SDI Review Form 1.6

Journal Name:	**International Neuropsychiatric Disease Journal**
Manuscript Number:	**Ms_INDJ_31801**
Title of the Manuscript:	**ACUTE NON-COMMUNICATING HYDROCEPHALUS DEVELOPED WITHIN 48 HOURS IN A PATIENT WITH SYSTEMIC LUPUS ERYTHEMATOSUS**
Type of the Article	**Case Report**

General guideline for Peer Review process:

This journal's peer review policy states that **NO** manuscript should be rejected only on the basis of '**lack of Novelty'**, provided the manuscript is scientifically robust and technically sound.
To know the complete guideline for Peer Review process, reviewers are requested to visit this link:

(http://www.sciencedomain.org/page.php?id=sdi-general-editorial-policy#Peer-Review-Guideline)

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PART 1: Review Comments

	Reviewer's comment	**Author's comment** *(if agreed with reviewer, correct the manuscript and highlight that part in the manuscript. It is mandatory that authors should write his/her feedback here)*
Compulsory REVISION comments		
Minor REVISION comments	**1.** Because of the hypertension and chronic renal failure are risk factors for ischemia stroke, I don't agree the author diagnosed the case with SLE-related cerebrovascular accident. Please clarify. **2. Based on the brain CT (Fig. 3), the fourth ventricle is enlarged, communicating hydrocephalus is more likely. Please clarify.** 3. In discussion section, the author say" In the case reported here, we theorize that direct compression of the aqueduct of Sylvius by SLE post-inflammatory reactions led to ischemic infarct and brain edema". I don't think it's proper the etiology is SLE post-inflammatory reaction. Please clarify	We have just mentioned that our patient was followed up in the Neurology and Rheumatolgy departments with diagnosis of SLE , the patient known to have hypertension and chronic renal failure, all of those chronic diseases considered to be as risk factors for cerebrovascular accident. We did not perform biopsy or autopsy on the patient to confirm defenitly of the deposition of immune comlexes that could cause inflammatory reaction which lead to ischemic infarct and brain odema, we only analyse the clinical data and the radiologic views available, we are aware of the week evidence materials available in our hand, all the questions which were said that need explanation by reviewer, they were the same questions and the thought that strnck on our mind, we discussed it and we have decided to documnent it in the litrature becaue it was rare occuring, exciting and challangable case. In all cases of SLE defenitly the cause of the hydrocephalus seems to be due to pathogenesis and pathophysiolgic mechanisms that lead to communicting hydrocephalus as observed in the most research mentioned in the discussion section, reviewing the litrature we can realize that our maniscription might be

		the first to mention about the multiple pathogeneses and pathophysiologic mechanisms that may act in a synergic way in the development of both communicating and non-communicating hydrocephalus. Papathogenes and pathothophysiologic mechanisms that can lead to communicating hydrocephalus and other neuropsychiatric manifestations are also predisposing risk factors for stroke and subsequently to noncommunicating hydrocephalus. we are going to send a thin slice imaging for the comparing the size of the fourth ventricle in the two axial plan cranial CT taken 48 hours apart to observe that there is no clear dilataion of the fourth ventricle like that seen in third and the fourth ventricular .
Optional/General comments	Overall, little evidence to prove this case SLE-related CVA	