Glucocorticoids in the treatment of chronic subdural hematoma

Glucocorticoides en el tratamiento del hematoma subdural crónico

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Resumen

Se revisan cincuenta y tres pacientes adultos consecutivos tratados por hematoma subdural crónico con el agregado de glucocorticoides en su plan de tratamiento. El concepto actual de la patogenia del hematoma subdural crónico y el mecanismo por el cual los glucocorticoides podrían mejorar el pronóstico de esta condición son discutidos. Se presentan los resultados clínicos de este régimen terapéutico y si se comparan con los resultados de otras series en las cuales el hematoma subdural crónico fue tratado sin el agregado de glucocorticoides. Da la impresión que los glucocorticoides merecen un lugar en el tratamiento del hematoma subdural crónico.

Palabras clave: Hematoma subdural crónico, Glucocorticoides.

Abstract

Fifty-three consecutive adult patients treated for chronic subdural hematoma with the addition of glucocorticoids in their management are reviewed. The current understanding of the pathogenesis of chronic subdural hematoma and the mechanism by which glucocorticoids might improve its prognosis is discussed. Clinical results of this therapeutic regimen are presented and if compared with other series where chronic subdural hematomas were treated without the addition of glucocorticoids, it would appear that glucocorticoids deserve a place in the management of chronic subdural hematoma.

Key words: Chronic subdural hematoma, Corticosteroids.

Introduction

While chronic subdural hematoma (CSH) is common in neurosurgical practice, it's optimal surgical treatment is still a matter of debate[1,13,16,20] with options ranging from craniotomy and membranectomy to craniostomy via one or two burr holes (with or without subdural drainage) to minicraniostomy by twist-drill.

Suggestions for medical options for the treatment of chronic subdural hematoma, are less frequently found in the literature. Glucocorticoids have been suggested as the sole treatment[2,3,15] or following burr hole evacuation[5,21] with apparently good results[2,3,5,15,21] .

Currently most cases of CSH are being treated worldwide by burr hole craniostomy with or without drainage but, regardless of the surgical technique used, a significant rate of recurrence persists[7] and, by and large, most neurosurgical centers still do not include the use of glucocorticoids in the treatment of CSH.

Materials and Method

During my neurosurgical practice, up to the year 2009, I collected 53 cases of CSH between 1982 and 1989[5] .The diagnosis was established by computerized tomography (CT) in the vast majority of these cases while in a few it was done by MRI. Small CSH without mass effect or symptoms or signs of cerebral dysfunction were treated only with Dexamethasone (Decadron^{R}, Merck,Sharp and Dome, West Point, PA 19486, USA)

and followed clinically until a CT scan confirmed their complete resolution.
More significant CSHs were operated under general anesthesia, although in a few cases this was done under sedation and local anesthesia. Surgery included two burr holes overlying the subdural collection with evacuation of the hematoma by repeated irrigation of the subdural space with physiological solution. Free communication between the two burr holes through the subdural space was confirmed and search for other subdural loculations overlying the cerebral cortex was carefully done. If present, these were opened by sharp dissection in as wide a fashion as allowed by the limited exposure provided by the burr holes. A round No.10 Jackson-Pratt drain (American Heyer-Schulte Co. Goleta, CA 93017, USA) was then introduced into the subdural space through the most convenient of the two burr holes and exteriorized through a separate stab wound. Once the operative wounds were closed, the subdural drain was connected to a closed drainage system. This subdural drainage was maintained for two to a maximum of six days postoperatively depending on the amount of subdural drainage obtained.
With the exception of one patient that was lost to follow-up, all others were followed clinically and by CT scan or by magnetic resonance imaging (MRI) until complete resolution of the CSH was demonstrated.
If while in the hospital or after discharge a patient showed too slow an improvement in his/her pre-operative condition or reappearance of symptoms after an initial improvement, a CT scan was obtained. If recollection of the CSH was seen, a short course of Dexamethasone was instituted (2 to 4 mg. PO four times a day for a period of 7 to 14 days) it´s actual duration being determined by the clinical condition of the patient. Only if this regimen failed to improve the situation was reoperation considered.
When CT scan demonstrated complete resolution of the subdural collection the CSH was thought to be cured and the follow-up discontinued. All patients with unresolved CSH at the conclusion of the study were contacted to assess their clinical condition.

