular necrosis in resected non-union dens fractures (Schatzker et al. 1975; Crockard et al., 1993).

CONCLUSION

There are multiple sources of blood to the odontoid process in addition to the anterior and posterior vertebral artery branches, including external carotid artery branches, vascular arcades, and branches traveling with the accessory ligament. The odontoid process does not lose its blood supply in the setting of a type 2 dens fracture. Explanations for non-healing type 2 dens fractures include a higher ratio of cortical to healing cancellous bone in the neck of the dens. Those who do not support a vascular supply explanation for non-union consider the bone density at the neck of the odontoid process to be significantly lower, leading to a higher incidence of fractures. There is a significantly greater surface area than in type 3 fractures through the body of the axis, so there are more healing trabeculae for fusion (Amling et al., 1995). However, significantly larger vascular foramina were identified in the lateral aspect of the neck of the dens bilaterally, which could provide a fulcrum (or an area of higher likelihood of fracture) as vascular foramina decrease the concentration of healing cancellous bone. Further studies are needed to elucidate the relationship of decreased cancellous bone to vascular foramina and its predisposition to fracture.

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