genetic syndromes with CMI, including many of skeletal abnormality, such as achondroplasia.[1,2]

CMI is thought to be a skeletal mesodermal disorder resulting from faulty division embryonic somites forming the skull base and cranoicervical junction.[2] Governing this process is the gene *Pax-1*, whose malfunction causes vertebral fusion and a small posterior cranial fossa.[2] Altered cerebrospinal fluid flow in this constricted environment produces the varied symptoms, the commonest being suboccipital headache. Ophthalmic symptoms occur in the majority of patients and include retro orbital pain, floaters, photopsia, photophobia, diplopia, and visual field loss. Hearing loss and vestibular impairment causing vertigo, oscillopsia, and nystagmus, as well as symptoms of spinal cord dysfunction, are prevalent.[4]

Extensive literature search has failed to identify another reported case of familial CMI in the United Kingdom. Learning points illustrated herein include the diagnostic challenges posed by CMI, and the importance of assessing family history to increase surveillance of potentially affected relatives.

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Sir,

A brief history of punctoplasty: the 3-snip revisited

We read with interest the article by Caesar *et al*[1] on 'A brief history of punctoplasty: the 3-snip revisited'. In the article, the authors reviewed the development of various surgical methods for treating punctal stenosis and reported their results of 3-snip punctoplasty. We feel that some issues that may affect the outcomes warrant further discussion.

All the patients were assessed for subjective improvement of epiphora at 1 week after operation. This relatively short-term symptomatic evaluation may not be reliable because some patients may, in contrary, experience increased epiphora in the early postoperative period due to surgical wound and associated inflammation.[2] In the four cases (8%) without improvement of epiphora, the reasons could be restenosis of punctum, coexistence of other obstructions in the lacrimal drainage system, lacrimal pump failure, tear hypersecretion, or even dry eye.[3] Information on patient selection, and outcome measures including anatomical success would be relevant in interpreting the results. To avoid operating on patients with symptoms of 'epiphora' caused by dry eye, we would recommend preoperative Shirmer test and fluorescein staining test.

Punctoplasty is usually performed under topical with or without adjacent subcutaneous anaesthetic agents.[4] We observed that some patients may still experience variable degree of intraoperative pain. It is probably due to inadequate penetration of anaesthetic agent into the surgical field that involves excision of a tissue block from the posterior lamella of eyelids. We found that a small amount of local anaesthetic agent such as 2% lignocaine hydrochloride (IMS, CA, USA) into the subcaruncle area instead of subcutaneous injection provides excellent anaethetic result.

We congratulate Caesar and co-workers for their good work. We hope that the discussion would enhance our understanding and treatment of punctal stenosis.

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Sir,

Reply to KSC Yuen *et al*

I would like to thank Dr Yuen and his colleagues for their helpful letter. He raises two important points. A transconjunctival local anaesthetic is indeed as effective as transcutaneous with the benefit of being less painful and causing less bruising; it is now my preferred technique. Secondly, to minimise postoperative inflammation and reduce the chance of early punctal restenosis I now routinely ask the patient to apply G Tobradex in a reducing dose for 4 weeks.

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