Results

Fifty-three consecutive patients were included in this study. There were 29 males and 24 females whose ages ranged from 16 to 97 years. Three patients (6%) had bilateral CSH which were treated simultaneously. 25 patients (47%) had minimal symptoms and were neurologically normal (Grade I). 24 patients (45%) showed mild symptoms and mild or no neurological deficits (Grade II). Two cases (4%) were drowsy or disoriented with variable neurological deficits (Grade III) and the last two cases (4%) were comatose with decerebrate or decorticate posturing (Grade IV).
Of these fifty-three cases, 27 (51%) reported a known head trauma as the cause of their CSH. Five patients (9%) were on anticoagulation treatment and one (2%) was found to have dural metastasis as the etiological factors responsible for their CSH. In 20 cases (38%) the cause was unknown.
During their postoperative period, ten of the 53 patients (19%) showed persistent or recurrent symptoms and a persistent significant subdural collection on CT scan, presenting a clear indication for reoperation. However, nine of these patients were treated instead with short courses of Dexamethasone. Eight of them became asymptomatic and in five of them, a CT scan revealed complete resolution of their CSH while in the other three, CT scans done 25, 82 and 137 days after their surgeries still showed residual subdural collections although these three patients remained asymptomatic 38, 50 and 64 months later, without reoperation. Only the ninth of these patients continued to worsen clinically despite Dexamethasone treatment and required repeated surgery, after which his CSH resolved completely, establishing a reoperation rate of 2% for this group of patients.
The only one in this group of ten patients that was not treated with Dexamethasone, developed sepsis from infected wounds sustained in the original trauma. He went on to develop a subdural empyema requiring craniotomy with partial recovery. Unfortunately he eventually died of pulmonary embolism 175 days after the surgery.
Of the 53 patients treated 41 (77%) had complete resolution of their CSH while in 11 (21%) their follow-up CT scans showed varying degrees of residual subdural collections. In spite of it, seven of these were asymptomatic. Another one remained with a mild dementia that preexisted his CSH. The remaining three patients died, one from pulmonary embolism, the second from pneumonia superimposed on her Alzheimer´s disease and the third from a brain-stem stroke while off anticoagulation. The last of the fifty three cases patient was lost to follow-up.
Post-operative complications occurred in eight cases. Three are the mortalities just mentioned. The fourth case developed an ipsilateral acute subdural hematoma following the burr-hole evacuation of her CSH. Immediate craniotomy revealed two small cortical arterial branches as the source of this complication. She went on to a complete recovery and follow-up CT scan revealed complete resolution of the hematoma. In two cases surgery was complicated by the development of small intraparenchymatous bleeds in the cerebral hemisphere ipsilateral to the CSH. Both resolved gradually without the need for additional surgery. Another patient developed a superficial infection at one of the burr hole sites which cleared after scalp debridement and antibiotic therapy. Finally the last complication relates to the only patient that developed a recollection of his CSH that required reoperation (2%).
At their last follow-up evaluation 39 (73%) patients were asymptomatic and neurologically intact (Grade I). Ten (19%) remained with mild symptoms and/ minimal neurological deficits (Grade II). Three (6%) were the known mortalities and 1 (2%) was lost to follow-up.

Discussion

From a pathophysiological perspective, blood in the subdural space, originating in torn bridging veins, incites an inflammatory reaction which results in the deposition of fibrin and the formation of a subdural neomembrane rich in leaking blood vessels[10] . Because of that vascularity, this neomembrane has large amounts of tissue plasminogen activator (that abounds in blood vessel walls)[8] that can diffuse easily into the hematoma[20]. It then transforms plasminogen into plasmin, the major fibrinolytic protease, that then breaks down the fibrin and fibrinogen present in the subdural clot, resulting in large amounts of fibrin degradation products in the subdural collection that results in a hyper fibrino-

lytic state that favors rebleeding.

What CSH has a tendency to persist and enlarge? As stated above, there is compelling evidence that demonstrate recurrent bleeding into the subdural hematoma as the cause of it[11,19,21]. Subdural fluid has been shown to cause fibrinolysis[9,12] and Vascular Endothelial Growth Factor (VEGF), a key inducer of angiogenesis and promoter of increased capillary permeability, is also found in high concentration in the hematoma fluid[9,17].

How can glucocorticoids ameliorate this situation?

By inhibiting the synthesis rate of protein mediators of inflammation, glucocorticoids inhibit neomembrane formation and the ingrowth of neocapillaries into it [6]. Furthermore, when treated with glucocorticoids, human fibroblasts and endothelial cells (present in the neomembrane) produce an inhibitor directed against plasminogen activator, thus decreasing the hyper fibrinolytic state in the subdural collection[4,22] and with it, reducing tendency for recurrent bleeding.

From a practical point of view, if a patient demonstrates on CT scan a relatively small subdural collection without midline shift and minimal symptoms, a short course of dexamethasone (2 mg PO QID for 7 to 14 days) should be undertaken before considering surgical intervention. In my experience[5] this regimen has been successful in the vast majority of cases, obviating the need for surgery. If reappearance of symptoms occurs shortly after the initial improvement that follows the surgical evacuation of the CSH, a computerized tomography will be required. If a subdural recollection is thus demonstrated, this same short course of dexamethasone should be attempted before considering reoperation. In my experience this regimen has obviated the need for reoperation in most cases[5].

Because this recommended treatment is of short duration, no significant side effects are to be expected, nevertheless, in diabetic patients, careful control of the blood sugar level should be maintained for the duration of it.

Conclusions

The addition of glucocorticoids to the management of CSH seems beneficial by avoiding surgery in Grade I patients and markedly decreasing the need for reoperation in those other cases affected by a subdural recollection.

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