chirurgiens libéraux. L’impact du revenu hospitalier dans ce choix n’est probablement pas négligeable, les internes de ces spécialités l’évoquent dans plus de 69 % des cas. 

À contrario, le temps libre n’est pas l’explication du départ vers le privé de certaines spécialités, il est partagé par l’ensemble des internes (p = 0,4353). Plus généralement, l’organisation semble être le principal reproche (82 %) fait à l’hôpital universitaire et ce, quelle que soit l’ancienneté des internes. C’est donc dès les études de médecines que ce sentiment existe. C’est de surcroît un facteur d’ajustement sans surcout (contrairement au salaire décrié par 64 % des internes) afin de revaloriser le travail à l’hôpital.

Conflits d’intérêts : aucun.

Références


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Corticosteroid-induced psychiatric episodes in consultation liaison psychiatry. Personality and temperament assessments

Épisodes psychiatriques cortico-induits en psychiatrie de liaison. Évaluation du tempérament et de la personnalité

It is well known that corticosteroids, which have been used since the 1950s, have somatic and psychiatric side effects [1–6]. While the somatic adverse effects are well described, the neuropsychiatric adverse effects have received less attention [7]. The acute side effects are often called corticosteroid...
psychosis, but they can range across most categories of psychopathology [7]. Warrington and Bostwick [7] report that some authors suggested that the occurrence of corticosteroid-induced psychosis depended on the patient's premorbid personality organization [8–9]. They also report that Brody [10] suggested that these reactions reflected an extreme version of a patient's usual stress reaction. Actually, Brody in a series of 8 patients noticed a variety of reactions to cortisone and ACTH. He concluded that this variety of attitudes depended on the personality of the patients and reflected their usual reaction pattern or an exaggeration of it. Unfortunately, his descriptions of personality cannot be referenced by the current definitions of personality [i.e. Diagnostic and Statistical Manual of Mental Disorders (DSM)].

Because we personally observed, in our clinical practice of consultation-liaison psychiatry, two cases of corticosteroid-induced psychosis in patients who presented a hyperthymic temperament, we therefore hypothesized that the underlying psychiatric status (personality, temperament and history of psychiatric disorder) might be a psychological risk factors for corticosteroid-induced psychosis.

We decided to systematically search for a personality and/or a temperament underlying disorder in patients presenting with corticosteroid psychosis, using two scales: Structured clinical interview for DSM III R personality disorders (SCID) II [11,12] and the Akiskal & Hantouche affective temperament questionnaire [13,14].

We collected all of the cases of corticosteroid-induced psychosis according to the DSM IV criteria [15], over a period of 6 months from 798 requests for Consultation-Liaison Psychiatry. We included every patient who presented with an acute symptomatology of the manic, melancholic or delusional type, which occurred while taking corticosteroids, or within 2 weeks of stopping them [15]. We assessed patients with SCID II, a self-administered questionnaire, which identifies 12 types of personality, validated by DSM-IV [11,12] and with a French version of the Akiskal & Hantouche temperament questionnaire, which is a semi-structured interview and self-assessment of affective temperaments [13,14]. Questionnaires were filled out eight days after improvement of the acute corticosteroid psychotonic episode (for enabling patients to correctly answer the questions).

We found 10 patients who presented a corticosteroid-induced psychosis (1.25% of the requests for consultation liaison psychiatry): 3 refused to participate to the assessments, 1 presented with a confusion, 6 accepted to fill out the questionnaires (6 females) aged between 31 and 74 years (M = 59.17; SD = 15.14) (table I).

The illnesses which required corticosteroids were severe (3 malignant hematological disorders, 2 corticoidependent systemic diseases and 1 acute preterm labor). A previous psychiatric disorder was found in 4 patients. The severity of the present episode was variable and 2 patients (N’2 N’5) required a hospitalization in a Psychiatric Department (table II).

