INTRODUCTION
Spontaneous intracranial hypotension (SIH) results from single or multi CSF leaks at spine level mainly in the cervical or upper thoracic regions and only rarely from skull base leakage. A progressive orthostatic bilateral headache worsened by Valsalva maneuver or head shaking is the main and most common symptom.[1] Venous vascular dilatation may be the substrate for Pseudo-SAH in SIH.

CASE REPORT
We report the case of a patient with subacute progressive headache whose first brain CT scan was suggestive of subarachnoid hemorrhage which instead had spontaneous intracranial hypotension.

DISCUSSION
Causes of pseudo-SAH
[2,3,4,5,6,7,8,9,10]
- Diffuse cerebral edema
- Anoxic-ischemic encephalopathy (sudden cardiac death/post-resuscitation encephalopathy, causes of cardiopulmonary failure, septic shock); metabolic-toxic encephalopathy (diabetic ketoacidosis, hypotension, narcotics, valproate).
- Chronic hypoxaemia.
- Cerebellar ischemic stroke, gliomatosis cerebri.
- Idiopathic changes in CSF pressure pseudotumour cerebri, SIH.
- Leptomeningitis pyogenic/aseptic/cryptococcal/leukemic meningitis.
- Venous sinus thrombosis, bilateral subdural hematoma.
- Contrast medium intrathecal administration, venous extravasation.
- Other: dural or vessels calcification, bone partial volume averaging.

Diagnostic criteria for SIH [11]
1 Signs/symptoms of decreased intracranial pressure
2 No focal neurological signs, except for cranial nerve
3 Two of the following:
   a) Brain MRI suggestive or normal (20% of cases).[1]
   b) Sustained improvement after epidural blood patching.
   c) CSF opening pressure in sitting position ≤60 mmH2O.
   d) Demonstration of an active spinal CSF leak.
   4 No dural puncture in the 4 weeks preceding onset
5 Not better accounted for by another disorder
ICHDA criteria for headache due to SIH [12]
A Any headache fulfilling criterion C
B CSF pressure ≤60 mmH2O or/and leakage evidence
C H. time-related to CSF low pressure/leakage/discovery
D Not better accounted for by another ICHD-III diagnosis.

REFERENCES
1. Lancet Neurol 2015; 14: 655–68