



Life Threatening Complication Arterial Bleeding During Atlas-Axis Fusion Surgery Stopped with Wound Clot

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Background

The C1-C2 motion segment is a unique and complex articulation which contributes half of the rotational motion in the cervical spine. Although congenital conditions, such as Down's syndrome or osteogenesis imperfecta, can cause atlantoaxial instability, the main etiologies in adults are acute trauma, or inflammatory pannus of rheumatoid arthritis. Infection can also be implicated, due to the rich arterial supply and venous plexus in the region providing a pathway for infectious agents [1]. The instability is characterized by excessive motion at the C1-C2 junction, due to an osseous or ligamentous abnormality. It can lead to abnormal biomechanics with neurological consequences if the spinal cord or adjacent nerve roots are compromised [1]. A common and accepted treatment for traumatic, post-traumatic, inflammatory and congenital atlas-axis instability is posterior fusion of these vertebrae. Early attempts at posterior spinal fixation of this area involved interspinous/interlaminar wiring [1]; although still in use, primary stability following sublaminar wiring and bone grafting is often poor, requiring lengthy postoperative immobilization, and demonstrates a significant degree of non-union [2]. This has evolved into pedicle screw/translaminar fusions. Two main methods of posterior screw fixation fusion, Harms's technique, or Magerl's technique, are important advancements in upper cervical fusion surgery. Both have demonstrated excellent fusion rates, nearly 100%, even without postsurgical immobilization. Magerl's technique consists of an "in situ" C1-C2 transarticular screw fixation with posterior wiring to hold the bone graft. In Harms's technique, polyaxial screws are independently inserted into the C1 articular masses and the C2 pedicles and connected by a small diameter rod [2]. Although results are typically excellent, the success of the surgery relies on proper indications and surgical technique [1].

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Case Report

A 68 year-old male patient, in the Department of Orthopedics, Barzilai Hospital, Ashkelon, Israel, who presented with C1-C2 instability, was undergoing Harms's posterior fixation surgical arthrodesis of the atlas-axis complex. During the insertion of a fixation screw into the atlas, the screw penetrated the right vertebral artery, resulting in immediate profuse arterial bleeding which would not stop by the screw insertion because of bone splitting. Several methods were attempted to control the hemorrhage but at no avail. The lack of direct access to the bleeding source and the pressure of the blood flow, caused Bone Wax to break down and to float out. Furthermore, any attempts to press directly onto the bleeding source failed due to the location under the atlas. A vascular surgeon was urgently called to the OR as the patient had already lost 1 liter of blood. Recommended Wound Clot Hemostat (WCH) as an intermediate emergency solution while he was scrubbing in WCH, manufactured by Core Scientific Creations, is a novel hemostat for bleeding control based on non-oxidized bio-absorbable cellulose. Due to its unique non-oxidation technology, the product can control mild to severe hemorrhage without applying compression onto the bleeding site. WCH may be applied to multiple tissue types, including blood vessels, internal organs, bones and complex wounds. It is bio-restorable and absorbed in the body within c.a. 7 days.

A 5 × 5 cm Wound Clot surgical gauze was placed directly onto the tear in the vertebral artery without any compression application. WCH was immediately able to reach the bleeding source using the blood saturated gel as an anchor to adhere to the source of hemorrhage. Bleeding from the artery instantly decreased, reaching complete stasis in less than 2 minutes (before the vascular surgeon was sterile and ready to operate). As of which, the vascular surgeon decided the hemostasis was stable and did not require any further surgical intervention. He instructed the surgeons to place two more 5 × 5 cm pieces of WCH and continue with an alternate fixation method.

The head surgeon stated, "We are sure that this product managed to save the patient's life and avoided an unnecessary extension of the surgery by very difficult exploration of the vertebral artery or the use of endo-vascular technique for embolization of the bleeding artery." During the follow-up examination, no re-bleeding could be detected. The patient felt very well and was walking the next day, with no apparent negative sequelae from the surgery.

Discussion

Wound Clot Surgical was placed on the bleeding source to treat un-controlled high pressure severe arterial hemorrhage. Although blood continuously flowed, once WCH was placed onto the bleeding site it began to absorb blood and transform into a gel state. WCH self-adhered to the artery, as it rapidly absorbed blood, utilizing muco-adhesion principles to counter forces exerted from the blood flow, without the need to apply pressure onto the bleeding vessel. Wound Clot's absorption capability, over 2500% its own weight, is a result of its unique capacity to form a stable gel when exposed to a large volume of blood. The elevated absorption, along with its maintenance of a stable membrane, aids in coagulation by pooling coagulants in significant quantities in the vicinity of the injured site and activating

them. Patented structured groups on the molecular level stimulates the intrinsic clotting mechanism and significantly multiplies the quantity and interaction of the different factors, enhancing the coagulation process and reducing the coagulation time by more than 50%. Therefore, the entire clotting cascade activity is enhanced and optimized to result in a stable biological clot and achieve natural hemostasis based on the blood's coagulation capabilities.

In conclusion, during this life-threatening complication in C1-C2 fixation surgery, Wound Clot Hemostat managed to stop high-pressure high-volume hemorrhage from the vertebral artery within less than 2 minutes without applying pressure onto the bleeding vessel.

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