Corticosteroid-induced symptomatology took different forms including mood disorders, psychotic disorders, delirium, and anxiety symptoms according to the DSM IV classification. All patients had delusional symptoms, most frequently delusions of persecution (4/6). The mood disorders were all of the manic type. The clinical and biological examinations did not show any other organic etiology linked to those symptoms.

Management of these corticosteroid-induced psychoses systematically involved a reduction or a cessation of the corticosteroid therapy, a cessation of the previously prescribed psychotropic treatment and (in 5 out of the 6 cases), the addition of tranquillizers (hydroxyzine, benzodiazepines) or neuroleptics (table II).

The short-term outcome was a rapid and complete improvement of the symptomatology within 48 hours in 4 out of the 6 cases. For patients N’2 and N’5, the improvement was slower and required them to be transferred to a psychiatric unit. In those two cases, psychotropic treatment was maintained after their discharge and a psychiatric ambulatory follow-up was organized. In the medium run, these 2 patients had a recurrence of the symptomatology that they had developed while taking corticosteroids, but this last episode was not pharmacologically induced. The other patients had no further recurrence of their psychiatric symptomatology (one year follow up monitoring by consultation liaison psychiatry) (table II).

The results of Akiskal & Hantouche affective temperament questionnaire showed that 3 of the 6 patients (N’1, N’4 and N’6) exhibited a specific affective temperament: depressive (N’1), hyperthymic (N’4) and cyclothymic (N’6). The results of the SCID II showed that 6 of the 6 patients had pathological personality traits, but only patient N’5 and patient N’6 exhibited true personality disorders, which were a handicap in their daily lives (table I).

Literature reports rates of corticosteroid-induced psychiatric episodes ranging from 1.8% to 57% in patients receiving corticosteroids [7]. Those large variations are probably linked to the unpredictability of these reactions, large variations in researcher’s criteria, the wide range of doses, and the diverse patient groups [7] and the nature of the Consultation-Liaison Psychiatry practice. In our situation we work on requests of the medico-surgical teams and mild disorders could be underdiagnosed.

In our study, psychiatric symptoms occurred few days after initiation (3 cases), during the course of the treatment (2 cases) or after the cessation of steroids (1 case). Warrington and Bostwick report that psychiatric symptoms can occur at any point corticosteroid treatment [7] even after cessation of treatment as in our study. Those authors report either that females have a minimally, but statistically significant increased risk of psychiatric disturbance: our sample is mostly female but
<table>
<thead>
<tr>
<th>Patient</th>
<th>Gender</th>
<th>Age</th>
<th>Personality (SCID II)</th>
<th>Temperament</th>
<th>DSM IV diagnosis of the corticosteroid-induced episode</th>
<th>Psychiatric History</th>
<th>Cause of Corticosteroid use</th>
</tr>
</thead>
<tbody>
<tr>
<td>N° 1</td>
<td>Female</td>
<td>56</td>
<td>Depressive paranoiac</td>
<td>Undetermined</td>
<td>Depressive Psychotic Episode with manic symptoms day 2. Improvement in 48 h</td>
<td>Major Depressive Episode (ambulatory care)</td>
<td>Lymphomatous transformation of Waldenstrom’s macroglobulinemia</td>
</tr>
<tr>
<td>N° 2</td>
<td>Female</td>
<td>69</td>
<td>Paranoiac schizoid</td>
<td>Undetermined</td>
<td>Undetermined Manic Episode with Psychotic Symptoms after 4 weeks. Hospitalization in Psychiatric Department. Improvement in 4 weeks</td>
<td>Major Depressive Episode with Psychotic symptoms admitted to Psychiatric Hospital</td>
<td>Horton Disease with multiple visceral lesions</td>
</tr>
<tr>
<td>N° 3</td>
<td>Female</td>
<td>64</td>
<td>Dependant Avoidant</td>
<td>Undetermined</td>
<td>Undetermined Psychotic Episode with manic symptoms day 3. Improvement in 72 h</td>
<td>None</td>
<td>Multiple Myeloma IIIa (Durie Salmon classification)</td>
</tr>
<tr>
<td>N° 4</td>
<td>Female</td>
<td>74</td>
<td>Obsessive-compulsive Schizoid Depressive</td>
<td>Undetermined</td>
<td>Hyperthymic Manic Episode with Psychotic Symptoms after 1 year of steroid Improvement in 48 h</td>
<td>None</td>
<td>Corticosteroid dependant Bullous Pemphigoid</td>
</tr>
<tr>
<td>N° 5</td>
<td>Female</td>
<td>31</td>
<td>Dependant Obsessive-compulsive Depressive</td>
<td>Schizoid Schizotypic</td>
<td>Undetermined Psychotic Episode day 2. Hospitalization in Psychiatric Department. Improvement in 4 weeks</td>
<td>Psychotic Episode in adolescence admitted to Psychiatric Hospital</td>
<td>Acute Preterm Labor</td>
</tr>
<tr>
<td>N° 6</td>
<td>Female</td>
<td>61</td>
<td>Border-line Depressive Avoidant</td>
<td>Cyclothymic</td>
<td>Manic Episode with Psychotic Symptoms. 6 days after the stop of steroids Improvement in 48 h</td>
<td>Dysthymia</td>
<td>Multiple Myeloma Illb (Durie Salmon classification)</td>
</tr>
</tbody>
</table>
Table II
Treatment and evolution.

