Steroid Pharmacology as Applied to Epidural Steroid Injections

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Introduction

Steroids are well known for their powerful anti-inflammatory properties. Hollander was the first to publish a report describing the injection of hydrocortisone and cortisone into painful arthritic joints in 1951. When it was discovered that injecting steroids into joints could alleviate joint pain, doctors focused their attention on the potential use of steroids in the treatment of spine-related disorders. It became clear that epidural steroid administration could be useful in the treatment of sciatica and low back pain. Later, other types of neural blockade techniques, such as facet joint injections, were described as a treatment option for low back pain. Various complications associated with neuraxial steroid therapy began to be reported around the same time. These included systemic effects of steroids, such as adrenal suppression and atrophy; some reports even advised against the use of neuraxial steroid therapy. The particle size of various steroids used during epidural injections is very important. Several reports in recent years have described devastating complications associated with Epidural Steroid Injections (ESIs), particularly during transforaminal approaches.

Pharmacology of steroids

The adrenal cortex's zona fasciculata is where glucocorticoids are produced. The Hypothalamic-Pituitary-Adrenal (HPA) axis maintains the negative feedback control of the hypothalamus and pituitary gland. Corticotropin-releasing hormone is produced by the hypothalamus, which stimulates the pituitary gland to synthesise Adrenal Corticotropic Hormone (ACTH). The main endogenous glucocorticoid, cortisol, is stimulated by ACTH. It is hypothesised that glucocorticoids improve immunologic activity and wound healing. They're also needed to keep carbohydrate, lipid, and protein metabolism running smoothly. Daily glucocorticoid secretion has been estimated to be equivalent to 20 mg/day-30 mg/day of oral hydrocortisone or 5 mg/day-7 mg/day of oral prednisone. Under severe stress, cortisol synthesis can increase 5 to 10 fold, reaching a maximum of approximately 100 mg/m2 /day. When the renin-angiotensin-aldosterone system is stimulated, mineral corticoids are produced in the adrenal zona glomerulosa. The main endogenous mineralocorticoid is aldosterone. It is involved in the regulation of sodium and potassium homeostasis as well as the maintenance of intravascular volume. The equivalent doses and anti-inflammatory potencies are also listed. According to a literature review, there is no difference in the anti-inflammatory potency of Depo-Medrol (Pfizer) and Kenalog (Bristol Myers Squibb). When the renin-angiotensin-aldosterone system is stimulated, mineral corticoids are produced in the adrenal zona glomerulosa.

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Glucocorticoids inhibit pituitary ACTH release. Exogenous steroids suppress the release of ACTH from the pituitary gland, which affects the normal function of various body systems. As a result, the adrenal cortex stops secreting endogenous corticosteroids, resulting in secondary adrenal cortical insufficiency. The degree and duration of HPA axis suppression vary between patients and are dependent on the dose, frequency, and intervals between administration, as well as the duration of steroid therapy.

Risks to consider

Buffers, polyethylene glycol, benzyl alcohol, and benzalkonium chloride are among the chemicals present in injectable steroid formulations. ESI-related toxicity is frequently delayed. These toxins may not become apparent for months or even years. As a result, many doctors may be unaware of these potentially life-threatening complications. The hypothalamic-pituitary axis may be suppressed by ESIs, resulting in decreased plasma ACTH and adrenal atrophy. Arachnoiditis, intrathecal injection, and particulate embolism are some of the other possible side effects.

Nelson claimed in 1988 that intraspinal therapy with methylprednisolone acetate causes neurotoxicity. His arguments, on the other hand, are regarded as contentious. Many other reports in the literature contradict his claims.

The potential toxicity of various additives used in the formulation of various steroids, namely benzyl alcohol, polyethylene glycol, polysorbate, monobasic and dibasic sodium phosphate, and benzalkonium chloride, has given rise to arguments about nerve toxicity after ESIs. The discovery of benzyl alcohol in Depo-Medrol (Pfizer) and Kenalog (Bristol-Myers Squibb) vials has sparked some concern. A case of flaccid paraplegia lasting 16 months has been linked to benzyl alcohol.

Abram and O'Connor examined the risks and complications associated with ESIs in over 7000 patients. They couldn't find a single case of arachnoiditis. Despite what has been published, Bogduk concluded that there is no direct evidence of neurotoxicity of steroids or their preservatives when injected intrathecally in the lumbar region away from the brain.

Kay JK examined 14 patients and concluded that weekly ESIs given over a three-week period caused a dramatic, acute, and chronic suppression of the HPA axis, with a median suppression of less than one month, and all patients recovered within three months. Hsu looked at plasma cortisol and ACTH levels and discovered that a single epidural injection of 40 mg of triamcinolone significantly reduced plasma cortisol after only 24 hours. The 80 mg triamcinolone dose resulted in decreases up to 14 days after treatment, with HPA axis function returning to normal after 35 days in both groups. Vision loss has been reported as a result of epidural injections, both with and without steroids. According to the available literature, retinal haemorrhages do not appear to be associated with steroid administration.

Several cases of central nervous system injury following transforaminal ESIs have been reported. The most commonly accepted explanation is that the steroid particulate matter occluded the segmental artery or embolized the vertebral artery. Tiso and Benzon looked into the particulate matter associated with common steroids. Segmental medullary vessels are supplied by the vertebral, ascending, and deep cervical arteries. During transforaminal ESIs, the ascending and deep cervical arteries have been reported to be within 2 mm of the needle's path. Many of the radicular arteries in the spine can be completely occluded by the large particles found in various steroid preparations.

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