<table>
<thead>
<tr>
<th>Patients</th>
<th>Corticoids (ICD)</th>
<th>Dose (prednisone equivalent), administration, mode duration</th>
<th>Associated Somatic Drugs</th>
<th>Corticosteroid management</th>
<th>Psychotropics prescribed and other pharmacological changes</th>
<th>Evolution of the Psychiatric Episode</th>
</tr>
</thead>
<tbody>
<tr>
<td>N° 1</td>
<td>Methylprednisolone</td>
<td>625 mg/24H IV, Day 1 to Day 4</td>
<td>*ESHAP Protocol</td>
<td>Decrease, followed by cessation</td>
<td>Neuroleptics</td>
<td>Improvement in 48h New Protocol without Methylprednisolone</td>
</tr>
<tr>
<td>N° 2</td>
<td>Prednisone</td>
<td>50 mg/24H per os for 4 weeks</td>
<td>Methotrexate</td>
<td>Progressive decrease, followed by alternative therapy</td>
<td>Neuroleptics</td>
<td>Hospitalization in Psychiatric Department Improvement in 4 weeks Discharged with Haloperidol (10 mg/day) (prescribed during 4 months) Spontaneous recurrence 6 months after</td>
</tr>
<tr>
<td>N° 3</td>
<td>Dexamethasone</td>
<td>266 mg/24H IV, Day 1 to Day 4</td>
<td>**VAD Protocol + Zolpidem (10 mg/day)</td>
<td>Cessation</td>
<td>Zolpidem Cessation Hydroxyzine</td>
<td>Improvement in 48 h Maintain of the Complete Protocol</td>
</tr>
<tr>
<td>N° 4</td>
<td>Hydrocortisone</td>
<td>2,5 mg/24H, per os, for 12 months</td>
<td>Levothyroxin, Acetabutol, Ferrous Iron, Omeprazole All prescribed before hydrocortisone</td>
<td>Cessation</td>
<td>None</td>
<td>Improvement in 48 h No more corticosteroids</td>
</tr>
<tr>
<td>N° 5</td>
<td>Bethamethasone</td>
<td>640 mg/24H, IV, Day 1 and Day 2</td>
<td>Salbutamol + Low Molecular Weight Heparin</td>
<td>Cessation</td>
<td>Benzodiazepines Neuroleptic</td>
<td>Hospitalization in Psychiatric Department Improvement in 4 weeks Amisulpride 800mg/day (6 months) Spontaneous recurrence after 7 months</td>
</tr>
<tr>
<td>N° 6</td>
<td>Dexamethasone</td>
<td>266 mg/24H, per os, Day 1 to Day 4</td>
<td>**VAD Protocol Fentanyl patch Fluoxetine (20 mg/day)</td>
<td>Interrupted since 6 days</td>
<td>Hydroxyzine Discontinuation of Fluoxetine</td>
<td>Improvement in 48 h New Protocol without Dexamethasone</td>
</tr>
</tbody>
</table>

*ESHAP Protocol: Etoposide, Methylprednisolone, High (dose) Aracytin, Cisplatin.
**VAD Protocol: Vincristin, Adriamycin, Dexamethasone (continuous intravenous infusion for 4 days).
it is not linked with the etiologies (e.g. systemic disease’s prevalence is mostly female). There is a difference between steroid induced and steroid withdrawal psychiatric conditions. Steroid withdrawal states are most often depressive in nature and respond to antidepressants. Steroid induced states are most often encephalopathies, manic deliria or manias that respond to mood stabilizers and antipsychotics and not antidepressants [7].

Five different corticosteroids were used. In all of the cases, they were prescribed at a high dose, either as a short parenteral course, or as long term oral treatment (accumulation). Literature only shows that corticosteroid dosage is the most important risk factor for the development of psychiatric symptoms [7] and suggests that the psychiatric symptoms during corticosteroid therapy are dose dependent [16].

In our cases, steroids were always used as a coprescription (in chemotherapy protocols or as the patients’ usual treatment), but psychotic or mood disorders linked to chemotherapy as etoposide, cytarabine (Aracytin®), cisplatin, methotrexate, vincristin, adriamycin are not described in the literature. In two cases (patient N° 3 and N° 6), this treatment included psychotropic drugs (zolpidem 10 mg/day, fluoxetine 20 mg/day) which are known to induced paradoxical excitation (zolpidem) [17] or mania (fluoxetine) [18]. In one case (patient N° 4) the role of levothyroxin - which could induce insomnia, irritability or excitation - still unclear (table II). All psychotropic drugs were stopped, but levothyroxin was continued. Therefore, we cannot confirm that corticosteroids are the only etiology of this symptomatology especially with the coprescription of psychotropic drugs or levothyroxin, but other patients improved with the decrease (or the cessation) of steroids while chemotherapy was maintained.

The role of somatic diseases in our 6 cases is important. To our knowledge, bullous pemphigoid is not known to be linked to psychiatric disorders. But, it is known that about one third of cancer patients suffer from a psychiatric disorder. However, psychiatric disorders in cancer are mostly adjustment disorder, anxiety and depression [19]. Mood disorder could also reveal temporal arteritis: Johnson et al reported a case of a patient presenting with a temporal arteritis whose symptomatology associated psychotic features and affective symptoms both on a background of cognitive impairment improved with corticosteroid treatment [20].

Psychiatric disorders during pregnancy are not rare [21]. Patient N° 5 presented with a psychotic episode during adolescence and a spontaneous recurrence after her corticosteroid induced episode. This case asks us on the role of corticosteroid. Did corticosteroid actually highlight a preexistent psychotic disorder or did corticosteroid induce psychotic disorder in the context of psychic and physic stress due to acute preterm labor?

The objective of our report was to systematically search for a personality and/or a temperament underlying disorder in patients presenting with corticosteroid psychosis using two scales: SCID II [11,12] and the Akiskal & Hantouche affective temperament questionnaire [13,14].

In our 6 cases reports, the Akiskal & Hantouche affective temperament questionnaire showed three different types of temperament in three out of the six patients. Similarly, the SCID II personality scale showed that only two out of the six patients had a true personality disorders and did not show any common personality disorder to the patients who develop a corticosteroid-induced psychosis. Our results do not support the hypothesis of a hyperthymic temperament or a specific personality disorder as risk factor for a corticosteroid-induced psychosis. However, our results allow some links between personality, temperament, the diagnosis of corticosteroid-induced psychosis and the previous psychiatric history (table I). For two patients (N° 4 and N° 5), there was a clinical concordance between the personality and temperament disorders, the previous psychiatric history and the symptomatology of the corticosteroid-induced psychosis. Indeed, patient N° 4 presented a manic episode with psychotic features when taking the corticosteroids, and she exhibited a hyperthymic temperament on testing. Patient N° 5 had schizoid and schizotypic type personality disorders, a previous history of a psychotic episode in adolescence, a psychotic episode when taking corticosteroids and a spontaneous recurrence. These two patients illustrate the continuum described by Kretschmer [22] between structure, personality and mental illness.

For two other patients (N° 1 and N° 6), a dimensional analysis suggests a bipolar disorder. Patient N° 1 had a depressive personality trait, a depressive temperament and a previous history of depression together with a psychotic episode with manic features when taking corticosteroids. Similarly, patient N° 6 had a depressive personality disorder, long-term dysthymia and a corticosteroid-induced manic episode, together with an underlying cyclothymic temperament. Even if manic symptoms occurred 6 days after corticoid cessation in patient N° 6, these two patients fitted in with the hypothesis made by Brown et al. of a similarity in the clinical features, the outcome and treatment of corticosteroid-induced psychosis and bipolar disease, [2,3,16].

Although the underlying personality and temperament do not seem to be determinant in our study for the development of corticosteroid-induced psychosis, the previous psychiatric history of four out of the six patients appears to be important (only the temperament for the patient N° 4).

The hypothesis of vulnerability linked to the presence of previous psychiatric illness is discussed in the literature. Lewis and Smith [23] reviewed 14 previously unreported and 79 cases from the literature of steroid induced psychiatric syndromes. None of their 14 cases had a past history of psychiatric illness unrelated to steroid therapy. Six were thought to have evidence of a premorbid personality disorder. But among 41 cases in the
literature they found 17% of prior history of psychiatric disorder unrelated to steroid therapy and 52% of abnormal premorbid personality disorder. But they were not able to determine whether past psychiatric illness or premorbid personality disorder were risk factors for the development of a steroid-induced psychiatric syndrome.

If Warrington and Bostwick report that a history of psychiatric illness does not predict occurrence [7], some authors, such as Copeman et al. [24], Dunlop et al. [25] Wayne et al. [26] and Stuart et al. [27] confirm that a history of past psychiatric disturbance increased the risk, and so do not allow corticosteroids to be used in psychotic and “mentally unstable” patients. Others, such as Lewis and Smith [23], Lidz et al. [28] disagree, and give low doses of corticosteroids to patients with different psychiatric disorders, without causing the acute symptomatology to reappear. This point still in discussion: in their review, Patten and Neutel [29] confirm that no study can confirm that past psychiatric illness could be a risk factor for the development of a steroid-induced psychiatric syndrome while Sirois [30] showed that some studies suggest the opposite.

Our results could be in favor of vulnerability, linked to the clinical severity of the previous psychiatric history. Patients N°2 and N°5 have had a history of severe psychiatric disease (hospitalization in a forensic psychiatric unit, psychotic symptomatology, long-term follow-up and treatment) which corresponds to long-lasting corticosteroid-induced psychosis. Patients N°1 and N°6 have had a history of psychiatric disease treated on a short-term basis as an outpatient and it corresponds to corticosteroid-induced psychotic episodes which improve more rapidly. The hypothesis of a vulnerability linked to the severity of the previous psychiatric history, means that in these patients, it would be useful to start preventative treatment based on lithium [27,31,32] or another new-generation mood regulator, as long as this is compatible with the other prescribed treatment [33]. However, in clinical practice quite a few diseases treated with corticosteroids provoke renal dysfunction, also corticosteroid-induced changes in sodium balance might increase the risk of lithium intoxication [30].

Clinically, the duration of the corticosteroid-induced psychotic episode in patients N°2 and N°5, seems to be more a recurrence of the original illness than an episode induced by a substance. The question is whether a recurrence of a psychiatric disease (bipolar disease or schizophrenia) is triggered by high-dose corticosteroids, rather than transitory psychiatric symptomatology being induced.

The personality and temperament tests performed on our 6 patients did not confirm our hypothesis of a specific personality and/or temperament as a risk factor for corticosteroid-induced psychosis episode. Moreover, it is not possible to determine whether past psychiatry illness disorder could be risk factors for the development of a corticosteroid-induced psychiatric episode. But, 4 cases highlight that patients experience a complete recovery, although two cases presenting with a history of recurrent psychiatric disorder allows to consider corticosteroids as a trigger of recurrence of their underlying psychiatric disease. These results open the discussion about preventive treatment to limit the emergence of corticosteroid-induced psychosis in patients with a previous history of severe recurrent psychiatric disease.

Conflicts of interests : None.

References

Les adénocarcinomes représentent plus de 50 % des tumeurs malignes affectant le sinus ethmoïde [1]. Il s’agit de maladies rares dont l’exposition chronique aux poussières de bois est un facteur de risque majeur qui lui confère une reconnaissance en tant que maladie professionnelle [2]. Les disséminations méningées sont rares et peu de cas ont été décrits. Celles-ci concernent le plus souvent les cancers du sein, du poumon, le mélanome et les cancers gastro-intestinaux. Le diagnostic difficile des méningites carcinomatose est souvent évoqué tardivement car il s’avère très polymorphe cliniquement, la présence de cellules néoplasiques dans le liquide céphalorachidien (LCR) est inconstante et les images neuroradiologiques non spécifiques.

**Observation**

Un patient de 54 ans, travaillait dans l’agroalimentaire après avoir exercé la profession d’ébéniste de l’âge de 15 ans à l’âge de 40 ans. Dans les antécédents, on retrouvait un accident de la voie publique à l’âge de 20 ans avec traumatisme facial dans la région inter-sourcière au-dessus de l’arête nasale. Le patient n’avait jamais fumé et était souffrant d’une exogène chronique depuis 12 ans. Depuis trois semaines, il avait des troubles de l’équilibre avec des vertiges rotatoires accompagnés de céphalées frontales et occipitales. L’hypothèse initiale était celle d’une neurone vestibulaire qui avait conduit à la prescription d’un traitement symptomatique par.trimétazidine, acétylleucine et corticoïdes solupred® 40 mg/jour. L’imagerie par résonance magnétique (IRM) pratiquée deux semaines plus tard objectivait une prise de contraste bilatérale des deux nerfs vestibulaires, compatible avec le diagnostic de neurone vestibulaire (figure 1).

Dix jours plus tard, le patient était admis aux urgences en début de nuit au décours d’un malaise lipothymique en se levant de sa chaise. L’examen neurologique initial trouvait un déficit sensitivomoteur et sans syndrome méningé. Les artères temporelles étaient bien palpées symétriquement. Le reste de l’examen somatique était normal. L’électrocardiogramme enregistrait un rythme sinusal sans anomalies. Le bilan biologique montrait une hyperleucocytose à 14,11 G/L (73 % de polynucléaires) une glycorachie abaissée (1,5 mmol/L), un rapport glycorachie sur glycémie diminué à 0,22 avec individualisation de 35 % de cellules atypiques. Une ponction lomboïdée réalisée au septième jour montrait une hyperproteinorachie (0,82 g/L), une glycorachie abaissée (1,5 mmol/L), un rapport glycorachie sur glycémie diminué à 0,22 avec individualisation de 35 % de cellules atypiques à l’examen des lames de LCR après coloration au May-Grunwald-Giemsa. L’hypothèse initiale était celle d’une neurone vestibulaire qui avait conduit à la prescription d’un traitement symptomatique par trimétazidine, acétylleucine et corticoïdes (solupred® 40 mg/jour). L’imagerie par résonance magnétique (IRM) pratiquée deux semaines plus tard objectivait une prise de contraste bilatérale des deux nerfs vestibulaires, compatible avec le diagnostic de neurone vestibulaire (figure 1).

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